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JANUARY 1989 • Volume 89, Number 1

MEDICAL SOCIETY OF THE STATE OF NEW YORK
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CIGARETTE SMOKING

FOCUS ON THE WORKPLACE

Contents

COMMENTARIES

Smoking and health: 25 years of progress 1
PASCAL JAMES IMPERATO, MD

Smoking in the workplace: Research needs and potential applications 2
SYDNEY PARKER, PHD, KENNETH E. WARNER, PHD

RESEARCH PAPERS

Evaluating worksite smoking policies: Methodologic issues 5
LOIS BIENER, PHD; DAVID B. ABRAMS, PHD; KAREN EMMONS, PHD; MICHAEL J. FOLLICK, PHD

Implementing nonsmoking policies in the private sector and assessing their effects 11
GLORIAN SORENSEN, PHD, MPH; TERRY F. PECHACEK, PHD

The social epidemiology of smoking during pregnancy among Mexican women 15
CRAIG A. MOLGAARD, PHD, MPH; RAFAEL LANIADO-LABORIN, MD; JOHN P. ELDER, PHD, MPH; CARL DE MOOR, MPH

REVIEW ARTICLES

Trends in the adoption of smoking restrictions in public places and worksites 19
NANCY A. RIGOTTI, MD

Health effects of involuntary smoking in the workplace 27
IRA B. TAGER, MD, MPH

Assessment of smoking behavior in relation to worksite smoking policies 31
RUSSELL E. GLASGOW, PHD

Nonsmoking/smoking policy implementation at the state level 34
KATHLEEN C. HARTY, MED; SUSAN ERSTED, MS; KATHLEEN DALY, PHD; BETH M. PESSIN; DONALD B. BISHOP, PHD

The smoke-free hospital: A smoke-free worksite 38
THOMAS E. KOTTKE, MD

Implementation of a smoking policy in the United States Army 42
ANTIONETTE HAGEY, RNC, MPH

Economic issues related to smoking in the workplace 44
MARVIN M. KRISTEIN, PHD

SPECIAL ARTICLE

Rainier Bancorporation's policy on smoking 48
PETER BROFFMAN, JD

NEWS BRIEFS 50

OBITUARIES 52

GUIDELINES FOR AUTHORS 54

MEDICAL MEETINGS AND LECTURES 6A

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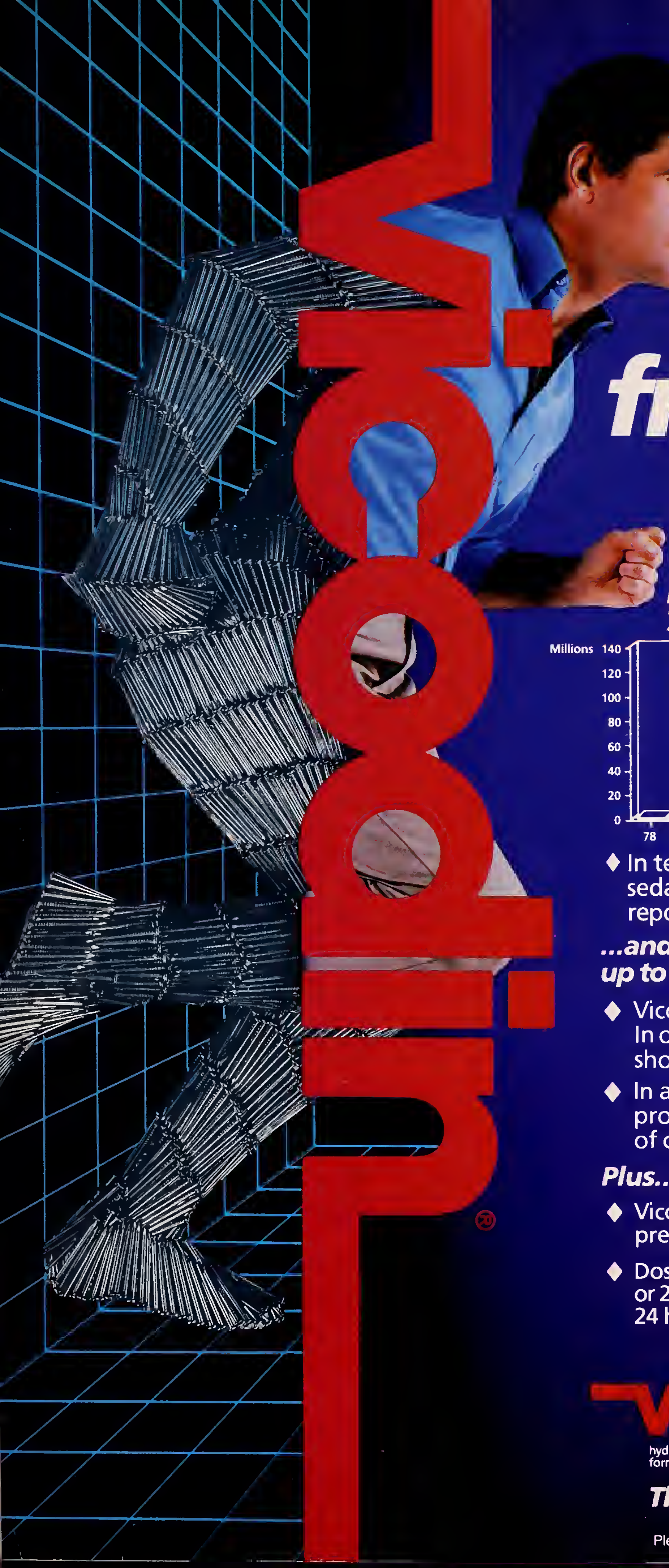

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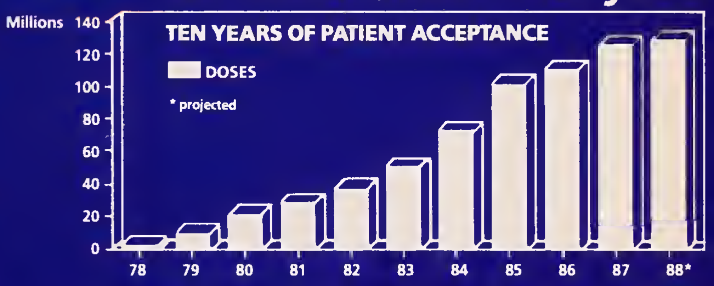

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1. Data on file, Knoll Pharmaceuticals
2. Hopkinson JH III. *Curr Ther Res* 24:503-516, 1978
3. Beaver WT. *Arch Intern Med*, 141:293-300, 1981.

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Head Injury and Increased Intracranial Pressure: The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a preexisting increase in intracranial pressure. Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries
Acute Abdominal Conditions: The administration of narcotics may obscure the diagnosis or clinical course of patients with acute abdominal conditions.
PRECAUTIONS:
Special Risk Patients: As with any narcotic analgesic agent, VICODIN should be used with caution in elderly or debilitated patients and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture. The usual precautions should be observed and the possibility of respiratory depression should be kept in mind.
Information for Patients: VICODIN, like all narcotics, may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery; patients should be cautioned accordingly.
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Drug Interactions: Patients receiving other narcotic analgesics, antipsychotics, antianxiety agents, or other CNS depressants (including alcohol) concomitantly with VICODIN may exhibit an additive CNS depression. When combined therapy is contemplated, the dose of one or both agents should be reduced
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Pediatric Use: Safety and effectiveness in children have not been established
ADVERSE REACTIONS:
Central Nervous System: Sedation, drowsiness, mental clouding, lethargy, impairment of mental and physical performance, anxiety, fear, dysphoria, dizziness, psychic dependence, mood changes.
Gastrointestinal System: Nausea and vomiting may occur, they are more frequent in ambulatory than in recumbent patients. The antiemetic phenothiazines are useful in suppressing these effects; however, some phenothiazine derivatives seem to be antianalgesic and to increase the amount of narcotic required to produce pain relief, while other phenothiazines reduce the amount of narcotic required to produce a given level of analgesia Prolonged administration of VICODIN may produce constipation.
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AROUND THE STATE

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Feb 16. **Current Issues in TB: A Long Island Focus.** 3 Cat I Credits. Nassau County Medical Center. Contact: Dr Ian Newmark, 175 Jericho Tpke, Syosset, NY 11791. Tel: (516) 496-7900.

MANHATTAN

Feb 2. **Myocardial Disease—1989.** 2 Cat I Credits. Helmsley Hotel. Contact: New York Cardiological Society, Inc, 84 Grove St, Suite 2, New York, NY 10014. Tel: (212) 924-5346.

AROUND THE NATION

ARIZONA

Feb 18–20. **Sixth Annual Mohs Micrographic Surgery Conference of the Skin Care Foundation.** Wyndham Paradise Valley Resort, Scottsdale. Contact: Mitzi Moulds, Executive Director, The Skin Care Foundation, 245 Fifth Ave, Suite 2402, New York, NY 10016. Tel: (212) 725-5176.

CALIFORNIA

Feb 10–12. **Current Concepts in Pediatric Medicine.** 18 Cat I Credits. San Diego Marriott and Marina, San Diego. Contact: CME Registration, American Academy of Pediatrics, PO Box 927, Elk Grove Village, IL 60009-0927. Tel: (312) 981-7884.

Feb 15–17. **Recent Advances in Neurology.** 15 ½ Cat I Credits. Fairmont Hotel, San Francisco. *Feb 17–18.* **Advances in Skin Cancer Management: A Multispecialty Approach.** 10 Cat I Credits. Ramada Renaissance Hotel, San Francisco. Contact: Extended Programs in Medical Education, University of California, Room U-569, San Francisco, CA 94143-0742. Tel: (415) 476-4251.

Feb 23–25. **Advanced Seminars in Diagnostic Imaging.** Town and Country Hotel, San Diego. *Feb 24–26.* **Advanced Seminars in Diagnostic Imaging.** Ritz-Carlton Hotel, Rancho Mirage (Palm Springs). 18 Cat I Credits. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077-1925. Tel: (404) 641-9773.

DISTRICT OF COLUMBIA

Feb 3–5. **Advanced Trauma Life Support Instructor Course.** 26 Cat I Credits. Washington. Contact: Delia Soto, Georgetown University Hospital, 3800 Reservoir Rd, NW, Washington, DC 20007. Tel: (202) 784-2074.

FLORIDA

Feb 3–5. **Drugs for the '90's: An Update on Drug Therapy.** 14 Cat I Credits. Colony Beach Resort, Longboat Key. Contact: Thelma Bassett, University of Miami South Campus, 12500 SW 152nd St, Miami, FL 33177. Tel: (305) 284-7350.

Feb 5–8. **American Society for Parenteral & Enteral Nutrition's 13th Clinical Congress.** Fontainebleau Hilton, Miami Beach. Contact: American Society for Parenteral & Enteral Nutrition, 8605 Cameron St, #500, Silver Spring, MD 20910. Tel: (301) 587-6315.

Feb 6–8. **12th Annual Gold Coast Ob/Gyn Conference: Obstetric and Gynecologic Option: Update Ultrasound and Laser Technology.** 9 Cat I Credits. Good Samaritan Hospital, West Palm Beach. *Feb 26–28.* **Vascular Surgery—1989.** 12 Cat I Credits. Palm Beach Airport Hilton, West Palm Beach. Contact: Laura J. Lyons, CME Coordinator, Good Samaritan Hospital, PO Box 3166, West Palm Beach, FL 33402. Tel: (407) 650-6236.

Feb 10–12. **Workshop in Bedside and Electrocardiographic Diagnosis.** Rolling Hills Golf Resort, Ft Lauderdale. Contact: Anne Criss, Rogers Heart Foundation, 1200 7th Ave N, St Petersburg, FL 33705. Tel: (813) 894-0790.

(*continued on p 12A*)

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COMMENTARIES

Smoking and health: 25 years of progress

January 11, 1989, marks the 25th anniversary of the publication of the surgeon general's advisory committee report, *Smoking and Health*.[1] This landmark publication represented the first official recognition in the United States of the enormous deleterious effects of tobacco on health. Writing in the pages of this *Journal* in December 1983, Luther L. Terry, MD, who as surgeon general of the US Public Health Service had appointed the advisory committee, characterized the report as " . . . America's first recognition of the enormous toll taken by tobacco."[2]

Scientific evidence linking tobacco use and adverse health effects had been growing since the late 1920s.[3-6] By the time the advisory committee met in late 1962 there were at least 7,000 articles in the literature on the subject of tobacco and health.[2] However, it was the report of the advisory committee, with its strong condemnation of tobacco use—especially cigarette smoking—that first focused public attention on the negative health impact of smoking. Since the issuance of the 1964 report, steady progress has been achieved in the United States in the public health effort to prevent tobacco use. Declines in per capita consumption and in the percentage of smokers in the United States have been encouraging. Yet some 54 million American men and women still smoke and of these half are heavy smokers, consuming 25 or more cigarettes a day.[7] Continued public health and other efforts are obviously still needed to bring about dramatic reductions in the number of smokers and in per capita consumption of cigarettes.

This issue of the *Journal* is the fourth thematic one uniquely devoted to tobacco use and health that has been published by the Medical Society of the State of New York in the past five years. The first, "The World Cigarette Pandemic," was published in December 1983, the second, "The World Cigarette Pandemic—Part II," in July 1985, and the third, "Cigarette Smoking, Tobacco and Health: International Perspectives," in September 1986. The focus of this issue is on cigarette smoking in the workplace. Most of the papers were presented at a workshop, "Smoking Policies in the Workplace: Research Needs and Potential Applications," sponsored by the Division of Lung Diseases, the National Heart, Lung and Blood Institute; the National Cancer Institute; and the Institute for the Study of Smoking Behavior and Policy, John F. Kennedy School of Government, Harvard University.

Smoking in the workplace has rapidly moved to the top of the public health agenda within a relatively short period of time. There are two forces which have brought this about: concerns about the health risks of passive (involuntary) smoking and altered public attitudes about the social acceptability of smoking.[8] Scientific data about the risks of passive smoking, while convincing, would probably have been insufficient to alter public policy with regard to smoking in public places and the workplace had not strong negative public attitudes about smoking also developed. It is clear now from a number of polls that the majority of adult Americans favor a ban on smoking in public places. Polls have also shown that the vast majority of Americans, including smokers, favor restricting smoking in the workplace to certain designated areas.

Rigotti[8] makes the cogent observation that Americans actually favor a higher level of restriction on smoking than that now in existence. She also points out that public support for restricting smoking preceded and did not follow either laws imposing it or much of the evidence about the health risks associated with passive smoking.[8] Workplaces have received intense attention because nonsmokers once had little or no choice about passive smoking in this setting. Another reason why the workplace has been the focus of public concern is that it is the principal site of exposure to passive smoke for those adults who do not live with smokers at home.[8] Another concern has been the possibility of synergism between passive smoke and environmental workplace toxins.[8]

Public policies aimed at the restriction of smoking at the workplace have as their principal aim the reduction of exposure to passive smoke incurred by nonsmokers. It is already clear that when these policies are promulgated and enforced they are successful. However, these policies may also contribute to preventing young people from starting the smoking habit and helping smokers in giving it up. It will require time and careful research to determine whether or not these desirable secondary effects of workplace smoking restrictions occur. A new frontier of smoking research has appeared with the promulgation of workplace smoking restrictions. As Rigotti observes, the evaluation of workplace smoking policies should become a high priority for research.[8] The purpose of this research must be not merely to evaluate the impact of laws on the reduction of passive smoke but, more importantly, to see if these new policies have any impact on smoking behavior.

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*The opinions expressed in this editorial are those of the author alone and do not constitute an expression of official opinion of the Medical Society of the State of New York.

In retrospectively looking back over the quarter of a century since *Smoking and Health* was published, it is clear that much progress has been made. The current effort at reducing the workplace exposure of nonsmokers to passive smoke is a measure of this progress. Obviously there is a great deal more that needs to be done. However, in looking at the challenges ahead one should not lose sight of the success already achieved nor of the unique accomplishments of the late Dr Luther L. Terry and the members of the Surgeon General's Advisory Committee on Smoking and Health who made public health history 25 years ago.

The *Journal* is pleased to present this thematic issue that focuses on smoking in the workplace. It is hoped that it will serve to inform readers about the complexities of this major public health issue and foster discussion of it. Special thanks are extended to Nancy A. Rigotti, MD, Associate Director, Institute for the Study of Smoking Behavior and Policy, Harvard University, who first suggested the possibility of this thematic issue, and to Sydney R. Parker, PhD, Chief, Prevention, Education and Research Training Branch, Division of Lung Diseases, National Heart, Lung, and Blood Institute, National Institute of Health, who made it happen.

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Smoking in the workplace: Research needs and potential applications

In the quarter century since the first report of the surgeon general on smoking and health in 1964,[1] attitudes toward smoking and restrictions on smoking in public places and workplaces have changed dramatically. In the space of a few years, restrictions on smoking in the workplace have gone from something of an aberration, an object of curiosity, to an emerging social norm. It is no longer rare to read in a local newspaper that another company, or fire department, has announced that it will not hire smokers. Towns have discussed complete smoking bans in restaurants, and at least one contemplates an ordinance to ban smoking in any public place, including outdoors. As one might expect from observing these activities, attitudes toward restrictions on smoking have changed markedly. Surveys consistently show that a majority of both nonsmokers *and* smokers favor restrictions on smoking in the workplace.

Certainly it was widely anticipated that there would be a movement in this direction in the wake of recent reports of the surgeon general[2,3] and the National Academy of Sciences[4] on the health effects of involuntary smoking. But the rapidity of social change in this area has occasionally bewildered even those who for years have been concerned with the health hazards of cigarette smoking.

Unfortunately, little research has been conducted on the implementation of policies and their effects. It is often assumed that nonsmoking restrictions contribute to cessation and to a reduction in total smoking. Yet few evaluation data are available on the effects of the implementation of smoking policies in the community or workplace. Several model policies have been developed and have proven useful to many organizations. However, research on their effects has been lacking.

This special issue of the *New York State Journal of Medicine* presents the proceedings of a workshop convened in 1987 to examine the issue of smoking policies in the workplace. The workshop, "Smoking Policies in the Workplace: Research Needs and Potential Applications," was convened by the National Heart, Lung, and Blood Institute and cosponsored by the National Cancer Institute and the Institute for the Study of Smoking Behavior and Policy at the John F. Kennedy School of Government at Harvard University. The workshop brought together smoking researchers, public health specialists, and representatives from businesses, hospitals, state governments, the military, and other organizations to discuss what is known about the prevalence, types, and effects of smoking policies in the community and in the workplace. The workshop identified gaps in information regarding the factors influencing the development, implementation, and enforcement of smoking policies, and made recommendations for future research.

Smoking policies have emerged primarily because of growing awareness of the harmful effects of exposure to environmental tobacco smoke (ETS). These have been documented in two comprehensive reports.[3,4] Tager's[5] article in this issue of the *Journal* notes that only a few studies have addressed the question of the contribution of workplace exposure to increased risk of harmful effects. The most significant acute effect of exposure to environmental tobacco smoke is irritation. Eye, nose, and throat irritation, cough, headache, and wheeze are associated with exposure. These irritant effects are frequently experienced when workers are exposed to ETS in the workplace, although the available studies do not always report them. Asthmatics particularly show increased airway hyperreactivity when exposed to ETS. Longer term studies of ETS exposure in the workplace have associated exposure with lowered lung function and the development of lung cancer. It is also possible that ETS in the workplace is harmful to the developing fetus. Exposure to ETS by pregnant women is associated with low birth weight, and some of this exposure may be derived from exposure at work.[5]

In her review of policies restricting smoking, Rigotti[6] documents the extent of implementation of restrictions on smoking in public places, including worksites. Although early policies were dictated by safety and morality concerns, in the 1970s the nonsmokers' rights movement based its antismoking activities on the rights of nonsmokers to a smoke-free environment. By 1987, 44 states and the District of Columbia regulated smoking in at least one place.[7] Comprehensiveness of the policies also increased over the years, with many states strengthening existing smoking statutes. Local communities have also become active in establishing ordinances restricting smoking.

Although restrictions on smoking specifically in the workplace have been generated by legislative and executive action of federal, state, and local governments, the private sector has itself been increasingly active in implementing policies. As Broffman's[8] article indicates, businesses have adopted these policies because of concerns for employee health, complaints about smoking in the workplace, the changing legal and regulatory climate regarding smoking, and court decisions upholding the rights of employees to a smoke-free workplace. Several surveys of businesses in the early 1980s showed that about one third of businesses had some kind of smoking policy. A recent report from the Bureau of National Affairs (BNA)[9] indicated that by 1987, more than one half (54%) of businesses responding to a BNA survey reported having policies, up from 30% found in a similar survey conducted by the same organization one year earlier.[10] Most of these policies specify where smoking is allowed, making nonsmoking the norm in the remainder of the facility.

Public and employee attitudes support the development of smoking policies in the workplace.[6] Even smokers are supportive of these policies. As Sorensen[11] points out in her study of seven worksites, the majority of smokers and recent quitters support separation of smoking and nonsmoking areas, smoking cessation programs, and restriction of smoking in the place of employment. They did not favor complete bans on smoking. Those who have recently quit or attempted to quit are more supportive of restrictions and prohibitions than are current smokers. The study also showed that smokers adapt to new policies rather than leaving their jobs.

This special issue of the *Journal* presents several examples of organizations that have implemented policies. These examples indicate that the implementation of policies is feasible and causes minimal disruption. Broffman[8] gives an account of the implementation of a complete smoking ban in a major bank, phased in over time, which was accepted by the employees and which caused minimal disruption to the organization. Harty et al[12] describe the implementation over a one-year period of a smoking ban in the Department of Health in the State of Minnesota. The policy was developed by a committee with participation by elected employee representatives as well as individuals appointed by the Commissioner of Health. Although it is difficult to tell what kind of effect the policy had on smoking prevalence, 30% of smokers indicated that they smoked less since the policy went into effect.

Hospitals are another organizational environment in which smoking restrictions and bans have been implemented. Kottke[13] describes several barriers in the social system of hospitals which must be overcome before change can take place. Biener et al[14] report a study comparing two hospitals in a quasi-experimental design, in which one hospital implemented a smoking policy. The policy was accepted by employees in the hospital and caused minimal disruption to the organization. Air quality at the policy hospital improved. The policy resulted in a decrease in the number of cigarettes smoked at work, but no significant effect on cessation of smoking. It did produce a short term increase in intentions to quit, however.

Hagey[15] presents a fascinating description of the United States Army's adoption of a smoking policy which was implemented quickly and comprehensively, and appears to have contributed to a reduction in the smoking rate in the army, as well as to a reduction in sales of tobacco products in army commissaries.

Although smoking policies are being implemented in an increasing number of organizations, rarely have they been evaluated. Companies apparently implement policies, experience minimal disruption, and see no need to evaluate their effects more thoroughly. However, there are several questions which remain. Answers to these questions would be useful to organizations considering implementing policies, as well as to those public health professionals who are using the worksite to transmit health messages to the workforce. The major questions which should be addressed fall into three categories.

- What differences do smoking policies make in total smoking behavior, on-the-job smoking, air quality, social norms regarding smoking, and the costs of smoking in the workplace? Are there unanticipated effects of policies on the organization, its personnel, or the work environment?

- How do organizational processes such as corporate leadership, culture, and norms influence the initiation, implementation, and success of programs? What factors facilitate or inhibit the implementation of policies? How can conflict be minimized when an organization is implementing a policy?
- How can policies be incorporated into health risk reduction programs to help the smoker quit initially and prevent relapse later?

Randomized controlled studies are the most rigorous way of studying the effects of smoking policies. Given the cost and logistical problems involved in these types of studies, however, evaluations of smoking policies as they are initiated and implemented often may be much more feasible. Assessments of these "naturally occurring experiments" can provide fascinating insights into a rapidly changing social phenomenon. Yet access to the data regarding the effects of smoking policies also may not be easy. Organizations are generally reluctant to allow outsiders to gather data from employees. Some organizations are willing to accommodate a research study if the study causes a minimum of disruption and benefits the organization in some way. It is also possible that existing data may be used in evaluating the effects of policies. Retrospective assessment of effects may be accomplished by use of sources, such as company specific data sets, aggregations of company-specific data, and survey data gathered by government or private sources.

Even when access to the worksite is assured, several methodological issues must be solved regarding the measurement of smoking behavior, including measurement of mediating variables such as organizational characteristics, employee subgroup characteristics, and implementation variables, which are discussed by Glasgow.[16] Measurement of air quality and exposure to ETS is also difficult, as Tager[5] describes. Biener et al[14] further describe issues regarding research design, such as the need for comparison groups, timing of assessments, sample selection, and the issue of cross-sectional versus longitudinal research designs.

Assessment of the effects of smoking policies on economic costs to employers is also difficult. Kristein[17] has reviewed current economic issues related to smoking and the cost effectiveness of restricting smoking in the workplace. Kristein concludes that employees' smoking habits represent a significant cost to employers. Whether an individual employer can actively show cost savings as a result of the implementation of a smoking policy is not yet resolved.

A significant proportion of the US workforce continues to smoke. More data are needed on the effects of restrictions on the smoking attitudes and behaviors of the workforce. Studies combining smoking cessation efforts with smoking policies should be undertaken to develop methods to assist smokers in quitting and to provide continuing support for those who have quit. It is possible that smoking policies may be most effective in preventing relapse in those who have quit, rather than in motivating cessation initially, since smokers seem to be adapting to the policies quickly. However, long term data are necessary to assess whether smoking restrictions encourage cessation at a later point. Smoking restrictions are feasible, and their increasing popularity points to the rapidly declining social acceptability of smoking in American society.

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Evaluating worksite smoking policies

Methodologic issues

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ABSTRACT. This paper discusses the methodologic issues one confronts when studying the impact of worksite smoking policies, using one such study as an example. Topics covered include defining the research questions, deciding on timing of measurements in relation to policy implementation, advantages and disadvantages of cohort versus cross-sectional research designs, and alternative measurement techniques. The sample study examined changes over one year in the smoking-related behaviors and attitudes of employees of a hospital that initiated a restrictive smoking policy, in relation to employees of a comparable hospital without a restrictive policy. The short term impact on ambient carbon monoxide was also examined. A case is made for studying the impact of smoking policy in the context of other strategies for reducing smoking prevalence at the worksite, such as individual treatment and organizational initiatives to motivate smoking cessation.

(NY State J Med 1989; 89:5–10)

The proliferation of increasingly restrictive worksite smoking policies is expected to be one of the more effective means of reducing smoking prevalence and contributing to the national goal of a smoke-free society.[1] As more and more employers respond to pressures to adopt such policies, they look to researchers for advice on how policies should be formulated and implemented. Although helpful guidelines are available from several sources,[2–4] none has been based on systematic evaluations of worksite smoking policy development.

Research on policy restrictions should be placed in a broader conceptual context so as to explore the potential for separate and interactive effects of policy and other organization-wide environmental change strategies and individual programs. Ultimately, a coherent, theory-driven model should be developed that can be used as a blueprint for developing and evaluating optimal approaches to health promotion at the workplace. Such comprehensive approaches should include smoking policy options, but within the context of multifaceted interventions, where synergistic effects can be obtained to improve overall effectiveness.[5] Interventions directed at multiple levels, eg, individuals, groups, and organizations, may have greater impact than those directed at a single level.

The ideal way to acquire knowledge about the effects of restrictive policies alone would be to conduct controlled experiments in which worksites are randomly assigned to receive policy changes which vary according to the parameters of interest (eg, level of restrictiveness, amount of forewarning, degree of enforcement). The most effective components of policy change could then be isolated. Next, the separate and interactive effects of other smoking reduction strategies at the worksite, such as incentives or quitting programs, could be tested in additive treatment designs. In this systematic approach, the active components within multifaceted treatments could be identified and included in the overall blueprint for action.

Although randomized controlled trials and programmatic research using components analysis provide the ideal scientific test of program efficacy, limitations in the researcher's ability to control corporate policy decisions tend to make this approach impractical. Nevertheless, we do not have to rely solely on anecdotal reports or on assessments of individual cases. As more and more worksites adopt restrictive policies, opportunities become available to take advantage of naturally occurring experiments. By studying changes in worksites that adopt restrictive policies relative to carefully chosen comparison worksites where no policy change takes place, one can develop empirically based guidelines for policy implementation. Such "quasi-experiments"[6] or "natural experiments" provide the most rigorous means available for studying events that the investigator cannot control independently. This paper describes one such study, with the goal of highlighting methodologic issues that arise when doing research of this kind. A comparison was made between a worksite implementing a policy change coupled with a self-help cessation program and a matched comparison worksite simply offering a cessation program alone.

Background. Two similar sized general hospitals affiliated with the Brown University Program in Medicine (Providence, RI) were recruited to participate in a larger

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Presented in June 1987 at a workshop in Bethesda, Md, "Smoking Policies in the Workplace: Research Needs and Potential Applications," sponsored by the Division of Lung Diseases, National Heart, Lung, and Blood Institute; the Institute for the Study of Smoking Behavior and Policy at the John F. Kennedy School of Government at Harvard University; and the National Cancer Institute.

study of a self-help smoking cessation program administered at the worksite.[7] Each hospital had approximately 250 beds and approximately 1,400 employees. At the beginning of the study, the smoking policy was similar in both hospitals. There were several designated no smoking areas, but smoking was permitted in many areas, including most offices and lounges. Four months prior to the scheduled implementation of the self-help smoking cessation programs, we learned that one of the hospitals was about to implement a smoking policy that would restrict employee smoking to the cafeteria and coffee shop. The other hospital, which was not planning any policy change, was well-matched in staff profile and size, and thus was a suitable comparison site. The administrators of both hospitals were easily convinced that this provided an excellent opportunity to learn something about the impact of the smoking policy on employee attitudes and behaviors—information that could be useful to both hospitals.

METHODS

The description of the methods used in the present study will be presented in the context of choices that may be available to future researchers. Although research designs are formulated within constraints of resources, time, and access to data, it is useful to consider the range of potential alternatives. In retrospect, some of our choices were more useful than others.

The Research Questions. Wherever possible, multiple measures of outcome should be considered along with measures of hypothesized mechanisms that mediate outcome. In this study, the evaluation was designed to provide information on four general issues: approval/acceptance of the restrictive policy; effect of the policy on indoor air quality; effects on attitudes and norms regarding smoking; and effects on employee smoking patterns. Other potentially relevant measures that were not included were the effects of the policy on organizational climate[8]; on job satisfaction; and on related outcomes such as the health status of employees, productivity, and the cost of health benefits. This section will present the range of measurement techniques for answering the four research questions and a description of the techniques used in the present study.

Approval/acceptance. A major concern of employers considering restrictive smoking policies is whether policy implementation will have an adverse effect on performance or provoke disruptive conflict among employees. These questions can be assessed through self-report and also through objective indicators such as absenteeism and resignations. The present study assessed self-reports of awareness and approval of policy change at the affected worksite, and employees' perceptions of changes in their performance.

Effects on air quality. A primary goal of a restrictive policy is to reduce nonsmokers' exposure to environmental tobacco smoke (ETS), and thereby reduce their disease risk. There are several ways to measure exposure: nonsmokers' perceptions of air quality; analysis of tobacco smoke constituents in the air such as respirable suspended particulates, carbon monoxide, or nicotine[9,10]; biochemical indicators of exposure in bodily fluids such as cotinine—a metabolite of nicotine found in serum, urine, or saliva[11–13]; and pulmonary function in nonsmokers.[14]

In the present study, we used nonsmokers' perceptions of air quality and, because of ease of measurement, levels of ambient carbon monoxide (CO). However, there are a number of methodologic considerations associated with assessment of air quality. First, ambient CO is produced in the vapor phase and dissipates quite quickly, often resulting in measurement of very low levels of CO. Other components of cigarette smoke that are present as particulate matter are longer lasting in both ambient air and in the respiratory system, and thus may be more easily measured and more sensitive to change in ambient air concentration.[15,16] One study examined ambient CO, particulate matter, and nicotine during and after working hours in various workrooms where smoking was permitted.[16] It was found that CO concentrations increased by only 1.1 units during working/smoking hours, while particulate matter increased by 133 units. It should also be noted that particulate matter, due to its longer presence both in the air and in the respiratory system, may be a more direct correlate of passive smoking-related disease, and thus may be an important marker for examining the relationship between reduced passive smoking exposure and disease reduction.

Another concern with using CO to estimate passive smoke exposure is its lack of specificity.[15] Carbon monoxide is generated by other environmental sources, such as gas stoves, machinery, and automobile exhaust.[17,18] Biochemical indicators such as blood and saliva cotinine have been found to be sensitive to both low levels of passive smoke exposure and changes in exposure levels.[19]

Sampling of nonsmokers' exposure is also possible through the use of a small filter ("passive sampler") that nonsmokers wear for a period of several days. Analysis of the passive sampling filter provides an estimate of exposure to nicotine, cadmium, and respirable particulate matter (RSP).[9] Pulmonary function measures provide another potentially important estimate of passive smoke exposure. There is evidence that nonsmokers with high levels of exposure to secondhand smoke have significantly reduced lung capacity, relative to normative predicted values and to lung capacity of individuals with low levels of exposure.[14] While these other measures may provide a more valid assessment of passive exposure to ETS, they tend to be costly in terms of equipment requirements and respondent burden.

Effects on attitudes and norms. It has been suggested that implementation of restrictive smoking policies will contribute to a change in social norms regarding the appropriateness of smoking, and hence may increase nonsmokers' willingness to express concerns about the hazards of smoking at work.[20] Objective indicators of norms could be derived from time-sampled observations of nonsmokers' reactions to smokers. Since confrontation with smokers is likely to be a low frequency behavior, however, reactions to staged instances of smoking might be more efficient. In this study, we assessed nonsmokers' beliefs about the hazards of secondhand smoke and self-reports of their willingness to confront coworkers whose smoking was bothersome. We also assessed smokers' perceptions of the extent to which their coworkers disapproved of their smoking.

Effects on smokers' behavior. The combination of reduced opportunities to smoke and the potential pressure of coworker disapproval could increase smokers' motivation to quit. Policy change could also reduce the situational cues that trigger smoking at work. If smokers who have recently quit have less opportunity to observe others smoking, it could reduce their risk of relapse. This could result in lower smoking prevalance at the worksite.[20,21] In this study, we measured smokers' behavior through self-report of smoking status and smoking patterns, as well as reports of motivation to quit smoking. Objective measures of smoking reduction/cessation such as saliva cotinine should also be considered, despite the cost of collection and analysis.[11]

Research Design. *Need for comparison groups.* The current media and legislative attention to the issue of smoking makes it difficult to disentangle effects of societal changes from effects of a policy in a particular worksite. The addition of matched comparison worksites assessed at the same time as the target worksite allows for estimation of these secular changes. For example, Rigotti et al[22] evaluated the impact of a restrictive policy on nurses in one hospital unit in comparison to nurses in another unit where no policy change occurred. In the present study, a survey of employees at a second hospital without a restrictive policy provided an index of background changes. Assessments of changes in ambient CO in the comparison hospital would have been very useful. However, limited resources precluded obtaining them, and only pre-test and post-test assessments were used for this measure. Items on acceptance and approval of the policy were, of course, relevant only to the target hospital.

Timing of assessments in relation to policy implementation.

Timing of assessments is crucial to the evaluation of policy change. Published studies of policy change frequently rely on retrospective reports in which smokers and nonsmokers are surveyed after a policy change has occurred. Respondents are asked about their current smoking behavior and attitudes and about how they felt prior to the change.[23,24] Since recollection of previous attitudes may be incomplete or subject to bias, collection of baseline data prior to policy implementation provides more valid information. However, determining the onset of the actual effects of a policy change is not a straightforward matter. Although the formal date of implementation of a policy change can be identified, the initiation of discussion of the impending change is difficult to pinpoint. Rumors and expectations of a restrictive policy may bring about changes in behaviors and attitudes, or at least instability in measures. Interviews with key employees can help the researcher to mark general outlines of periods of discussion of the policy. It is also important to recognize and document relevant events in the wider community. Such information will help in interpreting results.

Follow-up periods must also be considered. Both short term and long term follow-up are desirable because policy may have greatest impact on some outcomes at the time of implementation (eg, smoking rate and air quality), while other outcomes take longer to detect (eg, norm changes and employer cost savings). In the present study, we were able to collect data one month prior to the formal implementation of policy change, and to repeat assessments of employees at six and 12 months after policy implementation.

Sample selection. In order to draw conclusions about the impact of the policy, a representative sample of the employee population must be chosen. Stratified sampling that allows for oversampling of smokers will increase the ability to monitor effects on smoking behavior. Casual samples, such as cafeteria users who choose to return an anonymous questionnaire, are frequently used, but could be biased in ways that present a distorted picture of population attitudes. Cohort designs, whereby one can follow the same people over time, provide the most sensitive procedure for detecting change. Cohorts, however, have several disadvantages. First, the repeated contacts can be "reactive"—they can influence the behavior of interest. Second, attrition from a cohort sample can lead to loss of statistical power to detect differences because of the reduced sample size. Third, attrition may occur disproportionately in particular groups (smokers unhappy about the policy change) and can compromise the validity of conclusions.

Cross-sectional designs, or the sampling of repeated but different groupings of the population across time, eliminate some of the problems associated with cohort samples. However, cross-sectional sampling can add error variance to the analysis, and thus, for any given sample size, it is more difficult to detect changes. Cross-sectional samples also require certain assumptions to be made about baseline equivalence, and they preclude the use of covariance analyses. The alternative we chose was a mixed design that included both longitudinal and cross-sectional random samples. Although this procedure increases sample size requirements, it increases the probability of getting representative and interpretable results. It also permits examination of the possible reactive effects of repeated sampling by comparing cohort to cross-sectional samples at follow-up.

In the present study, lists were obtained of all full-time employees of the policy hospital and comparison hospital prior to the implementation of the policy at the target hospital. A random sample of 85 employees at each hospital was identified as a cohort, and individuals were interviewed by phone during working hours at one month prior to the policy change, and at six and 12 months following the policy change. The cross-sectional sample consisted of independent samples drawn without replacement from the same employee lists and interviewed at the same time periods. The intention was to sample 85 employees at each hospital at each time period, under the assumption that this would yield approximately 25 smokers per sample. However, the six-month assessment yielded only 14 smokers at the policy hospital and 18 at the comparison hospital. Hence, the sampling frame was increased to 120 employees for the 12-month assessment.

Air quality sampling. Ambient CO readings were taken in the policy hospital immediately before and after, and six months following policy implementation. Four locations were selected for carbon monoxide sampling: the nonsmoking and smoking sections of the cafeteria (which retained their designations after the policy changes), and a nurses' conference room and patient lounge (which were changed from smoking to nonsmoking areas). All measurements were taken using an Ecolyzer (Model 2000), which was calibrated daily using a standard carrier gas. Readings were recorded in parts per million. At each assessment period four sets of readings were taken over the course of two days in order to sample high and low usage of the areas. Each set consisted of 15 readings separated by a 20-second interval. This yielded a total of 60 ambient air samples per location per assessment time. The Ecolyzer was placed in the same location for all readings.

RESULTS

The complete presentation of statistical procedures and results of the interview data have been presented elsewhere.[25] They will be reviewed here in brief.

Response Rates. The initial response rate for the cohort was 99% and 98% for the policy hospital and comparison hospital, respectively. Twenty-nine percent of the respondents at each hospital identified themselves as current smokers. Unfortunately, there was attrition from the longitudinal sample because of refusal to be interviewed, staff turnover, and vacations. Only 71% of the policy and 63% of the comparison cohort were reinterviewed at the final assessment period. Furthermore, the attrition left us with a biased sample. At the policy hospital, smokers were significantly more likely than ex-smokers and nonsmokers to refuse to be re-interviewed (χ^2 (2 degrees of freedom) = 11.84, $p < 0.003$), but at the comparison hospital the rate of refusal was unrelated to smoking status. The biased attrition from the cohort at the policy hospital makes it difficult to interpret the results of the longitudinal analysis. The smokers who remained in the sample may well have been those most accepting of the restrictive policy. For that reason, only the results of analyses of the cross-sectional samples of smokers are presented.

The refusal rate for the six cross-sectional samples (two hospitals times three assessment periods) ranged from 2% to 9%. Respondents were similar to the employee populations from which they were sampled in sex (70% female), age (38 years), and occupational status (43% professional). Table I shows the number of respondents by smoking status for the two types of samples.

Approval/Acceptance. Results of analyses of approval and acceptance of the policy change appear in Table II. At the six-month post policy assessment, 95% of the policy hospital employees report-

TABLE I. Sample Sizes by Hospital, Time Period, and Smoking Status

Hospital	Smoking Status	Time Before	 Six Months	 12 Months
Cross-sectional sample				
Policy	Smokers	28	14	22
	Ex and nonsmokers	54	66	88
	Total	82	80	110
Comparison	Smokers	23	18	26
	Ex and nonsmokers	60	58	78
	Total	83	76	104
Cohort sample				
Policy	Smokers	25	21	16
	Ex and nonsmokers	59	50	44
	Total	84	71	60
Comparison	Smokers	24	21	16
	Ex and nonsmokers	59	50	36
	Total	83	76	52

TABLE II. Effectiveness of Implementation of Policy Change By Time and Smoking Status

	Six Months			12 Months		
	Smokers	Nonsmokers	Difference	Smokers	Nonsmokers	Difference
N	13	66	—	21	85	—
Aware of policy change	100%	94%	−6 (−16,4)*	—	—	—
Correctly identify smoking areas	100%	95%	−5 (−15,5)	95%	93%	−2 (−16,12)
Approve of policy	69%	92%	23 (−8,54)	67%	98%	31 (8,54)
Aware of more than "a few" instances of noncompliance	8%	12%	4 (−17,25)	10%	18%	8 (−10,26)
Performance improved	—	—	—	0%	21%	21 (9,33)
Performance deteriorated	—	—	—	19%	0%	−19 (−39,1)

* 95% confidence interval for the difference in proportions; statistical analyses in tables are for the cross-sectional sample only.

ed awareness of the policy, and almost all of these respondents could name one or both of the places where smoking was permitted. A large majority of respondents endorsed the policy and felt that the policy change was a good idea. The proportion of smokers who approved of the policy was lower than the proportion of nonsmokers who approved.

At the six- and 12-month assessments more than half of the respondents reported being aware of at least one instance of noncompliance with the policy. A much smaller percentage of subjects reported more than "a few instances of noncompliance."

At the 12-month follow-up, policy hospital employees were asked to evaluate the effect of the policy on their work performance. Although the majority of both smokers and nonsmokers felt that the policy had had no effect on their work performance, significantly more nonsmokers compared to smokers felt that their work had improved. In addition, significantly more smokers compared to nonsmokers felt that their performance had deteriorated. Nonsmokers attributed their increase in performance to improved concentration as a result of better air quality.

Air Quality. Results indicated a significant increase in CO levels in both the cafeteria smoking area and the adjacent nonsmoking area following the policy change. However, these increases were no longer evident at the six-month follow-up when levels had returned to their original value (Fig 1).

In the two areas that changed from smoking to nonsmoking (nurses' report room and patient lounge), there were no significant changes in ambient CO at any point in time. It should be noted that these rooms were often not fully occupied, and thus the sampling method may not have been sensitive enough to detect peak smoking periods prior to implementation of the policy change.

Subjective indicators of air quality demonstrated improvement due to the restrictive policy. At the 12-month assessment, respondents at both hospitals were asked to identify the locations where they had been bothered by smoke. The results suggest that the restrictive policy was successful in eliminating most of the environmental smoke from offices and staff lounges (Table III). In these areas, policy hospital employees were significantly less likely to report being bothered by smoke than staff at the comparison hospital. The policy did not affect air quality in the lavatories—the area where unauthorized smoking was reported—and it increased nonsmokers' discomfort in the vending machine area in the policy hospital. This area had become one of the smoking areas after the policy implementation, and all cafeteria users had to pass through it in order to get to the serving area.

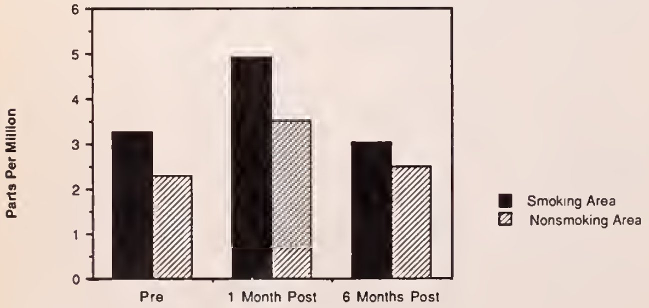

FIGURE 1. Ambient CO levels in cafeteria.

Nonsmokers' Attitudes and Organizational Norms. More than 80% of nonsmokers at both hospitals endorsed the statement, "Smoke affects the health of nonsmokers," and the strength of this belief did not change over time. At the 12-month assessment, approximately half of the nonsmokers at each hospital indicated that they would respond directly and assertively if they were bothered by a coworker's tobacco smoke. A change in nonsmokers' attitudes toward smoking at work might have been detected by the smokers. At each assessment period smokers at both hospitals were asked to indicate the extent to which they agreed with the statements, "My coworkers disapprove of my smoking." Analyses indicated that smokers at both hospitals were equally likely to agree that their coworkers disapproved of their smoking, and this belief did not change over time.

Effect on Smokers' Attitudes and Behavior. Very few smokers in the sample quit smoking during the year following the policy change. Among those individuals who reported having been smokers at the time of the baseline data collection, 7% at the policy hospital and 11% at the comparison hospital reported quitting by one year later. The implementation of the restrictive policy did affect smokers' intentions to quit smoking (Table IV). By the six-month assessment there was a substantial increase over baseline at the policy hospital in smokers' plans to quit smoking during the subsequent six months. However, by the 12-month assessment there was a significant reduction in the number of smokers intending to quit. In contrast, there was a trend toward increased plans to quit smoking at the comparison hospital at the 12-month assessment, compared to the six-month rate. Log linear analysis showed the hospital by time interaction to be significant beyond the 0.05 level.

At the policy hospital, there was a significant reduction in the number of cigarettes smoked at work following policy implementation, and this reduction was sustained through the 12-month assessment (Table V). The total number of cigarettes smoked per day declined at both hospitals, and hence appears not to have been affected by the policy change.

DISCUSSION

The restrictive smoking policy was implemented without causing undue conflict at the worksite. The majority of both smokers and nonsmokers approved of the policy. Compliance problems were minimal. While some smokers reported performance deficits due to the new policy, four

TABLE III. Percentage of Nonsmokers Reporting Being Bothered by Smoke at 12-Month Assessment

	Policy Hospital	Comparison Hospital	Significance
Offices	5%	25%	$p < 0.001$
Staff lounges	10%	30%	$p < 0.001$
Lavatories	18%	16%	Not significant
Vending machine area	36%	4%	$p < 0.001$

TABLE IV. Smokers' Attitudes and Behaviors by Hospital and Time*

		Time		
Variable	**Hospital**	**Before**	**Six Months**	**12 Months**
Percent reporting coworkers' disapproval of smoking	Policy	54	46	59
	Comparison	45	41	52
Percent perceiving health risk of environmental tobacco smoke	Policy	71	85	59
	Comparison	62	59	76
Percent considering quitting in next six months†	Policy	74	91	27
	Comparison	62	53	65
Percent who made an attempt to quit in prior six months	Policy	4	14	0
	Comparison	30	22	19

* Adapted from Biener L, Abrams DB, Follick MJ, Dean L: A comparative evaluation of a restrictive smoking policy in a general hospital. *Am J Pub Health* (in press).

† Hospital by time interaction significant at 0.05 level. No significant effects for other variables.

times as many nonsmokers felt that their performance had improved. Other studies have also documented high levels of acceptance and approval of restrictive policies, even among smokers.[22,26] In only one published report did a majority of the smokers disapprove of smoking restrictions.[24] In that case, the policy created a total ban on smoking, and the implementation strategy apparently left many of the smokers confused about their role in the decision-making process. Future studies should attempt to document actual changes in productivity, since such data would make arguments for smoke-free work areas more persuasive.

The restrictive policy did not seem to have an impact on nonsmokers' attitudes about health risks of environmental tobacco smoke, nor did it make them more likely to express their disapproval to smokers. Nonsmokers at both hospitals indicated a high level of concern about the effects of ETS. The societal changes in attitudes toward smoking may be more potent than changes in individual worksites. One might have expected that with the authority of a restrictive policy behind them, nonsmokers would be more likely to complain overtly to coworkers whose smoking was bothering them. That our data failed to demonstrate any increase in assertiveness may be due to the fact that smokers were not violating the rules to any significant degree, and that nonsmokers felt that they therefore had no right to complain. Being less bothered by smoke after the policy was implemented, nonsmokers may have had fewer opportunities to express their disapproval to smoking coworkers. As smoking becomes an activity that is carried out in segregated areas, smokers may experience less, rather than more, pressure to quit from nonsmokers.

TABLE V. Daily Smoking Rate and 95% Confidence Intervals by Hospital and Time*

Variable	Hospital	Before	Six Months	12 Months
Mean cigarettes smoked per day at work†	Policy	8.4 ± 2.5	3.5 ± 1.5	4.5 ± 1.4
	(N)	(28)	(16)	(23)
	Comparison	7.6 ± 2.6	7.4 ± 3.1	6.9 ± 2.0
	(N)	(21)	(21)	(29)
Mean cigarettes smoked per day at home	Policy	12.8 ± 2.5	9.4 ± 3.9	10.6 ± 3.1
	(N)	(28)	(16)	(23)
	Comparison	13.3 ± 4.1	7.7 ± 3.4	9.2 ± 2.6
	(N)	(21)	(21)	(29)
Mean cigarettes smoked on nonwork days	Policy	19.3 ± 2.9	13.2 ± 4.6	16.3 ± 3.6
	(N)	(28)	(16)	(23)
	Comparison	19.3 ± 5.8	12.5 ± 4.8	13.9 ± 4.1
	(N)	(21)	(21)	(29)

* Analyses of mean cigarettes smoked include scores of zero for three individuals at the policy hospital and six at the comparison hospital who reported having quit smoking since the pre-policy assessment. Unadjusted means are presented. (From Biener L, Abrams DB, Follick MJ, Dean L: A comparative evaluation of a restrictive smoking policy in a general hospital. *Am J Pub Health* (in press).)

† Hospital by time interaction significant at 0.05 level. No significant effects for other variables.

The policy change did have an impact on smokers' attitudes and behaviors, yet not as uniform an effect as clean air advocates might hope. The significant increase in CO levels in the cafeteria smoking section as well as in the adjacent nonsmoking area suggest that there was a rather dramatic change in smokers' behavior immediately after the policy was implemented. Smokers were apparently taking their breaks in the one area where smoking was still permitted, and the smoke drifted into the larger nonsmoking area. The fact that CO returned to baseline levels by six months after the policy change suggests that smokers found other ways to adapt. Although the objective indicators of air quality do not show reductions in ambient carbon monoxide in areas that changed from smoking to nonsmoking after policy implementation, this may be due to the lack of sensitivity of the CO measure or inadequate frequency of sampling. Employees reported significantly less annoyance from tobacco smoke as long as one year after the policy took effect. The average smoker also reduced the self-reported number of cigarettes smoked at work, and was able to refrain from smoking in the work areas where smoking was prohibited.

By the 12-month assessment, fewer smokers at the policy hospital reported plans to quit smoking than at the baseline hospital. It is likely that when smokers initially learned of the policy change they felt that smoking cessation would be necessary in order to continue functioning in their job. The results suggest that over time smokers may have adapted their smoking behavior to the policy restrictions, rather than using the restrictions as a catalyst for change.

The increase in intentions to quit at the comparison hospital detected at the 12-month follow-up may have been due to the fact that a large hospital in the area adopted a smoking ban around this time, and the comparison hospital's administrators started discussing a policy change of their own. The trends from this study suggest that the period prior to the implementation of a policy change may be the most opportune time to target smokers for cessation programs.

Future studies should compare the relative impact of a restrictive policy versus a total smoking ban. If adaptation of one's smoking patterns is less possible, a greater effect on cessation might be seen. Future studies should also carefully investigate the effect of anticipated policy change on smokers' motivation to quit. Different stages of readiness for change have been identified,[27] and this may make it possible to predict the impact that policy change will have on individual smokers' cessation efforts. For example, individuals who are in the "precontemplation" stage are not yet interested in taking action to stop smoking.[28] It is possible that a smoking ban may increase the precontemplative smoker's motivation to initiate change.

The results from this study illustrate the importance of investigating multiple outcomes when evaluating worksite smoking policies. Although policy implementation was somewhat successful, there were some areas in which the restrictive policy did not yield expected results. Careful evaluation of the impact of worksite smoking policies is crucial to the development of a comprehensive, theory-driven model that can be used to guide development of optimal approaches to worksite health promotion. It is likely that no single event, such as policy change alone, will be sufficient to modify smoking prevalence. However, over time, changes in environmental controls, in social norms, and in individual behavioral coping skills and patterns of smoking can all accumulate to accelerate reductions in smoking prevalence. Thus, sustained and multifaceted programs at the workplace, including policy changes, monetary incentives or competitions, as well as cessation programs aimed at individuals, could result in additive or synergistic effects on smoking outcomes. Policy change should be included as one important component of such comprehensive approaches but it should not be viewed as another panacea.[5,28]

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Implementing nonsmoking policies in the private sector and assessing their effects

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ABSTRACT. Nonsmoking policies are being implemented increasingly in worksites across the country. This cross-sectional study examined attitudes toward worksite nonsmoking policies and worksite norms about smoking and cessation. Data were collected by telephone interview from 711 smokers and recent ex-smokers employed in seven worksites in Bloomington, Minnesota, in 1986. The findings indicated widespread support for nonsmoking policies even among smokers. Support for nonsmoking policies was especially high among respondents interested in quitting smoking, those concerned about the health effects of smoking, those reporting high coworker support for prior cessation attempts, and those working with a high proportion of nonsmokers. Support for nonsmoking policies was also correlated with smoking cessation in the 18 months prior to the survey. The clustering of these social norms is indicative of a worksite "culture" that may either support nonsmoking or inhibit cessation.

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Public policies promoting nonsmoking have been heralded as the trend of the future. With the surgeon general's declaration of the goal of a smoke-free society by the year 2000,[1] and with mounting concern about the limited success of smoking cessation efforts aimed at the individual,[2,3] policy alternatives have received increasing attention. In addition to reducing nonsmokers' exposure to secondhand smoke, public nonsmoking policies are likely to encourage cessation by diminishing the social acceptability of smoking and enhancing public support for nonsmoking.

One site for the implementation of such policies is the workplace. Until recently, workplace policies were instituted primarily to limit danger to products and equipment.[4] Employers are now realizing other advantages of nonsmoking policies, including enhancement of the company's public image; a cost-free employee benefit for the majority of employees who are nonsmokers; reduced expenses related to absenteeism, disability, and health care costs; and protection from legal suits supporting nonsmokers' rights.[5,6] Indeed, health issues are becoming primary motivators for adoption of nonsmoking policies.[7] In the recent National Survey of Worksite Health Promotion Activities,[8] three fourths of worksites with 50 or more employees reported having a formal nonsmoking policy. The major reasons for adopting a policy included protection of the health of nonsmokers and compliance with regulations. Regardless of a company's motivation to implement a nonsmoking policy, the social norms defined by restricting smoking at work are likely to shape employee attitudes and behaviors related to smoking.

This cross-sectional study examined smokers' and recent ex-smokers' attitudes toward worksite nonsmoking policies, and the relationships of these attitudes to social norms about smoking and to smoking cessation. We hypothesized that smokers most interested in quitting and most concerned about the health risks of smoking would be most likely to favor worksite nonsmoking policies. Also, we proposed that perceiving the work environment as supporting nonsmoking would be correlated with support for worksite restrictions on smoking. Finally, it was hypothesized that smoking cessation would be positively related to support for worksite nonsmoking policies.

METHODS

Study Population and Data Collection. These data were collected as part of a smoking cessation intervention study conducted between 1984 and 1986.[9–11] Ten worksites in Bloomington, Minnesota, were selected in early 1984 on the basis of size, proportion of full-time employees, and recent participation in similar health promotion programs. Companies were excluded that had fewer than 100 or more than 1,200 employees, had a substantial number of part-time employees who would not be exposed to onsite intervention programs, or had offered smoking cessation programs recently to employees.

A baseline survey was conducted in the ten companies in the spring of 1984 to assess the smoking status of all employees at the study's onset. Eighty percent of employees responded to this one-page self-administered questionnaire. The smoking prevalence ranged from 15% to 53% at these ten sites. Two of the sites were excluded from the intervention study due to a low prevalence of smoking or the initiation of other smoking cessation programs. The remaining eight companies were randomly assigned to intervention and delayed intervention control groups in July 1984. The intervention program, consisting of onsite cessation programs, classes for nonsmokers on ways to help smokers quit, and consultation with employers on establishing nonsmoking policies, was offered in October–November 1984 in the initial inter-

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vention group and in May–June 1985 in the delayed intervention companies.

Analyses presented here are from data collected from 711 smokers who contributed to the baseline data and who continued to be employed in seven companies (Table I) in June–July 1986, about two years after the baseline survey. Employees of the eighth company were lost to follow-up due to the company's closing. At the time of the survey, all companies had received the intervention approximately 12 or 18 months earlier.

Table I includes smoking prevalence estimates from data collected in the 1984 baseline survey of all employees. The worksites ranged in size from 120 to 1,050 employees and represented diverse types of businesses. The overall prevalence of smoking in these companies, 35%, suggests that employees in these companies are generally representative of the broader population. Employees were evenly distributed across three occupational groups, including professionals and managers, clerical and sales workers, and blue collar workers.[10]

Measures. Attitudes toward nonsmoking policies at the worksite were assessed by six items. To examine the appropriateness of combining these items in a scale, the items were analyzed using principal components factor analysis; orthogonal rotation was used to assure the independence of the factors formed. Two discrete factors were developed, and factor-based scores were used to form each index. The distribution of responses to items included in the two indices is presented in Table II. Each scale ranged from zero to nine, with a high score showing support for policies restricting or prohibiting smoking. The "restrict smoking" scale reflects respondents' attitudes toward policies that restrict smoking, while the "prohibit smoking" scale includes attitudes toward policies that ban smoking at the worksite. Although these indices are correlated ($r = 0.34$, $p < 0.001$), they reflect independent attitudes toward worksite nonsmoking policies.

Worksite social norms about smoking are represented by two variables. "Coworker nonsmoking" was measured among all respondents by the following question: "Of the people you work with every day, how many would you say are smokers? Would you say almost all (1), more than half (2), about half (3), less than half (4), or almost none (5)." Respondents who had tried to quit smoking while working for their current employer (N = 441) were asked a series of questions about the support their coworkers offered during prior attempts to quit. Based on results from factor analysis, four items, each ranging from zero to three, were summed to form the index "perceived coworker support of quitting." The items in the index were as follows: "How often have your coworkers encouraged you to quit smoking?" "How often have your coworkers complimented your quit attempts?" "How often have they helped you think of substitutes for smoking?" and "How often have they refrained from smoking in your presence when you were trying to quit?"

"Concern about the health hazards of smoking" was assessed among all respondents by the extent to which respondents agreed with two items: "Smokers can escape most of the bad health effects of smoking just by quitting," and "Smoke from someone else's cigarettes is unhealthy for nonsmokers." Scores on each item, which ranged from one to four, were summed to form the index; a high score reflected high concern about the health effects of smoking.

TABLE I. Study Sites

Worksite	Number of Employees	Smoking Prevalence
Photographic equipment and sales	120	35%
Telecommunications	1,050	37%
Metal casting	560	41%
Utility and truck equipment	155	44%
General industrial machinery (excluding plant)	758	21%
Machinery sales and service	257	31%
Screw machine products	230	53%
Total	3,130	35%

TABLE II. Attitudes Toward Nonsmoking Policies

	Percent Agree
Items measuring the "restrict smoking" scale	
Employers should establish separate smoking and nonsmoking areas in the workplace. (N = 704)	80%
Employers should provide programs to help smokers quit smoking. (N = 707)	75%
There is strong support for restricting smoking at the place I work. (N = 686)	64%
Items measuring the "prohibit smoking" scale	
People should not be allowed to smoke anywhere at work. (N = 705)	9%
Cigarettes should not be sold at work. (N = 701)	30%
It would be easier for smokers to quit smoking if they could not smoke at work. (N = 703)	58%

Respondents still smoking at the time of this follow-up survey (N = 567) were asked about their smoking habits and attitudes toward quitting. "Interest in quitting smoking" was indicated by the question, "Currently, how interested are you in quitting: On a scale of zero to ten, where zero means not at all interested and ten means very interested, how interested are you in quitting?" Continuing smokers were also asked if they had tried to quit smoking in the last year ("quit attempt").

Smoking cessation also was examined retrospectively. As noted above, respondents were selected from persons who identified themselves as smokers at the baseline survey. "Smoking cessation" was determined by self-reported smoking status at the time of the follow-up survey.

Statistical Analyses. Differences by age, sex, education, and occupation were examined in mean values of the "restrict smoking" and "prohibit smoking" scales using analysis of variance. A series of multiple regression analyses was conducted to examine the relationship of support for nonsmoking policies to coworker support of quitting, concern about the health effects of smoking, and interest in quitting. These analyses were controlled for age, sex, and education. Controlling for these same covariates, analysis of covariance was used to examine differences in the mean values of the two scales by worksite, coworker nonsmoking, coworker requests not to smoke, existence of a current policy, cessation attempts (among smokers only), and smoking cessation since the baseline survey.

RESULTS

The respondents included 260 women and 451 men. By occupation, 41% were employed in blue collar positions, 46% in clerical and sales work, and 12% as professionals and managers. Only 8% had less than a high school education, and 16% had at least a college degree. Twenty-nine percent were under 30 years of age, 39% were between 30 and 39 years, and 20% were between 40 and 49. Twenty percent of the 711 respondents had quit smoking since the baseline survey conducted two years earlier (N = 144).

Table II presents the distribution of responses to the items measuring support for restricting and prohibiting smoking. The majority of these smokers and recent ex-smokers supported separate smoking and nonsmoking areas at the worksite, were supportive of smoking cessation programs being offered at the worksite, and felt that there was strong support for restricting smoking at their place of employment. Respondents were less supportive of policies that would prohibit smoking. Very few respondents thought that smoking should be banned entirely from the workplace, although nearly a third of these baseline smokers agreed that cigarettes should not be sold at work, and more than half stated it would be easier for smokers to quit if they could not smoke at work.

Demographic differences in the "restrict smoking" and "prohibit smoking" scales were examined using analysis of variance. No differences were found in the "prohibit smoking" scale by age, sex, education, or occupation, while significant differences on all these characteristics were noted in support for policies restricting smoking. Women were significantly more supportive of restrictions on smoking than were men (5.6 vs 6.4; $p < 0.001$.) Support for restrictions on smoking diminished slightly with age ($p = 0.07$). Mean values of "restrict smoking" were highest among professionals and managers (6.3), followed by clerical and sales workers (6.1) and blue collar workers (5.6) ($p < 0.001$). Support for smoking restrictions also varied directly with education ($p < 0.005$). Subsequent analyses were controlled for age, sex, and education. Due to the strong positive relationship between education and occupation (chi-square = 285.0, $p < 0.001$), occupation is not used as an additional covariate.

Controlling for age, sex, and education, differences in support for restrictive nonsmoking policies were observed by worksite. The highest mean support for nonsmoking policies was shown in a telecommunications firm (mean = 6.6) and in a screw machine products manufacturing firm. Respondents from these two companies were also most likely to report restrictions on smoking where they do most of their work. Lowest support for restrictions was found in a machinery sales and service firm (mean = 4.3), which had smoking restrictions only in the cafeteria. No significant worksite differences were found in attitudes towards prohibiting smoking at work.

Table III presents results from a series of multiple regression analyses examining correlates of support for policies restricting and prohibiting smoking. Respondents most interested in quitting smoking and most concerned about the health effects of smoking showed the most support for both types of worksite nonsmoking policies. Support for restrictive or prohibitive policies also was high among those who reported strong coworker support for prior attempts to quit.

Analysis of covariance was used to examine the relationships of these attitudes toward nonsmoking policies to other worksite norms about smoking (Table IV). Respondents who work primarily with nonsmokers were most likely to favor policies restricting or prohibiting smoking at work. Persons whose coworkers have asked them not to smoke around them also are most supportive of both types of nonsmoking policies. Support for policies restricting smoking, but not for those prohibiting smoking, was highest among those reporting that smoking is already restricted in the place where they do most of their work.

Support for worksite nonsmoking policies was also positively related to smoking cessation, as presented in Table V. Support for smoking prohibitions and restrictions was highest among smokers who had attempted to quit in the last year, and among persons who had quit in the last two years compared to continuing smokers.

DISCUSSION

Contrary to concerns that smokers may raise opposition to nonsmoking policies, these findings suggest that smokers and recent ex-smokers favor restrictions on smoking at work and perceive similar attitudes among their coworkers. Other studies have also indicated that 80% or more of employed smokers support restrictions on smoking.[12-14] In fact, this study also indicates that support for nonsmoking was highest among those already affected by a policy. Although positive attitudes toward nonsmoking policies may precede such a policy, these data are more likely to confirm other reports suggesting that smokers adapt to such policies rather than leave the company, as is sometimes forecast.[12,14,15] With the diminishing social acceptability of smoking, smokers may find defined areas for smoking preferable to asking their coworkers for permission to smoke. Media discussions that educate smokers about the dangers of secondhand smoke may further increase smokers' willingness to limit their smoking in public areas. Smokers concerned with the health effects of their smoking, both for themselves and for the nonsmokers around them, were most supportive of nonsmoking policies.

As indicated in other studies,[16,17] support was stronger for restricting than prohibiting smoking. Yet the majority of these smokers and recent ex-smokers agreed that quitting smoking would be easier if they could not smoke at work. Smokers' perception of the utility of nonsmoking policies in cessation attempts was further supported by the findings that smokers most supportive of nonsmoking policies were those most interested in quitting and those most

TABLE III. Correlates of Favoring Smoking Restrictions*

Correlate	Restrict Smoking	Prohibit Smoking
Concern about health effects of smoking	0.17†	0.32†
Interest in quitting smoking	0.29†	0.42†
Coworker support of cessation	0.17†	0.16‡

* Adjusted for age, sex, and education using multiple regression analysis.
† $P < 0.0005$.
‡ $P < 0.005$.

TABLE IV. Differences in Support for Nonsmoking Policies by Coworker Smoking and Nonsmoking*

Item	Restrict Smoking	Prohibit Smoking
Of the people you work with every day, how many are smokers?		
More than half, almost all (22%)	5.54	2.65
About half (25%)	5.79	2.61
Less than half, almost none (52%)	6.11	3.44
	$p < 0.0001$	$p < 0.001$
How often have your coworkers asked you not to smoke around them?		
Ever (35%)	6.29	3.42
Never (65%)	5.73	2.90
	$p < 0.0001$	$p < 0.05$
Are people allowed to smoke in the area where you do most of your work?		
Yes (75%)	5.72	3.10
No (25%)	6.45	2.90
	$p < 0.0001$	NS†

* Mean values controlling for age, sex, and education using analysis of covariance.
† NS = not significant.

TABLE V. Differences in Support for Nonsmoking Policy by Smoking Cessation*

Item	Restrict Smoking	Prohibit Smoking
Cessation attempts (smokers only)		
Tried to quit in the last year (42%)	6.17	3.62
Did not try to quit in the last year (58%)	5.53 $p < 0.0001$	2.44 $p < 0.0001$
Smoking cessation		
Quit smoking within last two years		
Yes (20%)	6.57	3.81
No (80%)	5.74	2.85
	$p < 0.0001$	$p < 0.0001$

* Mean values controlling for age, sex, and education using analysis of covariance.

likely to attempt and succeed with cessation. Prior reports from this study also suggested a relationship between the social environment and cessation.[9] Controlling for age, sex, education, coworker nonsmoking, and treatment group, the predicted cessation rate for smokers whose coworkers never requested them not to smoke was 4.4% compared to 26.6% among those whose coworkers frequently asked them not to smoke (odds ratio = 6.05). Although the perception of past events such as coworker requests not to smoke may be influenced by later behaviors or attitudes, the magnitude of this difference strongly suggests that coworker norms influence success with cessation.

Indeed, these findings suggest that the worksite "culture" is characterized by a constellation of social norms that are highly correlated with support for nonsmoking policies. Support for worksite restrictions of smoking varied significantly by worksite. The strongest support for nonsmoking policies was observed among respondents who worked primarily with nonsmokers, whose coworkers requested them not to smoke, and who reported support from coworkers for their attempts at cessation. The clustering of these worksite characteristics has also been noted in earlier reports from this study, when we found significant worksite differences in the prevalence of coworker smoking, smokers' interest in quitting, perceived coworker discouragement of prior attempts to quit, and smokers' desire to seek formal assistance in future attempts to quit. Worksite mean values of desire to seek formal help with cessation increased with rising mean values of coworker discouragement of cessation, further supporting the concept that each worksite is its own "community" with distinct social norms.[11] These norms are likely to be altered by worksite policies to promote nonsmoking.

The rapidly rising prevalence of worksite nonsmoking policies underlines the importance of continued research to assess the effects of these policies and the effectiveness of strategies employed to implement them. A survey from the Bureau of National Affairs[18] found that 36% of companies surveyed currently have a nonsmoking policy, and an additional 23% are planning a policy or have one under consideration. Several studies indicate that nonsmoking policies are often followed by reported reductions in the number of cigarettes smokers consume, especially at work.[12,14,16] Research methods of many studies to date have left open the question of the relationship of nonsmoking policies to cessation.[19]

Despite the lack of evidence, it is often assumed that nonsmoking restrictions contribute to increased cessation and reduced tobacco consumption. The effects of nonsmoking policies on smoking behaviors would ideally be assessed in randomized studies in which the external factors that influence cessation could be controlled. Yet the feasibility of such studies is limited given the difficulties of randomizing sites to policy and no-policy conditions. In the absence of opportunities for controlled randomized studies, the impact of nonsmoking policies may also be assessed using nonrandomized sites for comparison or a series of case studies of worksites that adopt nonsmoking policies. Such studies could address a variety of important questions. Do smokers reduce the number of cigarettes smoked when a worksite nonsmoking policy is introduced, or do they compensate at home for reduced smoking at work? Do nonsmoking policies increase smokers' motivation to quit smoking? Do policies increase cessation only among smokers who were already planning to quit, resulting in a rapid increase in cessation that levels off soon after the policy is enacted? Does the policy reduce the social acceptability of smoking and thereby increase cessation over a longer time frame? Do policies have differential effects on the smoking behaviors of varying subgroups, such as those grouped by age or occupation? To address these questions, studies are needed to follow the impact of restrictive and prohibitive nonsmoking policies over varying time periods among diverse groups of workers.

Information is needed as well on other effects of nonsmoking policies. Company management may be particularly interested in the economic impacts of nonsmoking policies, including possible reductions in insurance or maintenance costs. In addition, improvements in air quality, a goal of many nonsmoking policies, may not be readily achieved by smoking restrictions without proper ventilation. Assessing such consequences of nonsmoking policies will be useful to companies that are planning policy changes.

The effectiveness of recommendations for implementing nonsmoking policies has also not been examined adequately. General guidelines for implementing nonsmoking policies have been suggested by a variety of sources.[8,18,20–22] Recommendations generally include ways to solicit top management support for nonsmoking policies, to assess potential employee concerns about a policy, to involve employees in decisions about the nature of the policy and its enforcement, and to communicate and enforce the policy. The applicability of these recommendations may vary depending on company characteristics. For example, employee committees may not be the most appropriate way of involving employees in small businesses, which employ substantial numbers of workers nationwide. For many manufacturing firms, the nature of production line work diminishes the feasibility of structured breaks away from the work station. The concerns of organized labor about the role of collective bargaining in the adoption of a nonsmoking policy must be included in the identification of alternative strategies for policy implementation in unionized worksites. Thus, the process of effective policy implementation in diverse work settings needs further explication.

In conclusion, the increasing prevalence of worksite nonsmoking policies is limiting smoking during substantial time periods for growing numbers of workers. The findings of this study point to the growing acceptability of such policies even among smokers. Smokers report that restrictions on smoking are likely to aid their attempts to quit. Such policies also appear to foster the development of a constellation of social norms that support cessation. Nonsmoking policies represent a potentially important public health tool aimed at modifying the environment as a means of influencing individual behavior. To date, most research in the smoking cessation field has focused on evaluating smoking cessation methodologies that target individuals, given the relative ease of identifying these effects. In contrast, assessing the effects of nonsmoking policies may require the use of innovative methods and a variety of approaches that might yield convergent views of the issue. Such studies will contribute to our understanding of the role of nonsmoking policies in shaping smoking behaviors and will provide useful guidance to the growing number of worksites that are considering adopting nonsmoking policies.

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The social epidemiology of smoking during pregnancy among Mexican women

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ABSTRACT. The association between maternal tobacco smoke exposure and hazards to reproduction has been investigated since the turn of the century. Numerous studies have demonstrated an increased risk to the developing fetus as a result of maternal smoking. This risk continues to manifest itself postnatally into childhood. The present study examined both risk factors for and consequences of smoking behavior among a sample of 50 pregnant women consecutively admitted to the obstetrics ward of the Tijuana General Hospital, Tijuana, Mexico. A questionnaire completed postpartum examined social, cultural, and economic variables for the mother, and physical variables of the newborn. Twenty-one subjects were smokers and 29 were nonsmokers. The number of previous pregnancies per subject was significantly higher in the group of smoking mothers. Overall, 86.2% of the nonsmoking mothers had three or fewer pregnancies versus 52.4% of the smoking mothers ($p \leq 0.005$). It was also noted that the smoking subjects tended to be light smokers (90% reported smoking ten or fewer cigarettes a day, with an average of 4.27 years of smoking history). No difference in knowledge of the harmful effects of smoking during pregnancy was observed between the two groups; however, the proportion of subjects whose mothers and siblings also smoked was significantly higher ($p \leq 0.005$ and $p \leq 0.01$, respectively) in the smoking groups. The results are discussed in terms of antismoking prevention and health promotion programs for the developing world.

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Approximately one third of all women in the United States of childbearing age smoke,[1] and, according to US government reports,[2] approximately 20–25% of all pregnant women smoke during pregnancy. Though no reliable estimates are available, far greater numbers are likely to be

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passive or involuntary smokers.

The relationship between tobacco smoke and hazards to reproduction has been investigated since the turn of the century.[3] However, animal studies that demonstrated an increase in resorption, still births, and low birthweight in association with smoke exposure were largely ignored as they were not felt to be applicable to humans.[4] During the 1950s, however, independent epidemiologic investigations documented the hazards of maternal smoking to fetal growth, demonstrating that they were dose related and independent of maternal characteristics.[5] Simpson,[6] for example, reported that the rate of premature births among smoking mothers increased with the number of cigarettes smoked per day.

Since that time, numerous studies have confirmed the relationship between maternal smoking and retardation and impaired fetal development.[7] Maternal smoking has been associated with increased complications,[8] spontaneous abortions,[9] the likelihood of malformations,[10] increased perinatal death rate,[11] neonatal morbidity,[12] low birth weight,[13] and reduction in long term growth.[14] Increasing attention has been focused on the health effects associated with passive or involuntary smoking.[15] Recent studies have revealed hazards similar to those associated with direct smoking, including lung cancer, respiratory illness, and cardiovascular disease.

However, social and behavioral risk factors for smoking during pregnancy have largely been ignored. This is especially the case for developing countries in the Third World. Given the current refocusing of the tobacco industry's marketing energies and strategies to the Third World in response to the gains of health promotion in the US and other developed countries, there is a need to detail the behavioral risk profile for smoking among females of reproductive age in developing nations. Such an effort will allow us to develop health promotion intervention strategies tailored to specific cultures, in order to combat this extension and elaboration of the tobacco industry's market. The present study is an examination of the risk factors for smoking among Mexican women from a sample in Tijuana, Mexico.

METHODS

A referral series study was carried out during the months of October and November, 1987, in the obstetrics ward of the Tijuana General Hospital. Tijuana is a rapidly growing border community, with a population estimated to be as high as 1.8 million. Although this growth is largely a result of the relative prosperity of this community, the great proportion of the residents live in conditions of marked poverty. The General Hospital provides services to the poorer elements of the population.

Fifty consecutive pregnant subjects admitted to the obstetrics ward were included in the study. All the subjects were admitted during labor. All subjects answered a questionnaire, given out by the interns on duty. The interns were previously instructed by one of the authors on the interviewing techniques to ensure uniform results.

The questionnaire was completed during the first 24 hours postpartum. It covered social, cultural, and economic variables of the mother, and physical characteristics of the newborn. Students t test was used to compare the results between smoking and nonsmoking groups. The Z distribution was used when testing the hypothesis on proportions. The level for statistical significance was $p \le 0.05$. BMDP statistical software was used where appropriate.

TABLE I. Demographic Variables Comparing Smoking and Nonsmoking Mexican Women at Delivery (N = 50)

Variable*	Smokers (N = 21)	Standard Deviation	Nonsmokers (N = 29)	Standard Deviation
Age (years)	24.5	6.2	22.4	6.0
Residence in Tijuana (years)	7.6	6.7	6.5	6.4
Housewife	81.6%	—	86.2%	—
Catholic	76.2%	—	79.3%	—
Married	28.6%	—	27.8%	—
Years in school				
6 or less	57.1%	—	65.5%	—
7 or more	42.9%	—	34.5%	—
Income (pesos/weekly)†				
50,000 or less	71.4%	—	72.4%	—
51,000 or more	28.6%	—	27.6%	—
House owner: yes	24.1%	—	28.6%	—
Water system: yes	42.9%	—	44.8%	—
Sewage system: yes	42.9%	—	55.2%	—
Never reads newspapers	76.2%	—	82.8%	—
Watches television	66.7%	—	79.3%	—

* No significant differences were found.
† 2,250 pesos = $1 US.

RESULTS

The group of nonsmokers included 29 subjects, and the smokers group included 21 subjects. Table I shows that there were no significant differences between the two groups regarding age, time of residence in Tijuana, occupation, religion, marital status, level of school education, or other socioeconomic variables pertinent to the milieu of Tijuana, Mexico.

Despite the lack of difference on such demographic variables, the number of previous pregnancies per subject was significantly higher in the group of smoking mothers (Table II). For 51.7% of the subjects in the nonsmoking group, this was the first pregnancy, whereas only 14.3% of the smoking mothers were pregnant for the first time ($p \le 0.001$). Overall, 86.2% of the nonsmoking mothers had had three or fewer pregnancies versus 52.4% of the smoking mothers ($p \le 0.005$). There were no significant differences between the groups regarding normal pregnancies and deliveries, number of abortions, cesarean sections, and previous stillbirths.

As can be seen in Table III, most of the smoking subjects were light smokers: 90.5% reported smoking ten or fewer cigarettes per day, with an average of 4.3 years of smoking history per individual smoker.

Table IV summarizes the physical characteristics of the newborns in both groups. There were no significant differences in weight,

TABLE II. Comparison of Reproductive History Among Smoking and Nonsmoking Mexican Women at Delivery (N = 50)

Variable	Smokers (N = 21)	Nonsmokers (N = 29)	Significance
Number of pregnancies			
1	14.3%	51.7%	$p \le 0.001$
3 or less	52.4%	86.2%	$p \le 0.005$
Normal pregnancies and deliveries	66.7%	79.3%	NS*
Previous abortions	9.6%	5.1%	NS
Previous cesarean section	11.0%	5.1%	NS
Previous stillbirths	4.1%	5.1%	NS

* NS = not significant.

TABLE III. Smoking History* at Delivery Among a Sample of Mexican Women (N = 21)

Smoking During Pregnancy		Daily Smoking	
Increases during pregnancy	23.8%	Minimal (5/day)	76.2%
Decreases during pregnancy	42.8%	Moderate (6–10/day)	14.3%
No modification	33.3%	Heavy (>10/day)	9.5%

* Lifetime smoking: mean = 4.3 years; standard deviation = 4.0.

length, cephalic perimeter, thoracic perimeter, or Apgar scores.

In Table V, one can see that the smoking habits of the father, husband, and friends of the subjects were not significantly different between the two groups. On the other hand, the proportion of subjects whose mothers and siblings smoked was significantly higher ($p \leq 0.005$ and $p \leq 0.01$, respectively), for the smoking group. It should be noted that of the nonsmoking mothers, 92.3% knew that smoking during pregnancy could be harmful for the fetus; 85.7% of the smoking mothers also knew that fact; the difference was not significant ($p \leq 0.23$).

DISCUSSION

The prevalence of smoking behavior among mothers during pregnancy in this study was 42% (21/50). This is five-fold greater than the reported prevalence for Mexican females in the international literature.[16] While the sample in the present study is too small to make any definitive conclusions, and the effect of referral bias in a clinical series cannot be ruled out, this result is an intriguing finding and calls for additional study. It may be that this elevated proportion is due to direct exposure to the US culture, where there is a high prevalence of smoking among females.

The smoking and nonsmoking groups in this analysis were very similar in age, time of residence in Tijuana, occupation, religion (some forbid smoking), marital status, and educational level. In terms of education, a high proportion of subjects in both groups had six or fewer years of formal education.

It should also be noted that almost all of the subjects came from a very low economic bracket: 71.4% of the smoking group and 72.4% of the nonsmoking group had a weekly income of 50,000 pesos or less (about $20 US). The difference was not statistically significant. This could be due to the fact that almost all of the subjects who utilize Tijuana General Hospital are very poor and their average incomes are similar, regardless of other factors. As was seen in Table I, both groups lack basic public services (running water, sewers) and only one of every four of them owned the house in which they lived.

TABLE V. Smoking Habits of Significant Others Comparing Smoking and Nonsmoking Mexican Women at Delivery (N = 50)

Variable	Smokers (N = 21)	Nonsmokers (N = 29)	Significance
Father smokes	55.0%	57.1%	NS*
Mother smokes	50.0%	14.8%	($p \leq 0.005$)
Husband smokes	66.6%	73.1%	NS
Siblings smoke	66.6%	34.6%	($p \leq 0.01$)
Friends smoke	76.2%	69.0%	NS

* NS = not significant.

Billions of dollars are spent every year worldwide to promote cigarette use. Targeting women directly as smokers began in the 1920s, starting a hallmark of cigarette advertising directed at women.[16] In the present study, we had anticipated differences in the exposure of the two groups to mass communication media. However, it was found that in both groups the proportion of subjects that never read a newspaper was very high (82.8% in nonsmokers and 79.3% in smokers; not significantly different). The majority of subjects in both groups reported that they watched television on a regular basis (79.3% in the nonsmoking group and 66.7% in the smoking group; not significantly different). Cigarette advertising is not prohibited in Mexico, and the tobacco industry is one of the principal advertisers on Mexican television.

In a poll conducted in 1980 in the United States, it was found that 47% of women respondents were unaware that smoking during pregnancy increases the risk of losing the baby before or during birth.[16] In the study presented here, the proportion of pregnant nonsmoking women who knew that smoking could be harmful to the fetus was higher (92.3%) than the proportion of smoking women (85.7%); however, the difference was not significant.

The proportion of normal pregnancies and deliveries was higher in the nonsmoking group (79.3% vs 66.7%), and the proportion of previous abortions and cesarean sections was higher in the smoking group (9.6% vs 5.1% and 11.0% vs 5.1%, respectively). This is as expected from the

TABLE IV. Physical Characteristics of the Newborn by Smoking and Nonsmoking Mexican Women at Delivery (N = 50)

	Smokers (N = 21)		Nonsmokers (N = 29)		
Variable	Mean	SD*	Mean	SD	Significance
Weight (g)	3,262.9	432	3,262.6	458	NS†
Apgar score					
1 minute	8.1	0.79	7.8	1.9	NS
5 minutes	9.2	0.70	8.9	1.7	NS
Length (cm)	49.4	2.4	49.5	2.9	NS
Cephalic perimeter (cm)	34.3	1.1	34.2	1.8	NS
Thoracic perimeter (cm)	33.9	1.5	33.8	2.4	NS

* Standard deviation.
† NS = not significant.

literature. The differences were not statistically significant.

In concordance with the literature, the weight of the infants was higher in the nonsmoking group (plus 22.69 g). However, here, too, the differences were not significant. One reason for this lack of significance, other than the small sample size, is possibly the relatively light smoking of the subjects. During pregnancy, 90.5% of the smokers smoked ten or fewer cigarettes a day, and 76.2% smoked five or fewer cigarettes a day. It is known from the literature that the nicotine effects of tobacco on the fetus are dose-related, with light smokers having a 54% and heavy smokers a 130% increased risk of delivering infants weighing less than 2,500 g. In this study, the proportion of infants with weight of 2,500 g or less at birth was 10.5% for the smoking group and 4.2% for the nonsmoking group; the difference was not significant ($p \leq 0.20$).

The prevalence of smoking habits was significantly higher among the mothers and siblings of the subjects of the smoking group than among those of the nonsmoking group (50% vs 14.8%, $p \leq 0.005$; and 66.7% vs 34.6%, $p \leq 0.01$, respectively). Prevalence of smoking for fathers and husbands was not significantly different between the groups. This may be due to the fact that in the Mexican culture the maternal figure is much more important as an influence on the family than the father, due to the larger amount of time the mother spends at home when the children are growing up. If maternal smoking is one of the most important behavioral risk factors in Mexico concerning children's future smoking habits, the implications for health promotion campaigns in this country are obvious. Equivalent results were found in another study performed by the authors among school children in Tijuana in 1986,[17] where maternal smoking was the most important influential factor on the children's future smoking habits.

In summary, the prevalence of smoking during pregnancy in this Mexican referral series sample was 42%. Economic and cultural factors did not influence the smoking habit of the subjects in the study, and the majority of the females in both groups knew that smoking during pregnancy could be harmful to the growing fetus. A significant difference in weight at birth between the two groups, a universal finding in the literature concerning smoking during pregnancy, was not noted. This may be due to the fact that the majority of smokers in the group were light smokers, or it may be due to the size of the sample. The number of previous pregnancies per subject was significantly higher in the group of smoking mothers. Finally, the prevalence of smoking among the mothers and siblings of the subjects of the smoking group was significantly higher than among those of the nonsmoking group.

Health promotion campaigns aimed at tobacco use in Mexico and other developing nations will need such information if they are to begin to combat the marketing strategies of the tobacco corporations. Smoking among men, women, and children in developing countries represents a dangerous and growing threat to populations that can scarcely afford additional health problems. The proliferation of tobacco use in these areas is largely a result of aggressive marketing strategies developed by the Western industry. Aggressive intervention studies as well as further epidemiologic research will help to control this problem.

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Trends in the adoption of smoking restrictions in public places and worksites

NANCY A. RIGOTTI, MD

There is a rapid and profound change underway in the role of tobacco smoking in American society. A wave of social action in the public and private sectors is redefining where and when it is acceptable to smoke. Over the past 15 years, smoking in public places has been increasingly regulated by government action, largely by legislation at state and local levels. More recently, in the private sector, a growing number of businesses have adopted workplace smoking restrictions. A smaller number of employers have taken stronger action, banning smoking entirely or preferentially hiring nonsmokers. Smoking restrictions have also appeared in restaurants, hotels and motels, health care facilities, and transportation vehicles.

The development and implementation of smoking restrictions have generated discussion and, occasionally, controversy. Their impact on air quality and the behavior of smokers is a matter of considerable interest, particularly to individuals concerned about the health and economic costs of cigarette smoking.

FORCES FUELING THE TREND TOWARD SMOKING RESTRICTIONS

The recent changes in where smoking is permitted are the result of two interrelated forces: growing evidence about the health risks of passive (involuntary) smoking and changing public attitudes about the social acceptability of smoking.[1] Information about the health consequences of involuntary smoking has been accumulating for more than a decade, providing a strong rationale for policymakers' actions to regulate smoking. However, until recently, the evidence was regarded by some scientists as incomplete or controversial. In 1986, evidence about the health risks of involuntary smoking was independently evaluated by the National Academy of Sciences and the US surgeon general. They reached substantially similar conclusions in reports issued in November and December 1986.[2,3] The surgeon general concluded the following:

> (1) Involuntary smoking is the cause of disease, including lung cancer, in healthy nonsmokers.
>
> (2) The children of parents who smoke, compared to the children of nonsmoking parents, have an increased frequency of respiratory infections, increased respiratory symptoms, and slightly smaller rates of increase in lung function as the lung matures.
>
> (3) The simple separation of smokers and nonsmokers within the same indoor airspace can reduce, but does not eliminate, exposure to environmental tobacco smoke.

These reports indicated a growing scientific consensus about the health risks of involuntary smoking. They provided policymakers with a public health issue to address, and attracted broad public attention via the news media.

However, data from opinion polls indicate that negative public attitudes about tobacco smoking had begun to develop long before the health risks to nonsmokers were demonstrated. These negative attitudes, an important force supporting the growth in smoking restrictions, can be detected as early as 1964, shortly after the first surgeon general's report focused public attention on the health hazards of smoking. Public opinion polls since 1964 have documented the shift in attitudes about the acceptability of smoking.

The four Adult Use of Tobacco surveys, a series of nationwide surveys conducted between 1964 and 1975, measured attitudes in the decade after the health hazards of smoking were first widely appreciated.[4-6] As early as the first survey in 1964, a majority of nonsmokers agreed with these statements: "It is annoying to be near a person who is smoking cigarettes," and "Smoking should be allowed in fewer places than it is now." By 1970, a majority of both smokers and nonsmokers agreed with these statements. By 1975, a majority of smokers agreed with the idea of further restricting smoking, suggesting that there was public support for restricting smoking well before the first comprehensive Clean Indoor Air Act was passed in Minnesota in 1975. As early as 1973, 73% of the nonsmokers in a Minnesota poll felt that they had the right to a smoke-free environment.[7] Negative attitudes toward smoking in public places have continued to spread in the 1980s. Two nationwide Gallup polls, commissioned by the American

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Lung Association in 1983 and 1985, found that an increasing majority of adults, including current smokers, agree that smokers should refrain from smoking in the presence of nonsmokers[8,9] (Table I). A 1987 Gallup poll found, for the first time, that a majority of all adults (55%) favored a complete ban on smoking in all public places. This was supported by 69% of nonsmokers but only 25% of smokers.[10]

Opinion polls document a similar pattern of increasing support for smoking restrictions in the workplace. Support for an employer's right to restrict smoking to a designated area at work grew from 52% to 61% during the 1970s, according to Roper polls performed in 1976 and 1978,[11] and continued to increase in the 1980s. A 1985 Gallup poll found that 79% of adults in the United States, including 76% of smokers, favored restricting workplace smoking to designated areas, and an additional 8% favored a total workplace smoking ban. Only 12% of respondents (19% of smokers) opposed any worksite smoking policy[9] (Table I).

These survey data indicate that Americans favor a higher level of restriction on smoking than currently exists. They also demonstrate that public support for smoking restrictions preceded rather than followed both the laws restricting smoking and most of the evidence about the health consequences of involuntary smoking.[1] Negative attitudes about smoking presumably contributed to the development of laws and regulations about smoking in public. At the same time, publicity surrounding the adoption of smoking policies may have increased public awareness of the hazards of passive smoke, thereby decreasing the acceptability of smoking in public. The development of negative attitudes toward cigarette smoke before significant research findings about the hazards of involuntary smoking suggests that these attitudes are more broadly based. They may reflect a dislike of smoke exposure in addition to concern about recently publicized health hazards.

RESTRICTIONS ON SMOKING IN PUBLIC PLACES

Smoking in public places is regulated primarily by government actions. In general, state and local governments have been more active than the federal government. At the federal level, smoking has, until recently, been addressed by agency regulation rather than congressional action. Federal agencies currently regulate smoking in airplanes and on interstate buses and trains. The first successful congressional legislation occurred in 1987, when Congress banned smoking on domestic commercial airline flights scheduled for up to two hours' duration.

At the state level, cigarette smoking has been regulated to a limited degree by state laws for nearly a century. The goal of this early legislation was to prevent fires or explosions by restricting smoking in such places as stables or factories. A second form of early state legislation was the result of a moral crusade against cigarettes coincident with the moral crusade against alcohol that emerged at the turn of the century.[12,13] Its goal was a total ban on cigarettes, which were blamed for social evils and physical ills, based largely on unfounded claims. At the peak of the movement, cigarettes were banned in a dozen states, primarily in the midwest.[13,14] Enforcing the regulations proved controversial, and all state laws banning smoking were repealed by 1927. The handful of remaining state laws regulating smoking were limited in scope.

By the early 1970s, a new form of smoking legislation emerged, developed by the new nonsmokers' rights movement. The specific intent of these Clean Indoor Air Acts was the safety and comfort of nonsmokers. These laws covered a broader range of places and extended for the first time to privately owned facilities. The language became more restrictive, moving from permitting a nonsmoking section, to requiring one, to making nonsmoking the principal or assumed condition. The landmark event was the passage of the Minnesota Clean Indoor Air Act in 1975. This was the first piece of legislation to restrict smoking in a range of public places and to do so for health concerns. It became a model for other state statutes, and the pace of new legislation increased.

The number of states that restrict smoking in at least some public places has continued to increase (Fig 1). By the end of 1985, smoking was regulated in at least one public place in 41 states and the District of Columbia,

TABLE I. Survey of Attitudes Towards Smoking*

Should smokers refrain from smoking in the presence of nonsmokers?

	Agree		Disagree		No Opinion	
	'83	'85	'83	'85	'83	'85
Current smokers	55%	62%	39%	37%	6%	1%
Nonsmokers	82%	85%	14%	15%	4%	0
Former smokers	70%	78%	22%	22%	8%	†
All adults	69%	75%	25%	24%	6%	1%

Should companies have a policy on smoking at work?

	Assign certain areas for smoking	Totally ban smoking at work	No company policy	No opinion
Current smokers	76%	4%	19%	1%
Nonsmokers	80%	12%	6%	2%
Former smokers	80%	9%	10%	1%
All adults	79%	8%	12%	1%

* Survey by the Gallup Organization, Inc. Source: American Lung Association, 1983, 1985.
† Less than ½ of 1%.

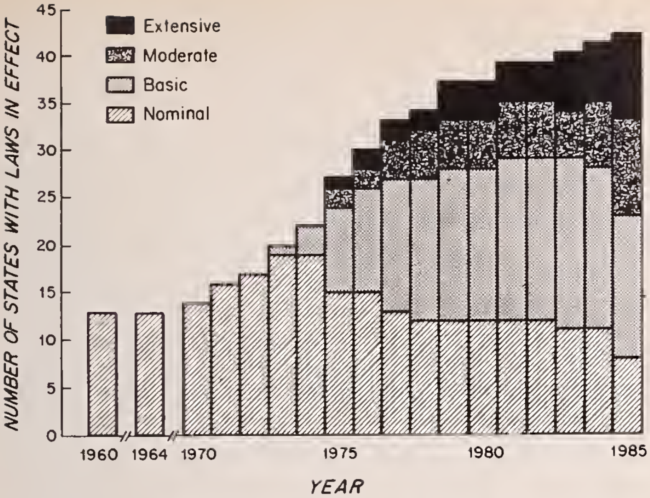

FIGURE 1. Prevalence and content of state laws restricting smoking in public places, 1960–1985. KEY: Extensive = law regulates smoking in private sector workplaces; Moderate = law regulates smoking in restaurants, but not workplaces; Basic = law regulates smoking in four or more public places, excluding restaurants and workplaces; Nominal = law restricts smoking in from one to three public places, excluding restaurants and workplaces. (Source: US Dept of Health and Human Services, *The Health Consequences of Involuntary Smoking: A Report of the Surgeon General*, 1986.)

leaving only nine states with no smoking restrictions. Whereas in 1970, only 8% of the US population lived in states where smoking regulations were in effect, by 1985 nearly 80% of the US population lived in states with some smoking regulation.[1] Not only the prevalence but also the comprehensiveness of state laws has increased. This was demonstrated in an analysis of the content of state laws, using an index which categorized the comprehensiveness of a state law based on the number of public places in which it restricted smoking.[1] As shown in Figure 1, the proportion of state laws in the more restrictive categories has increased over time.

The comprehensiveness of state laws varies widely. In 1986, state laws most often restricted smoking in public transportation (35 states), hospitals (33 states), elevators (31 states), indoor cultural or recreational facilities (29 states), schools (27 states), public meeting rooms (21 states), and libraries (19 states). Eighteen states restricted smoking in retail stores and in restaurants.[1] The prevalence and comprehensiveness of state laws varies by geographic region (Fig 2). There are fewer state laws and less restrictive state laws in southern states, compared with the rest of the US. Most of the states without smoking legislation are concentrated in the Southeast, and they include three of the six major tobacco producing states (North Carolina, Virginia, and Tennessee).

In the 1980s, legislative restrictions on smoking spread from states to local communities. The hallmark event was the passage in 1983 of San Francisco's ordinance restricting smoking in public places and at the worksite. It withstood a tobacco industry-sponsored public referendum after passage by city government and is currently in effect. Comprehensive legislation in other communities, notably in California, followed. As of April 1988, 125 California cities and counties had passed smoking ordinances, including 118 that required no-smoking sections in restaurants and 117 that restricted smoking in worksites.[15] Two groups that have recently monitored the extent of this trend nationwide (Americans for Nonsmokers' Rights and the Tobacco Free America Project) reported that over 320 cities and counties have passed ordinances restricting smoking in public. In general, local smoking ordinances are more comprehensive than corresponding state laws and are often enacted because of difficulty in enacting stronger state legislation. Many local ordinances extend smoking regulations to restaurants and workplaces.[1]

New York State currently has a weak statewide smoking law, which regulates smoking only on public transportation and in theaters and libraries; it does not cover restaurants or workplaces.[1] Broad smoking restrictions for public places and the workplace were developed and adopted by the state's Public Health Council in 1987; however, they were overturned by a successful legal challenge, on the grounds that the Public Health Council had overstepped its regulatory authority.[16] Subsequently, the state legislature has considered several Clean Indoor Air Acts in its 1988 session. As of November 1988, none had been enacted.[17]

In New York, as in other states, there has been recent action at the local governmental level. New York City adopted a sweeping Clean Indoor Air Act in April 1988.[18] It requires restaurants with more than 50 seats to set aside half for nonsmoking, and requires employers with more than 15 employees to provide smoke-free working areas. Elsewhere in New York State, four counties (Nassau, Westchester, Suffolk, and Monroe) regulate smoking in worksites, restaurants, and several public places.[19] These local ordinances have all been adopted since 1985.

Smoking restrictions legislated by state and local governments are generally implemented by public health officials, rather than police departments. The adequacy of implementation and the level of public compliance is un-

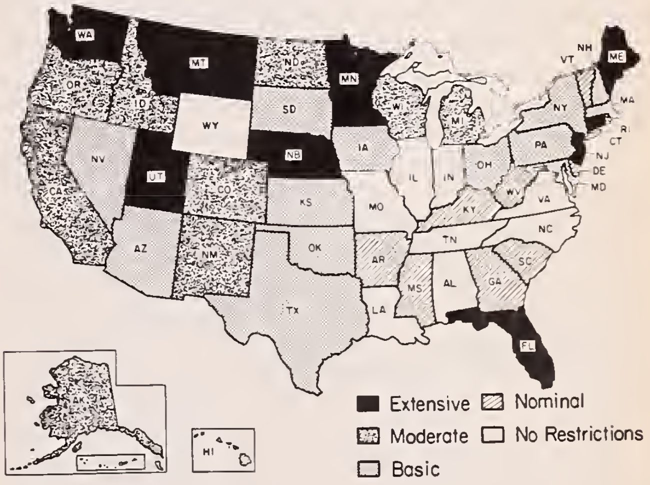

FIGURE 2. Geographic variability of state laws regulating smoking in public places, 1986. KEY: Extensive = law regulates smoking in private sector workplaces; Moderate = law regulates smoking in restaurants, but not workplaces; Basic = law regulates smoking in four or more public places, excluding restaurants and workplaces; Nominal = law restricts smoking in from one to three public places, excluding restaurants and workplaces. (Source: US Dept of Health and Human Services, *The Health Consequences of Involuntary Smoking: A Report of the Surgeon General*, 1986.)

known, as is any impact on smoking behavior or air quality. These policies are often said to be "self-enforcing."[1] The assumption is that the majority of smokers, being law abiding, will obey smoking restrictions, and that nonsmokers will enforce any noncompliance, freeing government from the need to actively police policies. In general, the lack of complaints is equated with adequate compliance.

There has been no systematic evaluation of state or local smoking legislation. The one published report examined the impact of San Francisco's ordinance. The city found that implementation required only a declining minority of a single employee's time. However, compliance was monitored passively rather than actively, ie, by response to complaints rather than active surveillance of compliance.[20] The experience of San Francisco counteracts the tobacco industry's claim that smoking laws will be expensive to implement and will serve as a drain on scarce city monies. The few studies of public opinion document strong support. In New York City, 73% of a random sample of city residents interviewed three months after the city's smoking law took effect favored the law, including 84% of nonsmokers and 43% of smokers.[21]

WORKSITE SMOKING RESTRICTIONS

The workplace has become a focus of efforts to restrict smoking for several reasons. First, the workplace is a major site of involuntary smoke exposure. Urban adults spend more time at work than at any other location except the home.[22] For adults who do not live with smokers, work is the main source of involuntary smoke exposure. The duration and intensity of smoke exposure that can occur at work makes it a major source of exposure, even for adults who live with smokers. A second reason for the focus on smoking at work is that individuals have less choice about their smoke exposure at work than in other public places. People bothered by smoke can more easily patronize a different store or restaurant than they can change their place of employment. Third, there may be synergism between passive smoke and known workplace toxins. Materials whose effects are synergistic with direct smoking, such as asbestos, may also be synergistic with the concentrations of smoke inhaled by passive smokers.[23]

A fourth reason for the focus on smoking at work is the potential for workplace restrictions to alter the smoking behavior of large numbers of adults. Changing the rules about where smoking is permitted at work is likely to alter workplace norms about smoking. This may enhance the participation in or effectiveness of ongoing worksite smoking cessation programs, increase the success of smokers' unaided efforts to quit, or reduce the number of young workers who adopt the smoking habit. By reducing the opportunities for smoking, worksite restrictions may reduce the cigarette consumption of smokers who do not quit. Each of these potential effects would contribute to reducing the prevalence of smoking among workers and thereby reduce the smoking prevalence among adults.[1]

Both public and private actions have contributed to worksite smoking restrictions. In contrast to the regulation of smoking in public places, most worksite smoking restrictions are the result of private initiative rather than governmental regulations. Most of this activity has occurred in the 1980s.

CONTRIBUTIONS BY GOVERNMENT

Government action has occurred at federal, state, and local levels and has primarily addressed smoking by government employees. The federal government has taken recent steps to restrict smoking by its own large workforce. The General Services Administration, the agency that manages half of nonmilitary federal workspace, adopted tighter smoking restrictions in February 1987.[24] The policy prohibits smoking except in designated areas, specifies areas where smoking is to be banned and where smoking may be permitted, but does not require that all working areas be smoke free. Individual federal agencies are permitted to adopt more stringent regulations, and several, including the Department of Health and Human Services and the Centers for Disease Control, have adopted virtual smoking bans.[25] Smoking by military personnel was addressed by a 1986 Department of Defense directive which ordered each military service to develop and implement a smoking policy.[26] The army has been the most active service branch to date. As a result of these actions, most federal workers are now covered by policies that restrict but do not entirely ban smoking at the workplace.

At the state level, both legislative and executive actions have regulated smoking. Most regulations are the result of legislation. As of 1986, laws in 22 states regulated smoking at the worksite for state employees, and in nine states, the law extended to cover workers in the private sector.[1] The content of worksite smoking laws is variable. The earliest laws simply empowered the employer to restrict smoking in factories. Later, more stringent laws required employers to issue a written smoking policy and to post signs, but did not stipulate the content of that policy. More restrictive laws required employers to designate nonsmoking areas at work, implying that smoking was the norm. The most restrictive laws prohibit smoking except in designated areas, making nonsmoking the norm.[26] In addition to legislation, governors in several states issued executive orders restricting smoking by state employees. Colorado and Massachusetts are two states in which this has occurred. In New York State, smoking in both public and private worksites was restricted to designated areas in regulations proposed by the Public Health Council and later overturned by the court.[16]

At the local level, a growing number of communities have passed ordinances restricting smoking at the workplace. A 1988 survey by the Americans for Nonsmokers Rights found that 117 California cities had adopted ordinances covering workplace smoking; 55% of the state's population lived in these cities.[15]

INITIATIVES BY THE PRIVATE SECTOR

A series of business surveys over the past decade document that smoking restrictions developed in the private sector worksites are largely a phenomenon of the 1980s. These surveys can be divided into two sets. The first set of five surveys, conducted between 1977 and 1980, shows relatively few restrictions in place. A second set, done in the mid-1980s, shows a remarkable change (Table II).

The 1977–1980 surveys vary widely in sample size, response rate, and estimated prevalence of smoking restrictions. The highest estimated prevalence, 64%, was reported by a survey restricted to Massachusetts.[27] Estimates of

TABLE II. Prevalence of Workplace Smoking Restrictions (Surveys Done 1977–1980 and 1984–1986)

Survey Name	Survey Year	Respondents (% Sample)	Restrict Smoking (%)
Dartnell's Business[28]	1977	250 (?)	30%
Bennett & Levy[27] (Mass. only)	1978–1979	128 (66%)	64%
National Inter-Agency Council[29]	1979	3,000 (29%)	50%
Dartnell's Business[28]	1980	325 (?)	23%
Administrative Management Society[30]	1980	500 (60%)	14%
Human Resources Policy Corp[31]*	1985	445 (40%)	32%
US Department of Health and Human Services (ODPHP)[32]†	1985	1,358 (85%)	27%
BNA[33]	1986	662 (34%)	36%
Petersen and Massengill[34]	1986	577 (53%)	56%
New York City Dept of Health[36]	1986	573 (75%)	4%
BNA[35]	1987	623 (29%)	54%

* Survey funded by Tobacco Institute.

† Office of Disease Prevention and Health Promotion.

23% and 30% were obtained from the Dartnell's Business Review, but details about this survey are lacking, and its representativeness is difficult to document.[28] The National Interagency Council conducted a large survey with a low response rate and recorded a 50% prevalence of policies.[29] Finally, the lowest prevalence, 14%, was reported in a survey conducted by the Administrative Management Society.[30]

While it is difficult from these surveys to determine the actual prevalence of policies in businesses at that time, several other conclusions can be drawn from the reports. First, policies appear to be more prevalent in larger companies and in blue collar rather than white collar areas. Second, management initiates most policies. Third, 25–37% of companies report employee complaints about smoking, suggesting that there is employee pressure on management for smoking restrictions. Finally, there were three main reasons for adopting smoking policies, according to these surveys: protection of equipment; workers' safety from explosion or fire hazards; and concern about appearance to customers and clients. Concerns about employee health were not mentioned.[1]

The second set of business surveys, done between 1984 and 1987, documented the phenomenon that occurred during the early 1980s. There is remarkable agreement among three nationwide surveys done 1984–1986 by such disparate groups as the tobacco industry, the US Department of Health and Human Services, and the Bureau of National Affairs.[31–33] The reported prevalence of policies in these three surveys varies between 27% and 36%, suggesting that approximately one third of private employers had smoking restrictions in place. A fourth survey reported a higher prevalence of smoking restrictions, but it included only white collar areas.[34] The BNA survey was repeated in 1987. The proportion of large businesses with smoking policies had increased from 36% in 1986 to 54% in 1987.[35]

Another survey, limited to New York City businesses, was done in August 1986, prior to the city's Clean Indoor Air Act.[36] This survey reported a much lower prevalence of policies, with only 4% of companies reporting written smoking policies. It is notable that this is the only one of these surveys to include a large number of smaller businesses. Indeed, half of that sample consisted of businesses with fewer than ten employees, and these were less likely than larger companies to have a smoking policy. Another explanation for the discrepancy is that businesses were asked about having a written smoking policy. Some small businesses may have a policy in place that is not written.

Several other conclusions can be drawn from a review of business surveys from the mid-1980s.[1] First, more than half of policies are less than five years old, providing evidence that they are a new phenomenon. Second, the areas where smoking is permitted are shrinking, but bans remain rare. At present, fewer than 5% of companies ban smoking at the worksite or take the additional step of restricting employment to nonsmokers. Third, worksite policies are now more prevalent in larger companies, certain types of industries (generally, nonmanufacturing industries), western or northeastern states, and areas where smoking laws are in effect. Fourth, we know little about how small businesses handle smoking. The only available data, from a survey of New York City businesses, may not be representative. The processes of decision making and implementation may be different in small versus large businesses.

Finally, the reasons for adopting smoking policies have changed over the past five years. Previously, equipment safety and explosion hazards were the major concerns; currently, the most common reason reported for adopting smoking policies is employee health and safety. The second most common reason is to comply with laws about smoking, and the third is employee pressure. Saving money is rarely mentioned, even though this is frequently pointed out by those advocating smoking policies. As reported by a tobacco industry-sponsored survey, the barriers to adopting smoking policies are union opposition, a perception that employees can handle smoking on their own, and concern about the difficulty or cost of implementation.[31]

TYPES OF WORKPLACE SMOKING POLICIES

Because smoke travels, the desires of smokers and nonsmokers will inevitably conflict in enclosed indoor areas. It is difficult to achieve simultaneously the goals of smoke-free air (ie, maximum employee protection), minimum smoker disruption, and minimum cost of implementation. The policy that a company adopts indicates the relative importance placed on each of these values.[1] For example, a company wishing to maximize protection of employee health might adopt a total ban on smoking, but such a policy is the most disrupting to current smokers. A smoking ban also offers the potential for minimal cost, because it requires no changes in the physical plant and offers a potential for reducing maintenance costs. Businesses concerned with disrupting smokers are likely to pay greater heed to smokers' wishes at the expense of smoke-free air, and may try to increase ventilation to avoid the necessity of behavioral change. This, however, is an expensive choice. An alternative is to separate smokers and nonsmokers, but this presents the logistic problem that smoke travels, even when smokers and nonsmokers are separated. Companies that do not adopt a policy do not risk alienating their smokers or incurring expenses, but do so at the cost of ignoring employee health concerns. Given the current climate of increasing nonsmoker activism, such companies are likely to face complaints from their employees, the majority of whom are nonsmokers.

Businesses developing smoking policies generally divide the physical plant into four categories: private offices; shared offices or work areas; small common use areas, such as elevators and bathrooms; large common use areas, such as conference rooms, meeting rooms, auditoriums and cafeterias. Surveys indicate that at present, most companies restrict smoking in small and large common use areas, with the exception of cafeterias, where there is generally a designated smoking section. Smoking is generally permitted in private offices.[33] There is less potential for involuntary smoke exposure in private offices, so long as nonsmokers are not required to go into the private office, and the smoke does not travel out of the office, either by diffusion or via a recirculating ventilation system. However, since the managers who make the decisions are often in private offices, permitting smoking in private offices and banning it in shared work areas present equity concerns which may endanger employee morale. Decisions about smoking rules for shared offices or work areas generate the most controversy, because the wishes of smokers and nonsmokers are in most direct conflict in these areas. Because workers spend most of their time at their primary work area, this is the location where the nonsmoker is at greatest risk for passive smoke exposure, and where smokers most often wish to smoke. Currently, most policies attempt some restrictions on smoking in these areas, but because of the diffusibility of smoke, it is difficult to contain smoke to a single area.

Current smoking policies may be categorized as a continuum[1]:

1. *No Explicit Policy.* (This is the so-called "common courtesy" approach.) The lack of policy is still the most prevalent situation. While it offers the least potential for disruption of smokers, it provides the least protection to nonsmokers. Given the increasingly negative attitudes of nonsmokers toward environmental tobacco smoke, it is unlikely to be a long term solution.
2. *Environmental Alterations.* These include partitions, air filters, or altered ventilation systems. While these offer the potential both for maximizing protection of employee health and minimizing smoker discomfort, they also maximize cost. The number of air changes required for adequate ventilation of an area where smokers are smoking requires very expensive ventilation systems which are prohibitive for most companies.[1]
3. *Separating Smokers and Nonsmokers.* The most common type of policy restricts employee smoking.[33] In the weaker of these policies, smoking is permitted except in designated nonsmoking areas, and location of the designated smoking areas is highly variable. Therefore, the extent of worker health protection is also variable, as is the extent of smoker discomfort. More stringent policies establish nonsmoking as the norm and permit smoking only in designated areas, which may be highly variable in extent.
4. *Banning Employee Smoking.* Fewer than 5% of US businesses currently have adopted this approach.[33] There are anecdotal reports of several large companies, such as Pacific Northwest Bell, which have done so without serious incident.
5. *Preferential or Exclusive Hiring of Nonsmokers.* This is the strongest policy option, one taken by fewer than 5% of US companies.[33]

Provisions for implementation and enforcement are also common components of good smoking policies. Such provisions may include requirements that nonsmokers' rights prevail when there is a dispute, that signs be posted as necessary, or that plans for enforcement be clearly outlined. Concerns about enforcement are shared by both employees and managers. Many policies designate who is responsible for enforcement, specify penalties for noncompliance, and guarantee protection for employees who bring complaints.

Policy implementation appears to be most successful when the following provisions are met: The policy is written and well-communicated to employees; the policy is formulated with broad input from employees, including both smokers and nonsmokers, both management and nonmanagement; the policy is implemented after a grace period, with advance notice to employees; smoking cessation programs are available during and after the grace period; and an oversight committee monitors the success of policy implementation and enforcement.[1] Additional recommendations regarding the implementation of smoking policies are outlined in manuals developed by several groups.[37–39]

EVALUATING THE IMPACT OF SMOKING POLICIES

Policies that regulate where smoking is permitted may have a number of direct and indirect effects which should be considered in policy evaluation.[1] In the short term, a policy that is adequately implemented and enforced will alter the behavior of smokers in areas where smoking is prohibited. Such a policy should result in a reduced concentration of tobacco smoke and less passive smoke exposure in that area. Beyond these direct effects, there is the potential for smoking restrictions to have broader, indirect effects on smoking behavior and on public attitudes about tobacco use.

The degree to which a smoking policy has been implemented as written is an essential initial consideration in evaluating its impact. Adequate policy implementation requires that workers or citizens be aware of, comply with, and enforce the policy. If these indices are not measured, one cannot interpret any finding of a lack of policy effect on air quality or smoking behavior. A second outcome to consider is the level of policy approval.

The primary goal of policies regulating smoking is the reduction of the individual's exposure to environmental tobacco smoke. Measures of air quality directly assess how well a policy meets this goal, and indirectly reflect either the adequacy of smoking restrictions or compliance by smokers or both. Measurements of air quality can be complex, and careful control of sample collection is necessary. Measurements of carbon monoxide, respirable particulates, and nicotine have all been employed.[3] Evidence of changes in nonsmokers' exposure to smoke using biochemical measures may also be possible for worksite policies.

At the worksite, smoking employees who comply with smoking restrictions will almost certainly smoke less at work. The impact on employees' overall smoking behavior is not known. The nicotine regulation model would predict that smokers will attempt to keep their overall cigarette consumption constant and will compensate for reduced opportunities to smoke at work by smoking more cigarettes before work, after work, and during break times. The alternative hypothesis, that restrictions will induce attempts to quit by the large number of smokers who express a desire to quit, would predict that smokers may take advantage of the smoking restrictions to reduce their cigarette consumption.

In addition to these effects, smoking restrictions may have indirect effects on attitudes and norms about the acceptability of smoking and on smoking behavior. Policies restricting smoking help to reinforce nonsmoking as the normative behavior, probably increase individuals' knowledge of the health risks of smoking and involuntary smoking, and may alter attitudes about the desirability of the smoking habit. Changes in social norms may influence smoking behavior by reducing social pressures to smoke and increasing social support for cessation.

Smoking restrictions, especially at work, may have several effects on smoking behavior. If employees cannot smoke at work, fewer people entering the workforce may use cigarettes to cope with work-related stresses, and there may be fewer young people who develop a regular smoking habit. Smoking restrictions may increase smoking cessation rates by increasing individuals' motivation to quit smoking and the success rate of individual quit attempts. There may be a reduction in cigarette consumption among continuing smokers, as a consequence of fewer opportunities to smoke.

In addition, worksite smoking restrictions may have economic effects. These effects will be positive if policies reverse the excess costs associated with smoking employees, which have been estimated at $300–600 per employee year.[40] While these are not the reasons for implementing the policies, they are of interest to managers who are considering adopting such policies. There is the potential for reduction in maintenance costs, employee absenteeism, health care costs, and increases in employee productivity. Maintenance costs should be lower if there is less smoking-related debris, as would occur if a policy is adequately enforced. Measurable changes in employee absenteeism or health care costs probably will occur only if a significant number of employees stop smoking, since it is known that smokers, on average, have higher absenteeism rates than nonsmokers. The effects on employee productivity are controversial and difficult to measure. Some claim that overall employee productivity will improve with less irritation from involuntary smoke. Others argue that the productivity of smokers will decline because of the necessity for them to take breaks in order to smoke.

CURRENT STATE OF THE EVIDENCE

Although a growing number of cities, states, businesses, and governmental agencies are adopting smoking restrictions, systematic evaluation of their impact is rare. A number of businesses have conducted baseline surveys of employee attitudes and behavior prior to adopting smoking restrictions, but few have done post-implementation surveys to assess impact. Virtually no studies include a control business in which no policy was adopted. To date, the best evaluations of worksite smoking policies have been conducted in health care facilities. There are no systematic studies of the impact of community-wide or statewide regulations on smoking in public places.

On the basis of case reports and a small number of systematic studies of worksite smoking policies, it appears that these policies improve air quality, are met with good compliance, are well accepted by both smokers and nonsmokers, decrease employees' cigarette consumption at work, increase enrollment in company-sponsored smoking cessation programs, and do not increase employee turnover rates. There is little information available on the effect of smoking policies on employees' overall smoking behavior, attitudes about the acceptability of smoking in public, or costs to employers.[1] (Evidence about the effects of worksite smoking policies is reviewed in more detail in the 1986 surgeon general's report.[1])

RECOMMENDATIONS FOR FUTURE RESEARCH

Smoking regulations affect the daily lives of a large and growing number of Americans. The potential public health impact of smoking restrictions, particularly those in the worksite, is great. These policies will be regarded as successful if they achieve their stated aim of reducing nonsmokers' exposure to smoke. However, they will assume far greater public health importance if they are found to increase the cessation rate of smokers, and reduce the rate at which the young take up smoking. If policies do have these effects on smoking behavior, they will make an important contribution toward reducing the prevalence of smoking in the US. They will represent an advance that can complement currently existing and more expensive, individually tailored smoking reduction efforts. Consequently, the evaluation of worksite smoking policies should be a high priority for research. At present, we know very little about the effect of smoking policies, particularly on smoking behavior.

Opportunities to evaluate worksite and community smoking policies will continue to appear. Worksite smoking policies represent a trend that is likely to accelerate, due to the increasing prevalence of laws governing smoking in the workplace and increasingly negative attitudes about smoking on the part of nonsmokers. Therefore, opportunities to study "natural experiments" will continue to appear, and researchers need to be ready to take advantage of them. To take advantage of these natural experiments, research on worksite smoking policies must overcome several barriers: limited interest by businesses in doing research after a policy has been implemented; limited awareness of opportunities by public health and behavioral scientists who are generally not in direct contact with the business community; difficulty in acquiring funding sufficiently rapidly to respond to these opportunities; and difficulty in developing study methodology and recruiting study personnel sufficiently rapidly to take advantage of research opportunities. In order to measure change, surveys must be conducted at baseline, before policies are implemented; this must often be done on short notice.

Research on worksite smoking policies could be made easier if the following information and funding were available to researchers. A standardized research methodology should include a list of outcomes to measure and standardized methods for their measurement. The availability of expert consultation is also important. Some funding should be available on short notice, in order to conduct baseline observations. This may only require a small amount of initial funds, but with rapid availability. It is possible to imagine funding mechanisms in which investigators who have succeeded in collecting baseline data are eligible to apply for larger grants which would permit follow-up data collection and analysis of all data. The ability to identify adequate research personnel on short notice is also necessary. Finally, access to businesses by researchers is obviously essential. Private consultants and groups such as the American Lung Association, which may have been contacted by businesses to help develop a smoking policy, might serve as the liaison between the business community and researchers.

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Health effects of involuntary smoking in the workplace

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Cigarette smoking remains a common practice despite a continuing trend toward decreased numbers of active smokers.[1] In 1985, approximately 33% of males and 28% of females were estimated to be current smokers.[1] Thus, for the foreseeable future, it is likely that individuals will experience involuntary exposure to the combustion products of cigarette tobacco, and that some of this exposure will occur in the workplace. This review summarizes the available data on health effects associated with involuntary exposure to tobacco smoke and attempts to identify those elements of the data that are relevant to workplace exposure. This presentation is not intended to be exhaustive; rather, it seeks to place the issue of workplace exposure into perspective in the light of current data about the health effects of involuntary exposure to tobacco smoke.

ENVIRONMENTAL TOBACCO SMOKE

Environmental tobacco smoke (ETS) is a complex mixture of mainstream smoke (the portion of the smoke stream that is inhaled by the active smoker) and sidestream smoke (the smoke emanating from the burning butt end of the cigarette). While these components of tobacco smoke are qualitatively similar in their makeup, they differ quantitatively[2] as a consequence of the differing temperatures involved in their production. Sidestream smoke is the larger contributor to environmental tobacco smoke and contains higher concentrations of potentially harmful chemicals such as nitrosamines, benzo[*a*]pyrene, benzene, hydrazine, and many others.[2]

Cigarette smoke contains thousands of chemicals, many of which are present in environmental tobacco smoke, and the actual exposure to any one or set of these cannot be inferred from knowledge of others. Moreover, the nature of the exposure is further complicated by contributing factors: particle growth by adsorption of water in the environment and in the respiratory tract; absorption of other compounds (eg, radon daughters) onto aerosol generated by cigarettes; chemical interactions of combustion products of tobacco with other chemicals in the air (eg, oxidation of nitric oxide to nitrogen dioxide); the ventilation characteristics of the exposure space; the time-activity patterns of the exposed individuals; and the number of smokers present at any given time.[3]

QUANTIFYING EXPOSURE

Attempts to quantitate exposure have taken various approaches. The use of "cigarette equivalents"[2] attempts to relate the particulate mass inhaled by passive exposure to environmental tobacco smoke to that inhaled by actively smoking one cigarette. This method is limited by the need for a substance-specific approach, the lack of consideration of the actual particulate mass deposited in the respiratory tract, and the differences in inhalation between nasal and mouth breathing. The use of monitoring devices to capture personal exposure to environmental tobacco smoke components such as nicotine and particulates also has been attempted,[4] but the data are very limited to date. The most frequent methods of exposure evaluation involve the use of standardized questionnaires or the use of biological markers.

Questionnaires have the advantage of ease of administration and the capacity to provide information on past exposures that cannot be directly measured. Questionnaires theoretically suffer from potential problems of recall bias, inaccurate recall, and false reporting of personal tobacco use. For example, approximately 5% of individuals who said that they do not smoke in one interview reported themselves to be active smokers in a previous interview.[3] Although such reporting errors do lead to some misclassification of smokers as nonsmokers, an extensive analysis by the National Research Council has indicated that the association of health effects with environmental tobacco smoke exposure cannot be solely the consequence of this misclassification.[3] This analysis was based on the fact that studies with biological markers have found questionnaires to be reasonably accurate, at least for short-term exposure,[3] with only a small percentage of nonsmokers having marker levels in the range found for smokers.[3] Among the most significant limitations of existing questionnaire data are the facts that many of the questions were not designed specifically to investigate involuntary exposure, or the questionnaires have been incomplete in their probing of the circumstances of exposure (eg, intensity, duration, specific location, etc). These limitations have made it difficult to provide even semi-quantitative exposure estimates over time.

Currently, the use of biological markers (defined as the detection of a product of tobacco smoke or a metabolite of such a product in a body "fluid") is the method most fre-

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quently relied on for objective definition of exposure to environmental tobacco smoke. Measurements of cotinine—a unique metabolite of nicotine—in urine, serum, or saliva, is the marker most frequently employed in current studies. Studies have demonstrated quantitative relationships between both smoking subjects' reporting of the quantity of their own smoking and nonsmoking subjects' estimates of exposure to environmental tobacco smoke, and the concentration of cotinine in body fluids.[5,6] An example of the data with regard to exposure to environmental tobacco smoke is given in Table I. Nonetheless, despite the capacity of cotinine to provide objective evidence of exposure, it, like all currently available markers, suffers from serious limitations: only recent exposures are measured; cotinine reflects exposure to only one constituent of tobacco smoke, nicotine, and thus provides no information about exposure to other, perhaps more important constituents; the final concentration measured is a function not only of exposure, but of the body's handling of the chemical (eg, hepatic metabolism and renal excretion in the case of the nicotine-cotinine pathway).

HEALTH EFFECTS

Thus, while a number of health effects have been associated with exposure to environmental tobacco smoke, the objective data on the nature of the exposure, including constituent(s), duration, intensity, critical time of life, etc, that might be relevant for any given health effect(s) presently are inadequate.

Table II lists health effects for which there are a reasonable body of data in support of an association with exposure to environmental tobacco smoke.[3] Table III lists health outcomes the association of which with environmental tobacco smoke exposure is either unclear or unsupported by existing data.[2] In this review, only those health effects that could reasonably be expected to result from environmental tobacco smoke exposure in the workplace will be discussed.

To relate health effects to workplace exposure, it must be established that workplace environments are relevant exposure environments. In a study of 44 different work rooms (almost exclusively involving white collar occupations) with a variety of smoking and ventilation conditions, Weber and Fischer[7] demonstrated increased concentrations of carbon monoxide (CO), nitrogen dioxide (NO_2), respirable suspended particulates (RSP), and nicotine, when these rooms were occupied by smokers (Table IV). The difference was greatest for RSP, and, in the case of CO and NO_2, the differences were greater than the indoor-outdoor gradients for these constituents (Table IV). Matsukura and colleagues[8] related nonsmoking subjects' reports of the number of their coworkers who smoked to urinary cotinine concentrations. These investigators demonstrated that, after adjustment for the number of smokers reported to be at home, there was a progressive increase in urinary cotinine concentration with increasing number of coworkers who reportedly smoked (Fig 1). A similar trend was observed based on subjects' reports of the smokiness of the work environment (the types of workplaces were not specified). Thus, it can be inferred that the workplace can provide relevant environments for exposure to ETS, at least based on current evaluation techniques.

TABLE I. Median Urinary Cotinine Concentration in Smokers and Nonsmokers*

Smoking Category	No.	Urinary Cotinine (ng/mL) Median
Nonsmokers		
No exposure to other people's smoke	22	2-0
Exposed to other people's smoke	199	6-0
All	221	4-0
Smokers		
Cigarettes only	131	1645
Cigars only	59	396
Pipes only	42	1920
All	232	1474

* Adapted from Table II in Wald et al.[6]

TABLE II. Health Effects Associated with Exposure to Environmental Tobacco Smoke

- Respiratory symptoms, signs and function
  - Acute respiratory illness in early childhood
  - Chronic cough, phlegm, and wheeze in children
  - Chronic middle ear effusions in children
  - Reduced levels and growth of lung function in children
  - Reduced levels of lung function in adults
  - Increased bronchial hyperresponsiveness (asthmatics)
  - Increased symptoms and decreased level of lung function in asthmatics
- Irritant effects
  - Eye, nose, throat
- Malignancy
  - Lung cancer
- Childhood development
  - Low birth weight
  - Decreased attained height

TABLE III. Health Effects Whose Association with Exposure to Environmental Tobacco Smoke Is Unclear

- Malignancy
  - Brain
  - Nasal sinuses
  - Breast
  - Cervix
  - Leukemia/lymphoma
  - Endocrine
- Cardiovascular
  - Overall cardiovascular mortality
    - Ischemic heart disease

TABLE IV. Levels of Selected Air Pollutants in a Sample of 44 Workrooms*

Mean (maximum) difference in concentration (occupied-unoccupied)	
CO (ppm)	1.1 (6.5)
NO (ppb)	32 (280)
RSP ($\mu g/m^3$)	132 (962)
Nicotine ($\mu g/m^3$)	0.9 (13.8)
Mean (maximum) difference in concentration (indoor-outdoor)	
CO (ppm)	0.8 (6.2)
NO (ppb)	22 (320)

* Adapted from Weber and Fischer.[7]

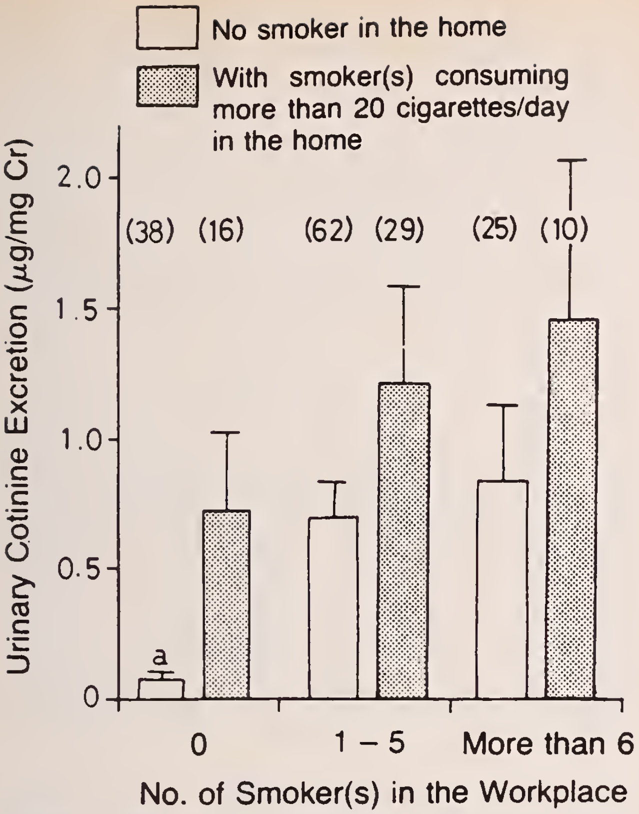

FIGURE 1. Relationship of the number of smokers reported at work and at home to urinary cotinine excretion (expressed as μmg cotinine/mg of urinary creatinine) (by permission, Matsukura et al[8]).

Acute exposure to ETS in the workplace could affect physical performance. Exposure chamber studies with healthy men and women exposed to machine-produced smoke[9,10] suggest that this is not likely under conditions of moderate exposure. Consistent alterations of cardiorespiratory function have not been demonstrated during either rest or exercise (Table V).

In contrast to the lack of alteration in cardiorespiratory function, acute exposure to environmental tobacco smoke has been associated with a variety of "irritant" symptoms (Table VI), although the available data are not uniform in support of the associations.[2,3] Exposure chamber studies have demonstrated an exposure-effect relationship between the amount of CO in the environment and eye irritation as assessed by blink frequency.[2] A study of office workers in their work environments has also demonstrated a correlation between ambient CO levels and eye irritation—an association not observed for nicotine and RSP concentrations.[7] However, a large study of nine office buildings, involving 1,100 subjects, could not demonstrate a difference in frequency of reported headache; fatigue; or nose, throat, and eye irritation in workers exposed and those not exposed to smoke.[11] This study did not include any direct measurements to assess the degree of environmental contamination by ETS in those workplaces where smoking was reported.

TABLE V. Physiologic Effects of Acute Passive Exposure to Cigarette Smoke

Cardiovascular effects
Rest
  Heart rate slightly increased in females
  No effect in males
Exercise
  No effect
Respiratory Effects
Rest
  Minimal increases in functional residual capacity (FRC) and minimal decreases in maximal flow at 25% vital capacity ($\dot{V}_{max}25$)
Exercise
  No significant change in FRC, expiratory reserve volume, total lung capacity, forced expiratory volume in 1 second, $\dot{V}_{max}50$; $\dot{V}_{max}25$

TABLE VI. Irritant Effects Attributed to Passive Exposure to Cigarette Smoking

Eye, nose and throat irritation
Cough
Headache
Wheeze*

* May involve an allergic mechanism for some individuals.

In 1980, White and Froeb[12] presented data suggesting that workplace exposure to ETS might lead to small reductions in forced mid-expiratory flow (FEF_{25-75}). This study has been criticized because of the "volunteer" nature of the population, the lack of complete data on the criteria for subject exclusion, and the fact that the instrument used to measure lung function did not meet accepted performance standards. Nonetheless, measurements of CO at the work stations of randomly selected subjects did tend to support the exposure histories obtained. Even if the study was flawless, the health significance of the small effect observed can be called into question.[13] A subsequent study of 1,351 volunteer office workers in Germany[14] failed to find any consistent relationship between a variety of measures of lung function and self-reported workplace exposure to ETS. Unfortunately, no data were provided on the nature of the questions asked.

Extensive evaluations by both the National Research Council[3] and the Office of the Surgeon General have concluded that exposure to environmental tobacco smoke confers an approximately 1.4-fold excess risk of lung cancer[3] in nonsmokers, compared to nonsmokers not exposed to ETS. By their estimation, approximately 20% of lung cancer in nonsmokers can be "attributed" to environmental tobacco smoke exposure.[3] The large body of data on this subject is best summarized in Figure 2, which is taken from the report of the National Research Council.[3]

Although most studies that have investigated the relationship between lung cancer and ETS exposure have been based on the risk to the nonsmoking spouses of smokers, there are several studies in which it is possible to separate out a possible contribution of workplace exposure (Table VII). Because of the seriousness of this health risk, a few of these studies will be reviewed. Kabat and Wynder[15] identified all cases of primary lung cancer among people who had never smoked from an ongoing study of tobacco-related cancers. Smoking history was obtained through standardized interviews. For a subset of cases and controls, specific questions about ETS exposure were asked. Among males, the odds ratio for lung cancer was 3.2 ($p = 0.05$) for work-exposed versus non-work-ex-

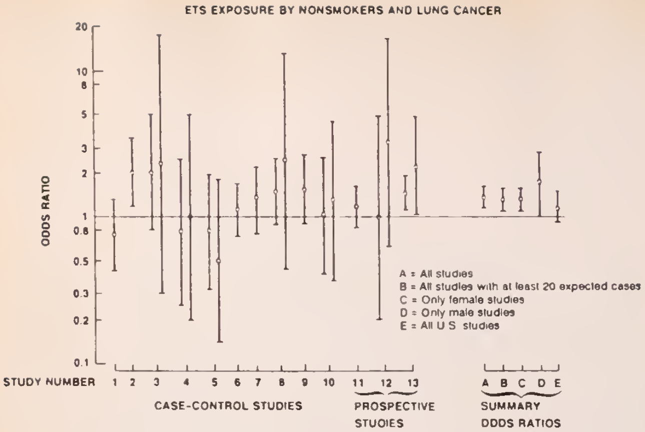

FIGURE 2. Summary of studies that have evaluated the risk of lung cancer in nonsmokers whose spouses smoke compared to those whose spouses do not smoke. Each point represents the relative risk derived from the study, and the bars represent the 95% confidence limits of the estimates (by permission, National Research Council[3]).

posed subjects. Spouse smoking was equivalent in both groups. No such relationship was observed for women.

Wu and colleagues[16] conducted a case-control study of female patients with primary adenocarcinoma or squamous carcinoma of the lung in Los Angeles. Cases were matched to neighborhood controls. Standard interviews were employed that included a lifetime job history. The personal smoking-adjusted work exposure odds ratios were 1.2 (95% confidence limit, 0.8–2.2) and 2.3 (95% confidence limit, 0.7–7.9) for adenocarcinoma and squamous carcinoma, respectively. No adjustment for passive exposure at home was provided.

The study of Garfinkel et al[17] involved histologically proven lung cancer in women from three hospitals and control subjects with colon cancer. Structured interviews with subjects or next-of-kin were employed. No association with work exposure over the "last five years" or "last 25 years" was observed (odds ratios < 1). No data were provided on the percentage of next-of-kin interviews, which makes it difficult to interpret the work exposure data.

In the study of Koo et al,[18] cancer patients reported more workplace exposure, but the numbers in this study were too small for meaningful estimates of risk. Finally, the National Research Council has concluded that the adjusted excess risk of lung cancer for nonsmokers married to nonsmokers compared to truly unexposed individuals is approximately 8%, and that some of this small excess risk may come from the workplace and other exposures.[3] Thus, there is only suggestive but not conclusive evidence that environmental tobacco smoke exposure in the work environment may be a hazard in terms of lung cancer risk.

TABLE VII. Summary of Data on Relationship of Exposure to ETS In the Workplace and Lung Cancer

Study	Subjects	Risk with Exposure (odds ratio)
Kabat[15] (1984) Case-control	Lung cancer in never-smokers; multicity registry	Males (3.27)
Wu[16] (1985) Case-control	Females with adenocarcinoma; squamous cell carcinoma from tumor registry	Adenocarcinoma (1.2) Squamous cell carcinoma (2.3)
Koo[18] (1984) Case-control	Female lung cancer; hospitalized patients	Suggestive; small numbers
Garfinkel[17] (1985) Case-control	Female lung cancer patients in four hospitals; registries	No

There is a considerable body of evidence that associates cigarette smoking during pregnancy to the occurrence of low birth weight in infants, an effect that is related to fetal growth retardation.[19] A recent study of 3,841 antenatal patients at Yale-New Haven Hospital extends these concerns to passive exposure of nonsmoking pregnant women.[20] This study showed that after adjustment for gestational age, race, maternal age, and parity, passive exposure to cigarette smoke (on questionnaire, exposure ≥ 2h/day) was associated with the birth of an infant weighing less than 2,500 grams (odds ratio = 2.2, 95% confidence limit, 1.1–4.5). Of particular interest for this review was the observation that, relative to all other groups, women who reported passive exposure were significantly more likely to be employed. These data by no means prove that the workplace was the source of the exposure, but they raise the spectre of this possibility.

CONCLUSIONS

The current body of data with regard to the workplace as an exposure source for environmental tobacco smoke and the health effects attendant on any such exposure is hardly complete. The nature of the exposure is poorly defined in terms of intensity of environmental contamination, and duration of exposure over the short and long term. With the possible exception of irritant effects, the workplace contribution to the health effects of ETS has been poorly studied. This is especially true with regard to excess lung cancer risk and possible effects on the unborn fetus.

The need for additional research is clear. Epidemiologic studies of environmental and health effects must provide more precise estimates of the range of exposures and the occurrence or nonoccurrence of health effects. That this research should occur expeditiously can be justified by the fact that there are already sufficient data to suggest that environmental tobacco smoke in the workplace may in fact be a cause of adverse health outcomes.

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Assessment of smoking behavior in relation to worksite smoking policies

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The recent trend to establish more restrictive worksite smoking policies presents an important opportunity to collect information regarding the effects of organizational policy on health-related behaviors in defined populations. However, assessment of the effects of smoking policies is an extremely complex issue. Other papers review evaluation of worksite air quality and the economic impact of smoking policy decisions.[1,2] This presentation will discuss the assessment of smoking behavior, which is itself a multifaceted topic.

This paper provides an overview and brief discussion of assessment issues in four separate but related areas: direct effects on employee smoking behavior during the work day; effects on smoking during nonwork hours; effects on cessation attempts and other behavior change efforts; and effects on potential mediating variables such as social norms and smoking-related attitudes. Within each of these four areas data can be collected in either a basic low cost manner or with more comprehensive and costly methods.

SMOKING AT THE WORKSITE

It is likely that worksite smoking policies will have the most direct impact on smoking behavior during work hours. By far the most common vehicle for assessing smoking during work hours consists of employee surveys administered prior to the introduction of a new policy and again at one or more points following implementation of the policy.

Such surveys can be an informative and very low cost assessment method if certain criteria are met. The primary threat to the validity of such surveys is their representativeness. Return rates for surveys are often quite low, and rarely exceed 85%. When possible, it is helpful to use other data sources to identify the characteristics of employees who fail to complete such surveys. The second issue requires balancing concerns about employee confidentiality and minimizing demand characteristics with the need to track individual employees over time. Anonymous surveys do not permit use of repeated-measures designs and are open to several alternative interpretations of results. On the other hand, surveys that identify individual employees may implicitly or explicitly produce strong demand characteristics to report compliance with the new smoking regulations (complete nonsmoking during work hours). The third issue has to do with the specificity of questions asked on the survey. Questionnaires should assess not only smoking behavior during work versus nonwork hours, but also such behavior in several different settings at the worksite, including work stations, break rooms and cafeterias, and outside of the building.

Some investigators[3] have developed observational and unobtrusive measures of smoking which can provide a more comprehensive assessment of smoking at work. Such procedures as having trained observers record smoking behavior in different worksite settings (as well as smoking "on the back porch"), and unobtrusive measures or indices such as vending machine sales, or the number or weight of cigarette butts in ash trays, can provide useful evidence of convergent validity for the low cost surveys mentioned above. The greatest difficulties with observational procedures are their potential reactivity and the length of time over which observations must be made. Finally, some biochemical measures may prove useful indices of smoking at work. Variables such as levels of carbon monoxide and/or nicotine, both of which have short half

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lives, could be used to assess smoking in the recent past (eg, over the past several hours). It is to be hoped that future evaluations of worksite smoking bans will include one or more of these measures to supplement the prominent survey evaluations.

SMOKING DURING NONWORK HOURS

The second major category of smoking behavior related to worksite smoking policy concerns smoking during nonwork hours. There are two major issues here. The first is the impact of a policy in stimulating smoking cessation, and the second is the possibility of "compensatory smoking."[4,5] Reports currently available on the impact of smoking bans suggest that relatively few persons stop smoking immediately following the institution of such policies.[6,7] However, the time period of such evaluations may be too short to draw conclusions regarding long term behavior change and the eventual health implications of smoking policies. Restrictive smoking policies may contribute to changing secular trends in such a way as to make smoking non-normative and to produce delayed or "sleeper" effects on smoking cessation rates.

In evaluating the health implications of smoking policies, it is important to assess smoking patterns before and after work, as well as during the work day. At least one early study[8] reported somewhat increased smoking rates during nonwork hours associated with a company no-smoking policy. Additional research is needed to evaluate the extent to which compensatory smoking does occur and, if it does, how it is expressed (increased number of cigarettes or topographical changes). It is also possible that different types of smokers (heavy smokers vs light smokers) may be differentially susceptible to compensatory smoking effects.

There are two relatively low cost methods of assessing smoking during nonwork hours. The first, as discussed above, is worksite surveys or questionnaires that clearly differentiate smoking during work hours and other times. Such information can also be collected through telephone surveys, which may reduce problems with demand characteristics, particularly if they are made by a third party rather than by the organization itself. The other relatively low cost measure is record keeping or self-monitoring of smoking frequency and topography by employees. Self-monitoring measures provide much more intensive, detailed information, but are susceptible to problems of noncompliance and reactivity.

More comprehensive measures of smoking during nonwork hours may again include biochemical measures, involving longer term indices of smoking exposure such as saliva cotinine or thiocyanate. Other more intrusive and/or expensive assessment procedures include the collection of cigarette butts, which can later be weighed and/or rated by research assistants,[9,10] or machine measures of smoking topography.[11]

CESSATION ATTEMPTS

In addition to potential direct effects on smoking cessation, worksite policies may have important effects on cessation attempts by employees and/or maintenance of nonsmoking by recent quitters. Research has suggested that individuals who have made a greater number of previous attempts to quit may be more likely to succeed at a current cessation effort.[12] A worksite policy may stimulate employees to make additional attempts—even if they are not successful—at long term cessation. Also of great potential importance is the impact of smoking policies on maintenance of nonsmoking behavior. Apparently no studies have directly reported on this issue. But employees may be more likely to maintain nonsmoking behavior or to "recycle" back to the active quitting stage[13] following a slip or relapse to smoking if they are in an environment where smoking does not occur for eight hours of the day.

Several definitional questions remain concerning exactly what constitutes a cessation attempt, a successful cessation, and a slip or lapse (vs a full-blown relapse). These issues are beyond the scope of the current discussion, but interested readers may refer to recommendations from the NHLBI Conference on Smoking Relapse recently published in *Health Psychology* for a discussion of these issues.[14]

The most basic assessment method consists of a single face-valid item or two on an employee survey asking whether or not employees made a serious attempt at quitting or, alternatively, the number of attempts they have made over a given time period. More comprehensive measures may be better acquired in the context of an interview that allows for probing of complex issues and ambiguous answers. Such interviews have the potential of covering more detailed issues such as the duration of cessation or an attempt at cessation and whether or not individuals "recycle" or reduce the latency between attempts as a result of worksite smoking policies.

MEDIATING VARIABLES

The variables discussed thus far refer to overt behaviors, which are at least potentially verifiable. However, many of the arguments in favor of worksite smoking policies concern the effects of smoking policies on social norms, worksite culture, or attitudes toward smoking rather than actual smoking behavior change. Although this issue is open to debate, many researchers see such smoking related cognitions as mediators or precursors of actual behavior change. Table I lists five categories of smoking related cognitions that have often been assessed in smoking cessation research. For each of these constructs, examples of the type of questions asked are listed. For each category it is usually possible to conduct a basic assessment, consisting of one or two items addressing the issue of interest, or to administer a relatively lengthy and more sophisticated instrument (eg, a social support questionnaire). Obviously, if the latter option is pursued, a given study will be able to study in detail only one or two of these issues.

In summary, assessment of smoking related cognitions is probably less critical than measures of actual smoking related behavior. Interesting preliminary work has been conducted on these measures, and it is recommended that policy evaluations include questions on at least some of these variables. Such measures can potentially help us to understand employee reactions to smoking bans as well as how policies may result in behavior change.

TABLE I. Smoking-Related Cognitions

Construct	Examples of Measures
Perceived social support (from coworkers, supervisor, company)	Encouragement to stop smoking Offers of cigarettes
Social norms*	Percent coworkers that smoke; percent quit recently Percent coworkers smoke on job Acceptability of smoking in public
Self-efficacy/intention	Confidence can quit Certainty will be smoking in a year
Attitudes toward smoking policy and secondhand smoke*	Approval of policy "People should not be allowed to smoke at work" "Smoking affects the health of others"
Stage of change (precontemplation, contemplation, action, maintenance)	Interest in quitting

*Indicates variables mentioned particularly often in discussions of worksite smoking policies.

METHODOLOGICAL/CONCEPTUAL ISSUES AND FUTURE DIRECTIONS

Up to this point, four basic types of measures have been considered: smoking behavior during work hours, smoking behavior outside of work hours, cessation attempts and recycling/maintenance, and smoking-related cognitions. There are other issues that may determine the long term impact of a smoking policy. First, evaluations should assess the effects of smoking restrictions on different subgroups of employees, as there may be differential effects. For example, the effects of smoking policies on heavy vs light smokers, white-collar vs blue-collar smokers, older vs younger employees, and men vs women will affect the eventual health costs to an organization.

It is also important that future evaluations include assessment of the long term (eg, at least two-year follow-up) effects of smoking policies and of potential side effects. As mentioned above, it does not appear that there will be large initial cessation effects resulting from implementation of smoking bans, but such policies may provide an important context for later attempts, especially when combined with other smoking control or cessation activities.

In addition, potential, often unintentional, side effects of policies should be considered. Such effects can be either positive or negative. For example, establishment of smoking areas at the worksite could become a cue for continuing smokers to smoke more than they otherwise would. Some employees who are lighter smokers may increase their association with heavier smokers during work breaks and thus become more confirmed smokers. It is also possible that policies may either benefit or detract from efforts to recruit new employees and/or keep current workers. To date, such side effects are entirely speculative, but it will be important for policy evaluations to be sensitive to such issues.

There are two additional classes of variables recommended for assessing any study of worksite smoking policies. These variables may be thought of as mediating variables, rather than measures of smoking behavior; however, they are important and may even have greater impact on employee reaction to a policy and how effective it is than the actual content of the policy. These factors include organizational characteristics and the way in which a policy is implemented. Although there are few empirical data on the relationship of organizational factors to worksite health promotion,[15,16] the following factors are probably deserving of at least initial consideration: number and type of employees, previous worksite smoking policy, degree of management support for the policy, smoking status of top company officials, history of health promotion and safety efforts in the worksite, labor-management relations, and economic climate of the worksite. In particular, the fit between organizational characteristics such as management style and philosophy and the way in which a policy is implemented may override the actual content of the smoking policy.

Table II lists several implementation variables potentially related to how a program will be received. It is believed that there have been no empirical studies relating such implementation variables to the effects of the policy, but it is to be hoped that future studies will assess variables such as those listed. At the minimum, persons responsible for implementing a worksite policy should carefully document the sequence of events from initial development of the ideas for a new smoking policy throughout its implementation and enforcement for at least a period of one year. The major consideration for several of the variables listed in Table II is the extent of employee involvement in the policy that is developed. Issues such as the involvement of smokers and labor representatives in decision making bodies, whether or not employees are given the opportunity to provide input and feedback on initial drafts of the policy, and the length of time between initial notification and actual implementation of the ban are important variables.

Employee awareness of the policy may be an issue in larger companies. This may include such items as the extent to which "no smoking" signs are posted, and whether employees are provided with written notification of a policy. Whether or not the company offers smoking cessation programs for employees and financial support for such activities (eg, time off work, partial payment or reimbursement for the costs of cessation services, etc) may also affect reactions to the ban. Finally, enforcement procedures and the way in which complaints and infractions are handled may be important factors in determining the long term status of the policy.[17]

In summary, we have a rare opportunity to evaluate an important natural public health experiment involving the institution of much more restrictive worksite smoking policies. The assessment of one or more measures within each

TABLE II. Implementation Variables

Degree of employee involvement
Timing and sequencing of implementation
Employee awareness of policy
Opportunity/company support for cessation efforts
Enforcement procedures

of four categories is highly recommended. Employee smoking behavior during work hours; smoking behavior outside of work, cessation attempts and maintenance or recycling efforts; and smoking-related cognitions should all be assessed in comprehensive evaluations. Each of these variables can be measured in either an efficient, low cost manner, or with more costly and sophisticated state-of-the-art procedures depending upon the purposes of the particular study. Also recommended is assessment of mediating variables, including organizational characteristics, the effects of smoking policies on different subsets of employees, and implementation variables that may determine the success of a policy.

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Nonsmoking/smoking policy implementation at the state level

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Minnesota has long been recognized as an innovator and leader in tobacco-use prevention efforts. It actively promotes a tobacco-free life style among its residents. In 1975, it set the stage as one of the first states to implement a Clean Indoor Air Act, which paved the way for more comprehensive nonsmoking legislation within the state.[1,2]

In the fall of 1983, Minnesota's prevention efforts were intensified, as the Commissioner of Health convened a technical advisory committee to design a comprehensive, long term, statewide plan for nonsmoking.[3] The plan would promote nonsmoking through a public information campaign based on marketing principles and would be combined with regulatory and economic measures. It was intended that the new plan would lead to further prevention-oriented legislation.

The Center for Nonsmoking and Health (CNSH) was organized in 1983 at the Minnesota Department of Health (MDH) to coordinate the statewide nonsmoking initiative.[3] CNSH staff collected and calculated Minnesota-specific data concerning the epidemiologic and health economic estimates of the disease impact of smoking, reviewed research literature, developed a database on the literature, and staffed the meetings of the technical advisory committee.

Because the statewide smoking prevention plan would ultimately be administered through MDH, it was decided to revise the department's in-house smoking/nonsmoking policy to make it more consistent with the new campaign. The updated policy would also be more compatible with the MDH's leadership role in public health. That is, through the revised policy, MDH would convey a public image of nonsmoking as normative and health-enhancing.

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The committee determined that the new policy would serve several functions. It would decrease nonsmokers' exposure to secondhand smoke, which is a recommendation of the National Research Council[4] and is supported by recent conclusions of the US surgeon general.[5] In addition to promoting a healthier work atmosphere for all employees, the policy would also serve as a model for other organizations wanting to adopt similar policies at their facilities. The committee also anticipated that MDH would realize economic benefits, such as reduced cleaning costs and a decrease in sick leave and medical expenses among employees.[6] Furthermore, research had indicated that worksite smoking policies might play a significant role in decreasing smoking prevalence among employees.[7]

Developing the smoking/nonsmoking policy was not an overnight occurrence; it involved a great deal of thought, organization, and committee work. The process, from the initial decision to revise MDH's policy to the actual implementation, took place over a 24-month period (Table I). Essentially, the process involved five stages: organization/policy development, information, initiation/transition, finalization, and evaluation. This paper chronicles the various stages of the process.

COMMITTEE ORGANIZATION

Successful planning and implementation of a new smoking/nonsmoking policy that was fair to all employees called for a representative committee of both smokers and nonsmokers. Five committee members were nominated and elected by fellow employees, and five were appointed by the Commissioner of Health. The ten-member committee included seven nonsmokers, several of whom were former smokers, and three smokers. Committee members were from various departments within MDH—represent-

TABLE I. Timeline for MDH Nonsmoking Policy

July 1984	Ten-member in-house committee organized to revise the department's smoking policy. Committee met approximately every two weeks from July 1984 to November 1984 to develop recommendations for a smoking policy more in line with MDH's role as a promoter of nonsmoking.
August 1984	All employees were asked to complete a survey about smoking status and give opinions about various policy options.
December 1984	Committee presented its recommendations to the Commissioner. New policy written.
Policy Implementation	
July 1985	Plans, promotions, and information concerning policy implementation communicated to all employees via in-house newsletter and memos from the Commissioner of Health. Employee reaction to new policy sought. Twenty percent of department vehicles designated as "smoking permitted" vehicles. Employees informed that if they wanted to smoke in a department vehicle they must request a "smoking permitted" vehicle. Smoking not allowed in other pool vehicles. Committee recommended that all new employees sign a statement to the effect that they had been informed of the policy and that they would comply with it.
August 1985	Staff planned and assessed cessation programs for employee use. Employee reaction to the new policy, which was printed in the previous month's in-house publication, appeared in staff newsletter.
September 1985	Smokers' lounge designated, smoking section in cafeteria reduced to 20% of the space. Smoking permitted in private and semiprivate offices if all occupants agreed. No change in bay area smoking policy. Smoking eliminated in all restrooms, locker rooms, entryways, in first floor lounge, and on the department's grounds. All ashtrays removed except in those places where smoking is allowed. "Quit and Win" smoking cessation kits made available for all interested employees. District offices informed that policy should be followed as closely as possible at those worksites. District offices also notified that smoking lounges will be established in July 1986.
October 1985	Information sessions on smoking addiction initiated to facilitate mutual understanding between smokers and nonsmokers. All interested employees, friends, and family members invited.
November 1985	Department arranged with a qualified MDH health educator to develop personalized smoking cessation plans for interested employees. Positive internal support system developed to help prevent relapse among those individuals who had quit smoking.
December 1985	Smoking cessation programs began to be offered at Central Office. Consistent with employee interest, cessation programs were offered through July 1987.
February 1986	Support groups initiated for employees who had quit smoking.
March 1986	Smoking eliminated from all work locations, including private and semiprivate offices, bay areas, and partitioned work spaces. Additionally, employees were informed that when representing the department at workshops or meetings, they are to refrain from smoking.
July 1986	Central Office smoking lounge was fully ventilated and smoking lounges were designated in each District Office. Smoking eliminated from the Central Office cafeteria. Overall evaluation of employee attitudes toward smoking policy and programs conducted through survey. Evaluation included analysis of attendance at information, cessation, and support group sessions. Additionally, complaints and resolution of complaints that went through the regular supervisory procedure were analyzed. Information sessions, cessation programs, and communication continued as needed through July 1987.

ing professional, technical, and clerical employees—as well as district and central offices. Additionally, two staff members were appointed by the commissioner to offer further guidance. This mix of individuals provided varying viewpoints and insights to the issue. They also offered different ideas on the logistics of organizing and implementing the smoking/nonsmoking policy.

The first task of the committee was to help design a survey to determine employee opinions about the adequacy of existing policy and about possible policy changes. Responses to the survey would also help in formulation of the policy.

Within a month, the completed survey was sent to all MDH employees. The three-page survey asked questions about the rights of both smokers and nonsmokers, where and if smoking should be restricted, and smoking habits among employees.

On completion of the survey, the first page, which included the respondent's name, was removed and sent separately to CNSH. This ensured that the respondents' answers would be anonymous. It also assured that the individual would not be contacted again for a response. Two weeks later, a second survey was sent to all employees who did not respond to the first inquiry. After another two-week period, a third and final survey was sent to nonrespondents. Overall, 564 of the 715 MDH employees (79%) responded to the survey. Individuals who did not respond in writing to the first, second, or third inquiry were subsequently contacted by phone and asked only whether they were smokers. As a result of the telephone follow-up, information was available on the smoking status of 99% of MDH employees.

Results from the survey indicated that 22% of MDH employees were smokers. This finding helped in determining the size of the nonsmoking area in the cafeteria during the transition stage of policy implementation, and in designating the number of smoke-free vehicles in the MDH car pool.

In the initial survey, only 30% of the 564 respondents thought the existing policy was adequate. Responses varied on where smoking should be prohibited. For example, 77% of the respondents thought smoking should be prohibited in bay areas, while employees were evenly divided on the issue of restricting smoking in private offices. Because fairness was of primary concern in policy development, the committee recommended that the same rules apply to all employees whether they were in bay areas or semiprivate or private offices.

To further assist in the development of the new policy, the committee reviewed nonsmoking policies from other organizations. In addition, committee members heard presentations by experts on policy implementation and ventilation issues (to help in designating smoking rooms with adequate ventilation). A smoking cessation program provider also addressed the nature of addiction and cessation.

In developing the policy, the committee met every other week from July 1984 through November 1984 to discuss issues related to the policy. Two subcommittees were organized to research and make recommendations in specific areas, such as communication of the policy, policy alternatives, and policy implementation. At the biweekly meetings, the subcommittees presented their findings and recommendations. Then the entire committee discussed the various options presented. Throughout the development stages, the committee tried to establish group consensus about policy recommendations.

The committee's ultimate goal was to create a policy that was fair to all employees—both smokers and nonsmokers. Therefore, policy recommendations were designed to protect the interests of both groups as much as possible.

COMMUNICATION OF POLICY

Five months after the committee first met, it submitted its recommendations to the commissioner. The committee's recommendations outlined measures that would be most effective in achieving nonsmoking by MDH employees on MDH premises and while representing the department off premises. In addition to proposing a more stringent smoking/nonsmoking policy, which went far beyond the 1975 Minnesota Clean Indoor Air Act (MCIAA), the committee submitted a timeline for policy implementation (Table I). For the most part, the commissioner agreed with the committee's recommendations. However, she introduced one additional provision which banned smoking on department grounds.

Prior to the actual policy implementation, there was a three-month period in which all employees were informed about the proposed changes through MDH's in-house newsletter and through memos from the commissioner to all employees. In addition, copies of the new policy were filed in the procedure manuals in each division, and copies were also available to all employees through the personnel office and the Center for Nonsmoking and Health.

One issue of the MDH newsletter used a question and answer format to address why the new policy was needed, how policy determinations were made, and where restrictions would apply. Employees were invited to respond to the proposed changes, and their comments appeared in the following month's newsletter. Furthermore, consultation with all major unions was maintained during the entire planning and implementation process. The goal was to keep employees informed on all aspects of the policy by communicating to them in a straightforward and open manner.

Consistent with the communication plan, a protocol was developed that required all new employees to be informed of the policy prior to beginning work at MDH and to be provided with a written copy of the policy. These individuals were required to sign a statement saying that they had read and agreed to abide by the policy before accepting employment at MDH. To further promote employee knowledge of related issues, literature on various topics such as cessation programs and quitting smoking was available through CNSH for any interested individual.

Throughout the entire process, there was an ongoing commitment to keep information and communication lines open. To promote a more cooperative spirit, employees were also encouraged to give input and recommendations to committee members.

The updated MDH smoking/nonsmoking policy was phased in slowly over a one-year period. Gradually, during that one-year time span, all smoking was eliminated in the building and on the grounds, with the exception of two small lounges for employee use only. A well-planned implementation program provided smokers with an opportunity to become accustomed to the changes.

For example, during the first month of implementation, 20% of the tables in the cafeteria were designated as smoking. This reflected the ratio of smokers to nonsmokers working at MDH. Along similar lines, 20% of the vehicles in the MDH car pool were designated as smoking. Also during the first month, smoking was eliminated in all restrooms, locker rooms, and entryways. Signs were posted at the building entrances stating that the building had designated smoking areas for employees, and that the public was not allowed to smoke on the premises. A large, immovable, sand-filled ashtray was also placed outside each entrance so that individuals could extinguish their smoking materials before entering the building.

COMMITMENT TO SUPPORT

Research has shown that a blending of a workplace smoking cessation program along with a smoking/nonsmoking policy presents a consistent message to employees.[8] Furthermore, Wilbur[9] maintains that offering cessation programs in conjunction with a clearly stated smoking/nonsmoking policy promotes and supports healthful practices among employees. Orleans and Shipley[7] indicated that smoking cessation programs in the workplace can be more effective than community programs if they utilize the peer support network within the organization as well as other reinforcements for nonsmoking such as a smoking/nonsmoking policy. They also explain that worksite smoking cessation programs play a vital role in the national antismoking campaign.

As part of the policy at MDH, the committee recommended that the department, by assorted approaches, establish an ongoing commitment to support employees who chose to quite smoking. In December 1985, the six-month mark of policy implementation, department smoking cessation programs began. Provisions were made for assisting employees and members of their households to participate in a variety of smoking cessation programs. Several of the on-premises classes were offered during the initial stages of implementation. However, as employee interest declined, the classes were discontinued.

Employees who wanted to quit smoking with the help of an outside smoking cessation program could attend any approved program, and the department would reimburse 75% of the fees to a maximum of $100 through CNSH. Employees could repeat programs or participate in as many different cessation programs as they wanted during the implementation period, and for up to one year after policy implementation. Support groups were also initiated in December 1985 to prevent relapse among employees who quit smoking. Support groups and reimbursement for attendance at smoking cessation classes continued through July 1987.

In the ninth month of policy implementation there was an evaluation of the provision that designated the number of MDH pool vehicles in which smoking was allowed. Also, smoking was eliminated in bay areas, partitioned space, semiprivate offices, and private offices.

Provisions for months 12 through 23 included establishing a fully ventilated smokers' lounge and eliminating smoking areas in the cafeteria. During the final month of policy implementation, a second survey was distributed among employees to determine the adequacy and acceptance level of the new policy.

POLICY ACCEPTANCE

In general, there was a high level of acceptance of the recommended and implemented changes. The commitment to keep all individuals informed of changes on an ongoing basis, and the support offered to smokers in their efforts to quit smoking contributed to the overall acceptance of the policy.

1986 SURVEY RESULTS

In July 1986, a second survey was conducted using procedures similar to those employed during the first survey. The survey was undertaken to evaluate employee attitudes toward the newly implemented nonsmoking policy. Some 88% of the 668 employees responded to the second survey. A majority of the 1986 respondents (73%) thought the updated policy was adequate, compared to 30% of the 1984 respondents who thought the policy was adequate. A large percentage of respondents (89%) thought that for the most part employees were complying with the policy.

Interestingly, 25 employees reported quitting smoking since the 1984 survey (conducted 11 months prior to policy implementation), and 17 of those individuals had quit since the policy went into effect in July 1985. Thirty-two percent (N = 8) of those who quit smoking since the 1984 survey indicated that the policy was somewhat or very influential in their decision to quit. Eleven employees participated in and ten completed the UNsmoke smoking cessation course in 1986. Of those individuals, eight had stopped smoking at the end of the course, and five were still not smoking at the 12-month follow-up.

Results from the 1986 survey also indicated that 21% of the employees were current smokers. Of those individuals responding to the survey, 30% indicated that they had smoked less since the policy went into effect, and 77% of those reported that the policy had had some effect on their reduction of smoking.

TECHNICAL ASSISTANCE

In 1984, as part of the tobacco use prevention plan, the state legislature approved funding for one position at MDH to provide technical and consultation services for implementation of the MCIAA and its rules. The CNSH staff also provides technical assistance to private and public agencies that want to implement a smoking/nonsmoking policy. As nonsmokers increasingly demand clean air at work, employers are requesting assistance in interpreting and applying the MCIAA. In addition to consultation and assistance provided through CNSH, a variety of publications are available for any Minnesota organization that wants to develop and implement a smoking policy.

SMOKING POLICY ACTIVITY IN OTHER STATES

MDH recently conducted a survey of other state health departments to assess their involvement in the planning and implementation of worksite smoking policies. All 50 states returned the questionnaire. Of these, 64% (N = 32) indicated that they had been at least moderately involved in the development or planning of worksite smoking legislation in their state.

Forty-six percent of the states (N = 23) were without any worksite smoking legislation, but six of these had bills pending in the state legislature. In those states with mandated smoking policies, the policy content varied considerably, and only six state health departments indicated any involvement in enforcement of worksite legislation. Twenty-one states assigned staff time to consult with worksites in the development of smoking policies, but the average staff assignment was slightly less than one half-time position.

Internally, about 88% (N = 44) of all state health departments have a written smoking policy. Of these, nine ban smoking altogether. There were clear regional differences, with the southern and southeastern states generally less involved in policy consultation or statewide legislation.

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The smoke-free hospital: A smoke-free worksite

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Smoking begins as a social behavior and is continued as a response to addictive and social pressures. Reduction of smoking prevalence will require the replacement of a culture that accepts and encourages smoking with a culture that encourages smoke-free lifestyles and environments. It is possible that smoke-free worksites are an effective way to create and reinforce the concept of a smoke-free culture. Hospitals are labor intensive environments and, given their health-related mission, are one ideal starting point for the development of smoke-free worksites.

The social sciences anthropology and sociology provide models of analysis that can be used to develop an understanding of why smoking in hospitals persists despite the known health hazards, and how the anachronistic policies can be changed. This paper reports insights about the movement to make hospitals smoke-free that may be useful to future policy makers.

CULTURE OR CULTURAL ARTIFACT?

There has been a great deal of attention devoted to the integration of preventive services into the acute care hospital. However, this attention has not produced the desired results. This lack of success may be due to the fact that social systems like those supporting the maintenance of smoking in hospitals are complex systems that have not been influenced by simple efforts at change. To illustrate the importance of this point, I ask the reader to consider four objects (Fig 1). The first is a fly whisk from Egypt made from horse hair, leather, and bone. The second is a pair of Berber slippers made from leather and wool. The third is a prosthetic hip joint, and the fourth is a computer diskette with a label that says "Cardiac Risk Reduction."

Each object provides information about the culture

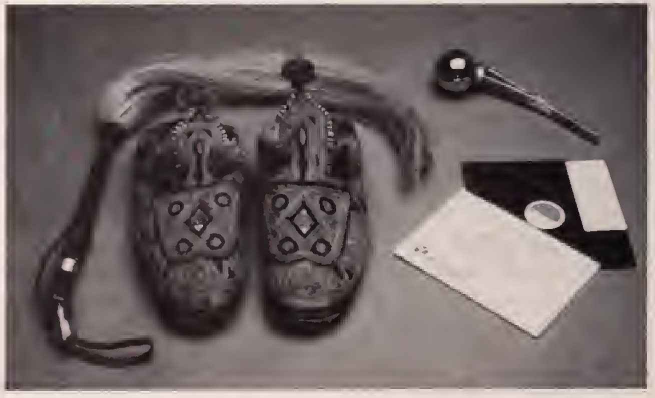

FIGURE 1. While it is easy to recognize that articles from foreign cultures are incomplete representations of the culture from which they arise, it is more difficult to recognize that phenomenon for one's own culture. The incorporation of health promotion into acute care hospitals will require far more than isolated artifacts such as computer-assisted instruction.

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Presented in June 1987 at a workshop in Bethesda, Md, "Smoking Policies in the Workplace: Research Needs and Potential Applications," sponsored by the Division of Lung Diseases, National Heart, Lung, and Blood Institute; the Institute for the Study of Smoking Behavior and Policy at the John F. Kennedy School of Government at Harvard University; and the National Cancer Institute.

from which it arises. The fly whisk tells us that the Egyptians have horses, that they know of elephants, and that they work with leather. The slippers tell us that the people of the Northern Sahara like decoration on their shoes, and that they know how to dye wool and work with leather. The hip joint tells us that medical science makes use of metallurgy. The diskette tells us that those who teach heart disease prevention skills like smoking cessation use high technology devices.

In examples such as the whisk and slippers, which are not related to the culture of medicine, it is easy to see that, while these cultural artifacts give information about a culture, they do not describe the culture in its entirety. In the case of the prosthetic hip joint, it is also easy to recognize that information other than that which can be gained by examining the hip joint is required in order to understand when and how surgeons use the device. However, in the case of health promotion initiatives, individuals tend to believe that the discovery of a single important artifact (eg, the usefulness of computer-assisted instruction) is equivalent to the discovery of a cultural system. Specifically, it has been postulated more than once that a computer program in a doctor's office should be sufficient to get patients to stop smoking.

Recognition of a System and How it Works

If a system is to be modified to accept new constructs, the change agent must first recognize that a system is present and understand how the system works. Consider the parable of the five blind men and the elephant, which is among the favorites of Western cultures (Fig 2). The five blind men disagree about what they are feeling. Is it a leaf? Is it a wall? Is it a tree stump? Is it a snake? Is it nothing at all? This parable exemplifies the problem related to a multi-attribute system.

Instead of trying to rank the attributes in order of importance, change agents should shift their strategy to the development of a "laundry list" of factors that have proved to be important in one case or another. They can then integrate the attributes into a single system, rather than disjointed components of a system. Once the system has been described, one can develop a rational action plan.

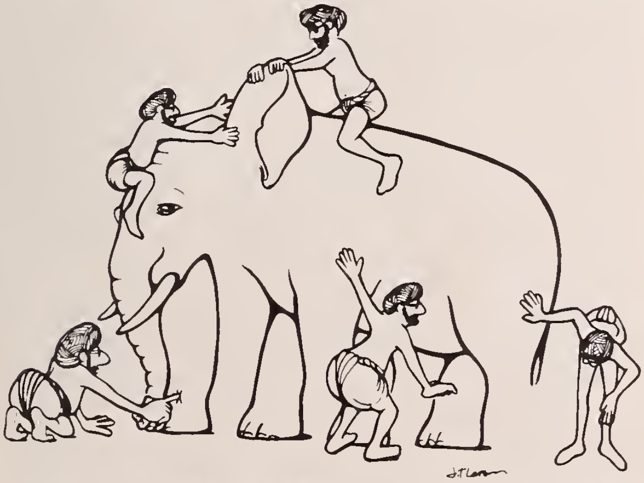

FIGURE 2. The parable of the five blind men and the elephant frequently arises during problem identification sessions. Its appearance should be used as a clue that the problem represents a multi-attribute system.

Identifying the Barriers to Smoke-free Hospitals

Medical change agents are frequently confronted with the situation in which a physician fails to adopt an intervention known to benefit most patients, in this case, smoke-free hospitals (Fig 3). The change agent's usual response is to assume that the physician is the primary obstacle (Fig 3, pathway A). This approach is not helpful. It "blames the victim"[1] by not distinguishing between physicians who want smoke-free hospitals for their patients but cannot change their environment (secondary defect) and those who do not care whether or not hospitals are smoke free (primary defect). On the other hand, pathway B, examining the physician's environment for factors that affect adoption, has been shown to be a useful strategy.[2] The factors that influence adoption include knowledge of clinical relevance, skills, organization, adequate return, perceived demand, perceived effectiveness, perceived legitimacy, confidence, and commitment (Table I). The remainder of the paper will discuss the barriers as they relate to smoke-free hospitals.

Knowledge of Clinical Relevance. Unless the smoke-free hospital is perceived as relevant to patient care, adoption cannot be expected.[3,4] In the case of smoke-free hospitals, the physician can easily be provided with information that documents this relevance. A 1984 survey at the University of Minnesota found that 38% of patients agreed with the statement, "When smoking is permitted in the hospital it leads me to believe that doctors and nurses approve of smoking."[5]

Skills. The change agent needs the skills to identify the goals of the hospital and phrase arguments in terms that are consistent with these goals.[6] Everett Rogers[1] examined several hundred studies of diffusion of innovations and found properties that discriminated between innovations that diffused rapidly and innovations that did not diffuse. The properties he discovered included relative advantage, compatibility, complexity, "trialability," and observability (Table II). The innovation must be shown to be compatible with the social environment in which the hospital operates, so that little overall change is required to accommodate the intervention. Becoming smoke free

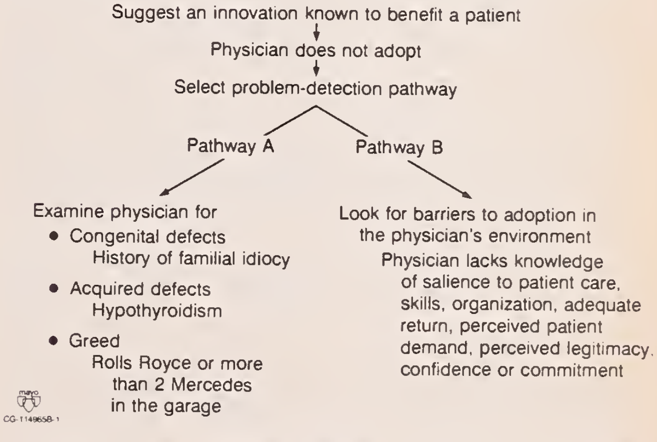
Suggest an innovation known to benefit a patient
Physician does not adopt
Select problem-detection pathway
Pathway A
Pathway B
Examine physician for
• Congenital defects
History of familial idiocy
• Acquired defects
Hypothyroidism
• Greed
Rolls Royce or more than 2 Mercedes in the garage
Look for barriers to adoption in the physician's environment
Physician lacks knowledge of salience to patient care, skills, organization, adequate return, perceived patient demand, perceived legitimacy, confidence or commitment
mayo
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FIGURE 3. Problem identification can take two paths when a phsyician fails to adopt an innovation known to benefit a patient. Pathway A assumes that the physician is the primary cause. It "blames the victim" and is rarely successful. Pathway B examines the physicians' environment for barriers to adoption. It is far more likely to be associated with success.

TABLE I. Barriers to Implementation of a Smoke-Free Hospital*

Knowledge of clinical relevance. The physician does not understand or accept how the patient's health will benefit from the prevention intervention.

Skills. The physician lacks the skills to deliver the intervention effectively.

Organization. There is no system to cue the physician to intervene or to follow patients who have received an intervention. Physicians lack guidelines for intervention.

Adequate return. The intervention loses money for the physician or requires financial subsidy.

Perceived patient demand. The physician does not believe that patients want the intervention.

Perceived legitimacy. The physician does not believe that preventive intervention is part of the professional role.

Confidence. The physician lacks confidence to deliver the preventive intervention.

Commitment. The physician lacks commitment to deliver the intervention and to practice preventive medicine.

*Adapted from Kottke et al.[2]

must be shown to be simple to implement (low in complexity). The idea of a smoke-free environment must be offered to the hospital on a temporary basis before requiring permanent adoption without trial. Finally, the hospital must be able to observe the positive results of becoming smoke free. These five properties should be used by the change agent as a checklist to describe and package the process so that it is optimally acceptable to the hospital.

Studying the Black Power and Pentecostalist movements, Gerlach and Hine[7] described five attributes of successful social movements: organization, recruitment, commitment, ideology, and opposition (Table III). Their studies imply that hospitals attempting to become smoke free should be organized so that decision making is decentralized, giving hospitals the opportunity to exchange and share ideas freely. The change agent recruits new hospitals through face-to-face contact, demonstrating how the innovation will satisfy the hospital's own needs. Hospital commitment is engendered by the combination of the change agent's demonstrating commitment and the hospital's successfully practicing the innovation. A "party line" or key phrase provides a shorthand definition of the innovation for the hospital, for example, "Smoke-free care is quality care." A feeling of opposition is engendered to the needless death and disability from smoking which results from suboptimal medical care that ignores smoking as a problem.

TABLE II. Key Attributes of Rapidly Diffusing Innovations*

Relative advantage is the degree to which an innovation is perceived as better than the idea it supersedes.

Compatibility is the degree to which an innovation is perceived as consistent with the existing values, past experiences, and needs of potential adopters.

Complexity is the degree to which an innovation is perceived as relatively difficult to understand and to use.

Trialability is the degree to which an innovation may be sampled on a limited basis.

Observability is the degree to which the results of an innovation are visible to others.

*From Rogers.[1(pp238-240)]

TABLE III. Key Elements of Successful Social Change Efforts

Organization. Successful social change efforts are based on decentralized decision making with the hospitals or groups of hospitals freely communicating and exchanging ideas among themselves.[7(p33)]

Recruitment. "The type of structural relationship (brother, spouse, parent, fellow church member, neighbor, patron, peer) is less important to understand recruitment to a movement than (a) the frequency of interaction between recruiter and potential member or (b) the effect of the relationship (positive or negative) on the potential convert."[7(p84)] Effective recruitment is based on face-to-face encounters that meet one or more of the hospital's needs.

Commitment. Commitment is a social phenomenon consisting of seven steps: initial contact with a participant; focus of needs through demonstration; re-education through group interaction, decision and surrender; the commitment event; testifying to the experience; and group support for changed cognitive and behavioral patterns."[7(p110)]

Ideology. "The 'party line' provides patterned answers to questions that will be raised . . . and provides confidence and assurance in the face of opposition."[7(pp162-163)]

Opposition. Opposition serves to focus the attention and activity of the hospital.[7(p183)] In the case of health and health care, opposition can be the opposition to unnecessary death or pain and suffering, or it can be opposition to suboptimal or inefficent health care practices.

Organization. Eliot Freidson[8(p xi)] has written extensively on the organization of medicine and has observed that "occupational organization constitutes a dimension quite as distinct and fully as important as its knowledge." Knapp et al[9] have studied the organization of hospitals and the decision making process that applies to the adoption of a smoke-free policy. Knapp's studies can help the change agent understand the hospital and avoid needless mistakes.

Adequate Return. The hospital must be able to expect adequate return on hospital investment in the innovation. Wells et al[10] found that physicians cannot be expected to subsidize smoking interventions. Hospitals appear to behave in a similar manner, and must be satisfied that they will not lose patients if they become smoke free. To date, the positive comments about the Mayo Clinic's smoke-free facilities, for example, have far outweighed the negative comments (personal communication, Kenneth G. Berge and Richard D. Hurt, 9/15/88), and the Mayo Clinic continues to prosper in a contracting market environment.

Perceived Demand. Physicians expect patient demand for services, because patient demand rewards the physician's ego, and because it protects him or her from charges of "medicalizing."[8(p355-356)] A 1984 survey found that 40% of patients said they would view the hospital more favorably if it were smoke free, and only 8% said they would seek health care at another facility if smoking were not permitted.[3] It is important for a hospital to realize that a certain proportion of patients would also be attracted to a smoke-free hospital. Eighty-three percent of patients in the survey agreed with the statement, "A smoke-free hospital would be an improvement in medical care." Selecting among policy options, 53% of the respondents chose "no smoking anywhere in the hospital," and another 20% chose "smoking only with the permission of a physician."

The population appears to want smoke-free hospitals. Even though a majority of patients want a smoke-free hospital, a certain level of dissent must be expected. In the 1984 survey quoted above, one quarter of the patients agreed with the statement, "Relatives of patients deserve the right to smoke in the hospital."[4]

Perceived Effectiveness. Hospitals must perceive that becoming smoke free improves patient care, and that they will be able to undertake the task successfully. The change agent might point out to physicians that 69% of patients in the hospital survey described above agreed with the statement that they would stop smoking if told to by their doctor.[3] In a randomized trial by Geoffrey Rose, smoking declined more than 50% as the result of four 15-minute interventions over the period of one year.[11] The Mayo Clinic became smoke free with little problem, and 56 of 166 general hospitals in Minnesota were smoke free as of July 1, 1988 (Judy Knapp, personal communication, 8/1/88).

Perceived Legitimacy. While smoking in private is a First Amendment right, smoking in public is not. The legitimacy of limiting smoking has been challenged. Thirty-seven percent of the patients responding to the University of Minnesota survey agreed with the statement, "Even though some patients may benefit from a smoke-free hospital it is unfair to ask other patients not to smoke in the hospital."[3] The public must be informed that while smoking in private is a right, the right to freedom from involuntary side-stream smoke takes precedence. The surgeon general has documented the risk of side-stream smoke,[12] and, in a jointly signed letter, the surgeon general and the director of the Health Care Financing Administration have asked the hospitals that provide Medicare services to become smoke free.[13] Smoking in public is no more a First Amendment right than other nuisances such as the industrial pollution of air and water.

Confidence. Confidence is necessary for action, and the hospital gains confidence through the knowledge that others have already accomplished the task. The change agent should point out that hospitals like Loma Linda have always been smoke free, and that the Indian Health Service and Mayo Clinic are now smoke free.

Commitment. Times change, knowledge changes, and hospitals must also change. Outcomes that were once acceptable can become unacceptable on the basis of new knowledge. In the past, over 17% of women giving birth died of puerperal fever.[14] In the past, dogs were allowed in the operating room. Wangensteen and Wangensteen noted that in 1830, when a Scottish peasant woman, Ailie, had a breast removed for cancer without anesthesia, by the Scottish surgeon, John Syme, "[Ailie] entered the theater . . . accompanied by her husband James and their dog Rab . . . her pale face showed the pain but she was still silent. Rab's soul was working within him . . . blood flowing from his mistress . . . his ragged ear was up . . . he growled . . . James held him firm."[15] Syme was an outstanding surgeon for his day but, like his peers, he did not recognize that a dog held over the operating field was a source of sepsis.

Later, E. S. Bishop[16] wrote, "Let us not convert our theaters into semblance of Mohammedan harems or Spanish torture chambers nor terrify our patients out of what little courage they still possess." He was not writing of prohibiting smoking in the hospital; he was arguing that he should not have to wear a mask and cap in the operating room. Finally, Harvard University's David Edsall[17] described a teacher of his: "Already an ardent advocate of antisepsis, [he] would regularly scrub his hands thoroughly and after that, he would reach into his trousers pocket for a pocket knife, scrape beneath his finger nails, replace the knife, and proceed to operate." All of these practices, once considered acceptable, are now thought of with horror. Smoke-free hospitals deserve the commitment accorded to sterile operating fields and safe childbirth.

Commitment comes through successful practice of the innovative concept. Patients expect and demand the change agent's commitment. In the 1984 survey of the University of Minnesota Hospitals, 85% of the patients agreed with the statement, "Doctors, nurses and others who work in the hospital should set an example for patients and visitors by not smoking."[3] As Gerlach and Hine[7] observed, "Commitment is a social phenomenon that cannot occur or cannot be maintained in a social vacuum."[7] In Cleveland, the American Heart Association, the American Lung Association, the March of Dimes, and the American Cancer Society committed themselves to a city-wide public effort to reduce the burden of smoking on pregnant women. As a result, the Rainbow Children's Hospital in Cleveland is now smoke free.

CONCLUSIONS

The implementation of smoke-free policies in hospitals is feasible if the social needs of hospitals are considered. This means satisfying the hospitals' need for salience to patient care, skills, organization, adequate return, perceived patient demand, perceived legitimacy, confidence, and commitment. The same principles apply to other worksites. If these tools, developed by the observers of social change and innovation,[1,6,7] are used, the implementation of smoke-free worksites is feasible.

It was because of men like Semmelweiss, who declared in 1854, "There must be no poison on the physician's hands,"[18(p416)] that hospitals post such signs as, "Labor and Delivery—No Admittance," and women no longer die of puerperal sepsis. Surgeons are not prodded to observe sterile technique; those with unacceptable infection rates lose their privileges. In the introduction to their book, *The Rise of Surgery*, Wangensteen and Wangensteen[18(p4)] quote Nobel laureate Maurice Maeterlinck, "At every cross-way on the road that leads to the future, [tradition] has placed against each of us, 10,000 men to guard the past." Smoking in hospitals and other worksites is a cultural anachronism. When we properly address this problem, all persons must decide for themselves whether they are Maeterlinck's traveler on the road to the future or are one of the guardians of the past.

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Implementation of a smoking policy in the United States Army

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The purpose of this article is to share the chronology of events and experience of the United States Army in conducting an intensive antismoking campaign and establishing new smoking restrictions during the period from March 1986 through February 1987.

BACKGROUND

Secretary of Defense Weinberger issued a memorandum[1] for the Secretaries of the Military Departments on March 10, 1986, directing that an intense antismoking campaign be conducted at all levels of all services, and that it become a major responsibility of all commanders, up to and including the highest levels of each service and the department.

Department of Defense (DOD) Directive 1010.10, Health Promotion, dated March 11, 1986, established health promotion policy within the DOD to improve and maintain military readiness and the quality of life of personnel. The directive placed increased emphasis on educational programs regarding health risks associated with smoking, use of drugs and alcohol, diet, lack of exercise, stress, and hypertension.

The DOD smoking prevention and cessation program aimed at creating a social environment that would support abstinence and discourage the use of tobacco products, create a healthy work environment, and provide smokers with assistance in quitting. Smoking was prohibited in auditoriums, conference areas and classrooms, buses, vans, elevators, eating areas, common work areas, and areas lacking adequate space and ventilation to ensure a healthy environment for nonsmokers. Participation of the DOD with manufacturers or distributors of tobacco products is not permitted in promotional programs, activities, or contests aimed primarily at DOD personnel.

A DOD Working Group on Smoking was established to enhance communication among the services, provide a basic framework for the campaign, and review progress on implementation plans. The group met until a DOD Health Promotion Coordinating Committee was established and assumed responsibility for the campaign as part of the overall Health Promotion Program.

The framework of the DOD antismoking program, completed in April 1986, established program goals and organized the services' programs into a coherent and comprehensive approach. The original goal of the DOD smoking reduction program was to reduce the smoking rate among personnel on active duty by 5% per year over the coming two years and to reduce the DOD beneficiary smoking rate to civilian rate levels within five years. By November 1986, this goal was updated[2] with the aim of reducing all tobacco use rates to levels significantly below civilian rates, and to reduce smoking rates of personnel on active duty by at least 10% per year. The objectives of the smoking reduction program were to provide information on smoking reduction, motivational and educational programs to DOD beneficiaries, and smoking cessation assistance programs, and to specify designated places for smoking in order to minimize nonsmokers' exposure to tobacco smoke.

The Deputy Secretary of Defense, in an April 14, 1986,

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Presented in June 1987 at a workshop in Bethesda, Md, "Smoking Policies in the Workplace: Research Needs and Potential Applications," sponsored by the Division of Lung Diseases, National Heart, Lung, and Blood Institute; the Institute for the Study of Smoking Behavior and Policy at the John F. Kennedy School of Government at Harvard University; and the National Cancer Institute.

The opinions and views expressed here are those of the author and do not necessarily reflect the position of the Department of the Army.

memorandum[3] for the secretaries of the military departments, outlined a policy on the age requirement for the purchase of tobacco overseas. Effective October 1987, the authorized age for the purchase of tobacco products in the overseas military retail system would be not less than 18 years of age. This policy applied to personnel on active duty, family members, and other authorized patrons of the military resale outlets.

ARMY ANTI-TOBACCO CAMPAIGN

The army anti-tobacco campaign was kicked off on April 29, 1986. The campaign was preceded by a two-day workshop held in March 1986 with representatives of the army, voluntary organizations, and federal agencies. A comprehensive game plan and objectives for the campaign and the policy changes related to smoking in the workplace were developed at the workshop.

The emphasis of the army's anti-tobacco campaign was on voluntary cessation by educated and informed soldiers, civilian employees, retirees, and family members. Informational and educational programs were developed to stress the dangers of tobacco use and the benefits of quitting, and to support and encourage individuals who wanted to quit. A special poster series was developed around the theme of "quit for the health of it." In collaboration with the American Cancer Society, free cessation clinics and materials for personnel in Korea, Japan, and Europe were made available.

The army's smoking policy became effective on July 7, 1986. The use of tobacco products was banned in basic training and was restricted in other military courses. Army training centers and service schools included information on the effects of tobacco in their curriculum. The policy established "no smoking" as the norm, prohibiting smoking except in designated areas. A smoking area could be so designated only if the "secondhand" smoke from tobacco products could be sufficiently isolated to protect nonsmokers from its effects. Clearly this represented a major philosophic shift from previous policy, which required that "no smoking" areas be posted. (An army regulation controlling smoking in army-occupied space had been in effect since 1977.)

In November 1986, the army celebrated its first annual "Great Army Smokeout" in concert with the American Cancer Society's tenth annual "Great American Smokeout." The news media were very supportive of this undertaking, and the national emphasis on reduction of the use of tobacco products was an invaluable asset for this event.

The army anti-tobacco campaign had three main components: information, education, and policy. The campaign encompassed community, worksite, and clinical strategies, and was developed to meet the following objectives:

- To foster a total army family fully aware of the detrimental consequences of tobacco use and the benefits of not using tobacco products.
- To develop, implement, and review programs and policies to combat and eliminate tobacco use with initial focus on cigarette smoking cessation.
- To reduce significantly the use of all tobacco products and to meet the national goal by the year 1990.

The objective of the information program was to heighten awareness, establish a new tobacco-free image as desirable, and provide basic information on the dangers of smoking. The strategy employed was an information program based on developing community awareness through the use of a media blitz. Following are examples of media-related opportunities:

- Disseminate information regarding the nature of the health risks from tobacco usage and the immediate benefits of stopping.
- Promote distribution of information relevant to high risk smokers, such as pregnant women, those with multiple health risks secondary to life style, and those with potential job-related exposures worsened by smoking.
- Provide periodic giveaway attention-getters, including buttons, refrigerator magnets, and bumper stickers during community activities.
- Disseminate program posters, flyers, news briefs, and service announcements at least quarterly.
- Support the annual "Great American Smokeout" campaign in cooperation with the American Cancer Society each November.
- Utilize public service announcements on closed circuit television and radio.

The educational component of the program was designed to remove the glamour from tobacco use, create a positive image of stopping tobacco use, provide practical information for nonsmokers and smokers, and disseminate information on the negative effects of tobacco use. The strategy encompassed the following:

- *Training:* Strategy was aimed at providing general information classes regarding tobacco use during basic and advanced training, enlisted personnel education/training courses, and all long term training programs. This included the Military Academy and the unit training schedule as well.
- *Command information briefs:* Strategy was aimed at disseminating information briefs suitable for use by personnel in leadership positions, including personnel engaged in recruiting and conducting ROTC, Reserve, and National Guard training.
- *Classes:* Strategy was aimed at providing smoking and tobacco use information classes for the community at large. Closed circuit television instruction on tobacco and smoking for units, worksites, and family housing areas may be a means of providing this information.
- *Print material:* Strategy provided ready access to printed information suitable for the target groups.

Cessation services rely heavily on the support of programs and materials of voluntary organizations, as well as local community resources. The highest priority for cessation services is to promote independent cessation, through self-help approaches, group interventions, and one-to-one counseling. The combination of these services with an aggressive education and mass media information campaign and a smoking restriction policy in the workplace formed the nucleus of the campaign.

Progress was analyzed from three perspectives: assessments by commanders of major commands, trends in tobacco sales, and soldier opinion as determined by an army-wide survey. Overall, commanders felt that the smoking restriction policy was fair. The most common difficulty encountered by commanders was providing smoking areas due to space limitations. Cigarette sales declined by 10.4% between March and December 1986 in the commissaries serving army personnel.

The DOD worldwide surveys provided the baseline data for evaluation of the DOD anti-tobacco campaign. The 1980, 1982, and 1985 data stemmed from a representative worldwide sampling of each of the four services. The survey instrument is a self-administered questionnaire covering drug, alcohol, and tobacco use, with the focus on knowledge, attitude, and behavior. The most recent worldwide survey was conducted in 1988. The army "Survey of Soldier Opinions about Tobacco Use" was conducted in February 1987, and included a sampling of approximately 13,000 soldiers stationed in the United States and overseas. This survey was a self-administered questionnaire and was concerned only with tobacco issues.

Soldiers were found to be knowledgeable about many of the specifics of the army policy. Eighty-two percent of the enlisted soldiers and 90% of the officers knew that smoking was prohibited except in designated areas. Sixty-two percent of enlisted soldiers and 81% of officers surveyed agreed or strongly agreed with the smoking restrictions. More than half of the soldiers who smoked reported that they would like to quit. Preliminary results suggested that the overall smoking rate dropped from 52% in 1985 to 41% in 1987. Caution, however, was exercised concerning comparisons of data from the DOD worldwide surveys and the army survey because of differences in the questionnaires, sampling methodology, and field procedures.

In summary, the United States Army implemented a comprehensive anti-tobacco campaign in 1986. The campaign was built on a three-pronged approach: information, education, and a smoking restriction policy. The data received so far suggest that this comprehensive approach will reduce tobacco use in the military. Efforts to refine and improve the campaign are ongoing. Time will tell if this approach can change the image and habits of soldiers concerning tobacco use.

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Economic issues related to smoking in the workplace

MARVIN M. KRISTEIN, PHD

In the last 25 years since the first surgeon general's report on smoking, the proportion of adult smokers in the United States population has declined from about 45% (more than 50% of adult males) to about 25% (less than 30% of adult males).[1-4] In the last ten years since *Shimp v New Jersey Bell*[5] (the first legal victory assuring an employee's right to protection from involuntary smoking), dozens of states and localities have established regulations regarding smoking in the workplace. In a 1986 survey of industry, the Bureau of National Affairs found that almost 60% of employers had adopted and/or were considering policies dealing with smoking in the workplace.[6] A 1987 follow-up survey found that almost 80% of employers had or were considering restrictions on smoking in the workplace; total bans had risen from 6% to 12%; open work area smoking bans increased from 41% to 51%.[7] As Erikson[8(p97)] has said, " . . . a workplace smoking control program should definitely be considered good business."

The clinical and epidemiologic evidence regarding the adverse health (and other) effects of smoking on the smoker are overwhelming to all but those in the pay of the tobacco industry. While the evidence regarding passive or involuntary smoking is less extensive, and may remain so because of the nature of the studies that would be required, the existing studies are consistent and getting stronger.[9-11] There is a real and present danger to the health and well-being of passive smokers, who now outnumber smokers almost three to one in the US population. For example, "Never smokers married to smokers had about a two-fold increased risk of lung cancer."[12] Further, a 1985 Gallup poll found that almost 90% of those sur-

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veyed (including 80% of the smokers) agreed that a workplace smoking policy was necessary.[6(p8)]

COSTS OF SMOKING

Deriving the costs of smoking involves "economic epidemiology" calculations of the health impact on smokers and involuntary smokers plus a range of indirect health costs, such as fire risk, damage to property and furnishings, cleaning costs, workers' compensation, disability, retirement, accidents, life insurance, absenteeism, productivity losses, occupational health risks (especially involving synergies with smoking), passive smoker effects, etc. The best data available regarding costs involve the "valuing" of national epidemiologic data, special occupational health studies, a few detailed studies of cost areas, including time and motion studies, and extrapolations of these numbers. In terms of preventable illnesses, premature mortality, and other costs that reduce the Gross National Product, we may reasonably estimate that smoking is costing the US society as a whole more than $60 billion per year.[6(p7),13] The costs to an individual employer would depend on multiple factors. (One must be careful about extrapolating from "macro" studies for answers to "micro" problems and policy—especially in the health care fields.) However, the larger an employer and the longer the time period of concern, the more one may defend the assumption that the employees being studied represent an average population. A 1983 paper presents average macro data extrapolated to a per smoker basis and related to the typical employer's share of these costs.[14] When these figures are adjusted for inflation, the typical smoking employee in 1988 cost the typical employer at least $1,000 per year in total excess costs (as compared to employing an otherwise similar nonsmoker); more than half of these costs are indirect health costs and, therefore, should be recoverable in a shorter period than health care costs).[15]

The supporting data and evidence regarding the cost consequences of smoking are distinct from the evidence on the health consequences of smoking. But though we may be less certain of the exact numbers to assign to the financial cost of smoking, we should not be misled. The costs of smoking to individuals, employers, and the community are very real and significantly greater than zero. One of the problems in this area is that we have been unwilling to spend the resources in time and money to evaluate the cost issue as compared to other issues regarding smoking. Also, we would need many studies, particularly of workplaces, because of the problems associated with a small sample size and the high degree of variability to be expected.

Employers appear to be increasingly convinced that smokers are in fact costing them money. Further, several company-sponsored studies of smoking cessation efforts (largely as part of health promotion programs) indicate significant decreases after two to three years in terms of absenteeism, plus strong hints of lower medical care costs. The relatively low cost of many workplace smoking restriction and control policies along with high rates of employee approval have convinced many that the gains to an employer may easily cover the costs. Studies of actual employer savings of smoker-related costs and other benefits will depend on the passage of time and the willingness to pursue the research. Presently, most programs are too young, and carefully designed studies are rare.

Several issues should be considered in planning future studies of workplace smoking policies: types of programs, costs and benefits, and some popular confusions.

WORKPLACE PROGRAMS

There are at least three different categories of workplace programs of interest to employers: those that restrict smoking in the workplace; those that provide cessation programs for smoking employees; those that use both of these approaches as part of a health promotion program.

Restricting smoking in the workplace can range from putting up a few "no smoking" signs in certain or all indoor locations, to an extensive restructuring of the workplace to accommodate smokers and nonsmokers, to a total ban of smoking in the workplace as well as a policy of hiring only nonsmokers. The General Services Administration has estimated that a program to restrict smoking in all federal governmental facilities, with concern for the comfort of smokers who wish to continue smoking, but preventing harm to nonsmokers, would cost approximately $10 per employee as a one-time expense.[16] Similar costs were estimated for similar programs for a few major corporations. One large company with 2,500 employees reported a cost of up to $50 per employee, reflecting the need for restructuring and erecting partitions and implementing extensive corporate "culture massage."[6(pp50ff)] Costs may be further increased by paying for smoking cessation programs for smokers. Some localities and businesses have reported virtually zero costs for implementing their public and/or workplace smoking restrictions. Many employers may be concluding that a total ban of smoking in the workplace is the cheapest and most effective way of dealing with this issue. (Pacific Bell and Michigan Bell are examples of companies utilizing this approach.)

BENEFITS AND COSTS

A key problem in the economics of prevention is that costs are clear in the present, while the benefits are realized slowly in the uncertain future. A total ban may minimize present costs and should achieve most of the short-run indirect health-related gains. One may be able to obtain substantial savings in terms of reducing cleaning costs (already reported by smoke-free hotels), fire risk, accidents, damage to furnishings, harm to nonsmokers, time wasted by smoking rituals, etc. There is a great deal of evidence that, generally, the costs of compliance with smoking restrictions are quite low, compliance rates are high, and the confrontation scenarios predicted by the Tobacco Institute are almost nonexistent. If we are not perfectionist in expectations and are reasonably sensitive to local cultural conditions, banning smoking in the workplace may be highly cost effective. For certain types of companies, in which workplace risks are highly synergistic with smoking, banning workplace smoking is particularly compelling. For example, asbestos workers who smoke increase their illness risk by 11 times over that of nonsmoking asbestos workers, whose risk is five times that of nonsmokers who did not work with asbestos.

Capturing Benefits. Whether or not a given employer may indeed capture any cost savings from restricting smoking in the workplace depends on a variety of factors, including the nature of the smokers, the type of program instituted, the nature of the workplace, labor turnover, and the time horizon. From an economic point of view, smokers are drug addicts engaged in a polluting, unhealthy behavior. Their behavior increases their risk of imposing high health care costs on themselves, their employers, and society. Their insistence on smoking in public adds an additional dimension of social costs (analytically similar to those added by drug addicts who steal to support their habits). The share of a given employer in paying these costs varies, as does his potential for gain from their reduction. For example, from the point of view of society, many insurance payments are a form of transfer payments and not a real cost; from a single employer's point of view, a dollar saved is a dollar earned.

Whose Cost, Whose Benefit? Cost-benefit analyses are tricky, especially in the case of complicated social programs. It is important to consider the question of whom it is costing and whom it is benefiting. For example, an employer may save money on his current employee population as a result of a smoking restriction program that reduces smoking by those employees. In the longer run, more of these employees may survive into (longer) retirement. These survivors may cost their employers and/or society much greater sums than the employer saved in terms of the social security system (a transfer payment not a real cost to society) and Medicare (a real cost).

Some studies suggest that a single company will save more over the longer run by not promoting the health of its employees in any way, since the retirement and longer life medical care costs overwhelm any savings in present expenses due to unhealthful life styles. Since smoking helps kill people relatively early, this argument is particularly relevant to efforts to reduce smoking. While the broader aspects of this line of confused and confusing thinking are discussed below, at this level it points to the need not only to be clear about whose benefit and whose cost, but also about the particular pocket or account to be debited or credited. It should be noted that defending smoking as a way of protecting pension systems is more socially inefficient and crueler than simply poisoning a selective group of the population over 65 chosen by lottery.

BENEFITS OF A SMOKE-FREE SOCIETY

Prevention vs Cure. In recent years a number of unhelpful confusions, particularly in the area of health care, have become popular. Some of these involve the misapplication of good economic thinking.

Of special concern to studies of the prevention of illness and health promotion is the confusion regarding savings and benefits. It is generally argued that proposed prevention programs must be expected to demonstrate positive net present value in order to be supported. This is basically unfair to prevention programs. It is rare that any established medical procedure is supported by a cost-benefit analysis; it is even rarer that "cure" is properly compared to prevention. Thus, smoking cessation efforts, which average a 20–25% success rate, are regarded as primitive as compared to lung cancer "cure," with a bare 5% survival rate (and research funding for serious work reflects this prejudice even more strikingly). In fact, regarding spending on medical care as an addition to the Gross National Product is inaccurate. *Consumer Reports* reduces the rating of an automobile in direct relation to the defects it shows and the repairs it needs. Similarly, all else being held constant, the more medical care a group needs, the poorer it is. Even if the cost of preventing an illness were equal to the cost of treating and curing it (and curing is very rare for major chronic diseases), prevention would be more beneficial. Illness, even if cured, is a strong negative for all involved. Good health has value beyond the costs of the illnesses it eliminates. A person in his early 60s who recently survived bypass surgery may be better off than being dead or a total invalid, but it cannot compare with being in good health and never having had a heart attack at that age.

LONG RUN VS SHORT RUN

A number of analyses of what is wrong with current American business competitiveness in the world economy point to a business school overemphasis on short-run rates of return and the drive for quick gains in our highly competitive financial markets. These analyses suggest that we may be sacrificing longer range planning and strategic thinking to short-run tactics. This is a particularly difficult issue in dealing with prevention in a democratic society. For example, economically, tobacco use involves a very small group currently enjoying great economic gains at the expense of great longer-run economic and non-economic costs spread thinly over a large population. The gainers have spent and are spending huge sums to recruit and maintain a group of unfortunate addicts who are the major losers—and this has been developing over the last 70–80 years. A recent study suggests that the tobacco industry is fighting a tough battle to sow controversy, confusion, conflicting analyses, etc. It would be unwise to play with such ground rules. Especially in regard to the prevention of illnesses we cannot cure, we must take a long-run planning view.

A COMING DISASTER

One key point in the area of illness prevention is to remember that we have a rapidly aging population. The peak birth cohorts of the "baby boom" generation will be reaching 65 years of age in the years 2020 to 2030.[17] Given a continuation of current clinical and epidemiologic relationships regarding medical and hospital care usage by age groups, the problem of medical care spending and availability of providers at that time will make our current situation look like the Garden of Eden. Unless we start working hard now to prevent the range of chronic diseases that can be prevented (and smoking is the lead item), we are headed for a major disaster in our health care system. Businesses and governments will share in this disaster, as they now do in supporting our current system.

Competitive Markets and Social Values. It has been suggested that efficient labor markets really eliminate the excess costs of employing smokers vs equivalent nonsmokers by reducing the wages of smokers to reflect their extra costs to the employer. This would leave the costs of smoking to society intact, but the labor market would compensate for the costs of a smoker. This is a bit like arguing that, in an efficiently functioning market, the public would pay less for a given manufacturer's product if he were a polluter than for a similar product made by a nonpolluter. This would be necessary in a normative argument, since the polluter is probably polluting because it is cheaper to do so, and he might be able to drive the nonpolluter out of business profitably. One begins to suspect that the reason we have laws about pollution is that our market system rarely compensates for such costs and does not of itself protect nonpolluters from polluters. Highly competitive markets rarely support social and humane interests and values.

For markets to eliminate the excess cost of smokers to their employers, a number of assumptions would be required—all of which appear to be unreasonable in the overwhelming number of real workplace situations.

First, a given employer would have to know the exact cost attributable to a given smoker in his given place of employment at a given point in time and over a period of time. Second, that cost would have to be the same for all smoking employees (or a functional relationship would be needed). Third, the labor market would need to be organized on a piecework or hourly basis, and hiring would have to be done on a longshoreman-type shape-up basis. In addition, there could be no standard wage rate, nor a contract running longer than the time period in which the costs of smoking change. Finally, all employers in a given labor market would have to discriminate against smokers uniformly. It is remotely possible that when all employers have imposed a uniform ban on smoking in the workplace and a ban on hiring smokers, the labor markets would then find the remuneration of smokers discounted by their excess costs to employers. Until then, we may be reasonably sure that smokers are involving both their employers and society in extra, avoidable, costs.

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Rainier Bancorporation's policy on smoking*

PETER BROFFMAN, JD

Rainier Bancorporation and its subsidiaries employ approximately 6,000 people in approximately 200 offices. While its primary place of business is the state of Washington, Rainier also has offices in Alaska, Oregon, California, Hawaii, Arizona, New York, and the Far East.

The corporation's policy on smoking was implemented in two phases. Phase I, a one-year period that began in October 1985, was a transition phase. During this period, smoking was restricted to designated areas, as determined by the manager of each department or work unit. When the policy was announced, employees were also advised that at the end of the transition phase, smoking would become prohibited. Rainier offered in-house smoking cessation classes and subsidized enrollment in outside smoking cessation programs.

Phase II was implemented October 1, 1986. At that time, smoking by employees was prohibited in all of Rainier's domestic facilities.

Three factors gave rise to the development of the policy. First, Rainier's Social Policy Committee, which examines the company's position with regard to contemporary social issues, requested an examination of smoking in the workplace as part of an overall concern with employee health and wellness. Second, the company's Employee Relations Department had received an increasing number of complaints by employees about smoke in the workplace. Third, the legal and regulatory climate was clearly changing. The state of Washington had adopted a Clean Indoor Air Act that prohibited smoking in public places, including the public areas of banks. A prohibition on smoking in private work space was narrowly defeated, but the action clearly sent a warning to employers that unless they reduced or eliminated smoke through voluntary policies, the legislature would mandate the action by law. At the same time, an increasing number of courts, including those in Washington, were upholding the right of the employee to a smoke-free workplace.

POLICY DEVELOPMENT

The first step in the process of policy development at Rainier was to conduct research on the issue and to prepare findings and recommendations for Rainier's Management Committee. This research, conducted by two members of the Employee Relations Department, was aided considerably by use of a workbook entitled *It Can Be Done: Creating a Smokefree Workplace.* The book is published by the Center for Health Promotion at Group Health Cooperative of Puget Sound, the largest health care provider in the state of Washington. The book was specifically designed to acquaint employers with issues surrounding smoking in the workplace, and gives practical suggestions on the research, development, and implementation of a workplace smoking policy.

Rainier's research covered health issues; cost of smoking with regard to absenteeism, insurance, maintenance, and productivity; legal considerations; employee relations issues; policy alternatives; and information on how other companies have handled the issue. The recommendation of the employee relations specialists was to adopt a policy that would prohibit smoking. They recommended a transition period of six months to phase in the program.

The recommendation was presented to the Management Committee for approval. This committee, composed of the 18 most senior officers of the company, tends to adopt major policies through a consensus-building process. While the opinion of the chief executive officer is most persuasive, the style of management is to build broad management support for a policy prior to its adoption. Such was the case here.

At the outset, some of the committee members were in complete support of the recommendation. A few were in clear opposition. Most, however, wanted time to explore the ramifications before making a commitment. After a few months, however, it became clear that the leadership and the majority of the Management Committee supported the policy. It was adopted with one significant modification. Since some members felt that a longer transition period was necessary before the company became completely smoke free, they agreed on a one-year period.

During the deliberations by the Management Committee, there was little debate about the basic rationale regarding employee health, estimates of the economic benefits of a policy, or the legal issues. Also, there was no request for employee survey data or air quality studies. Almost all concerns were of a sociopolitical nature. A significant issue was whether a private employer had the ethical right to legislate the personal habits of employees. While most members of the Management Committee un-

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* As of January 1, 1989, Rainier Bancorporation's name has been changed to Security Pacific Bank Washington.

derstood Rainier's desire to protect the rights of nonsmokers, a few suggested that the company had an obligation to protect the perceived rights of smokers. This was expressed as sentiment for a policy less restrictive than a total ban on smoking.

In consideration of the sensitive nature of this issue, the company was careful at all times to emphasize that Rainier would not be telling employees not to be smokers. It was attempting to prohibit *smoke* in the workplace, not smokers, and did not presume to tell employees what personal habits to engage in outside the office. Employers have always had the right to control the physical environment and to direct, through policy, the behavior of employees while at the workplace. Just as an employer can ban the consumption of alcohol or the playing of loud music, so can it ban the production of smoke.

In addition to the general concern, specific issues about practical considerations were raised by some members of management:

- Would the company really take disciplinary action against an otherwise good employee, particularly one who brings in a lot of money? (Yes. This would be enforced like any other company policy regarding employee conduct in the workplace).
- Would the company enforce the policy in the executive suite? (Yes. Executive management would be expected to provide leadership in implementation of the policy.)
- How would it look to have employees standing outside smoking in front of buildings during breaks? (It may present a problem in a few locations, but would be minimal. Employees would be encouraged not to stand in front of building entrances while smoking during breaks.)

These concerns were addressed to the satisfaction of the Management Committee. It became clear that while there was a risk of some negative aspects to implementation, the negative factors could be minimized and should be considered reasonable tradeoffs when weighed against the benefits of the policy.

When it came time to announce and implement the policy, Rainier made a conscious decision to keep it well publicized, but relatively low key.

IMPLEMENTATION

There were some initial protests by employees who smoked when the policy was announced, but they did not last long. A few employees were genuinely concerned about the physical and emotional stress they anticipated going through. There was a brief flurry of enrollment in the smoking cessation classes. For the most part, however, all this was short-lived. In fact, after the initial burst of interest, Rainier cancelled a number of smoking cessation classes for lack of interest. The Employee Relations Department received no formal complaints about the policy.

Two problems arose early in the implementation of Phase I, but they were eventually resolved. One problem was disputes about the specific areas that some managers designated as smoking areas. These disputes ordinarily occurred in the smaller branch offices, where there were few options on the allocation of space. A second problem was that in offices in which the manager was a smoker, enforcement of the policy was lax. In both cases, these problems were short lived. Some were resolved by involvement of the Personnel staff. For the most part, the problems simply disappeared with the implementation of Phase II, the prohibition of smoking.

Six months into Phase I, Rainier conducted a companywide survey to ask how employees felt about the implementation of the policy. Sixty-four percent of the employees responded, and the vast majority supported the policy.

When it came time to implement Phase II, it was done in a very low-key, matter-of-fact manner. A simple reminder was sent to employees that smoking would be prohibited in all work areas. There was, as with the initial announcement of the policy, a brief flurry of minor protest, lasting only a couple of days. There were no formal complaints and no problems in recruiting, and there have been very few comments since, either positive or negative. The policy seems to have been accepted and is no longer a matter of discussion. The only remaining comments that the company receives are isolated expressions of concern that, at some offices, employees do stand outside and smoke, which presents a negative image to customers. The problem is minimal and is considered a reasonable tradeoff for what has otherwise been a very positive experience.

RESEARCH NEEDS AND ISSUES

Although research was valuable in helping to develop a policy, Rainier's employee relations specialists do not believe that further research is needed to justify maintaining the policy.

The company recognizes two issues that would arise in formulating a model for follow-up research. First, how does one isolate this policy as the reason for a change in costs to the organization? For example, with respect to health care costs, Rainier has a work force that is generally becoming more attentive to health and wellness. In addition, the company is about to restructure its health benefits program. If health care costs or absenteeism due to sick leave are reduced, can one isolate this policy as the reason? At best, should it be considered as only one contributing factor? Similarly, with regard to maintenance, Rainier is undergoing a significant change in maintenance procedures that have nothing to do with this policy. The company expects the changes to reduce costs significantly. How, then, does one factor in this policy?

A second issue is, how does one conduct research on behavior change in an industry that has high turnover rates? The banking industry nationwide experiences approximately 20% annual turnover. What methods can be used to conduct long term research on behavior change when the work force at the end of the research period may be an entirely different group of people than at the beginning of the research period?

These are issues for health care researchers to consider when studying the effects of a corporate policy on smoking such as the one implemented at Rainier Bancorporation.

Assessing the effects of long term nicotine chewing gum use

Nicotine chewing gum is a relatively recent aid available to smokers to help them quit their smoking habit. It is, in general, an effective approach when used as part of a controlled smoking cessation program. Previous studies have shown it to be a particularly effective aid for more dependent, heavier smokers. Authors Hajek, Jackson, and Belcher evaluated the long term use of nicotine chewing gum in a smoking cessation clinic (*JAMA* 1988; 260:1593–1596).

Earlier reports found 2–9% of gum users were still using the gum after one year, and that stopping chewing the gum after long term use provoked many of the same symptoms associated with cigarette withdrawal. Hajek et al sought to clarify these findings based on their follow-up of 538 participants in a smoking cessation program. The group treatment program lasted four weeks. Participants were encouraged to use nicotine chewing gum (2 mg) for up to three months, chewing as many as 15 pieces per day.

"One-year success was defined as sustained abstinence from any form of tobacco use following the treatment . . . Long-term gum use was defined as any use of gum at one year." At one year of follow-up, 6.3% of the group were still using the gum, and 25% of successful quitters still relied on the gum.

The authors found a significant correlation ($p < 0.01$) between the quantity of gum chewed by long term users at one year follow-up and both pretreatment carbon monoxide levels and cigarette consumption. Whereas the manufacturer of the gum recommends its use for three months, the successful quitters in this study used the gum for an average of 5.6 months. The weight gain among long term gum users was significantly ($p < 0.05$) less than among other treatment successes.

The authors concluded from these and other findings that "although there is a chance that a heavy smoker who manages to quit with the help of the gum will go on using it beyond one year, in our opinion recommending the gum remains well justified." Among long term gum users, it appears that their dependence on nicotine has been transferred from cigarettes to the gum. While the authors do not advocate long term gum use, they note that since the gum does not contain many of the harmful components of tobacco, such as tar and carbon monoxide, and it emits no pollutants, it is preferable for long term gum users to continue to chew the gum rather than reverting to cigarette smoking.

"Smokeless cigarette" faces legal challenge

R.J. Reynolds is test marketing its new nicotine-based product, Premier, in Arizona and Missouri. Describing the product as a new delivery system for the drug nicotine, the American Medical Association (AMA), Arizona Medical Association, Missouri State Medical Association, and three county medical societies in those states filed legal petitions with the Arizona State Board of Pharmacy and the Missouri Department of Health to stop the sale of Premier.

The petitioners are requesting that the product be shown to be safe for human consumption before it can be offered for sale. Nicotine, the petition states, is "a psychoactive and addictive drug which has been implicated in cardiovascular disease, complications of hypertension, reproductive disorders, cancer and gastrointestinal disorders." As a drug or drug delivery system, the nicotine product is required by Arizona and Missouri state laws to be proven safe, according to the AMA and participating medical associations and societies.

With regard to the questionable safety of Premier, AMA Executive Vice President James H. Sammons, MD, speaks for the association, saying, "On this issue, we are simply unwilling to accept on faith the new product of an industry that still denies that smoking is unhealthy."

On receipt of the petition, the Arizona State Board of Pharmacy unofficially asked R.J. Reynolds to withdraw the product voluntarily. The company refused.

Heavy vs light smokers: Is there a difference in cigarette dependence?

Cigarette smoking has been likened to a drug dependency state, in which nicotine is the addictive agent. A tobacco withdrawal syndrome has been described; it encompasses a variety of physiologic and psychologic changes that accompany tobacco deprivation. While there is evidence to suggest that heavy smokers are more dependent on cigarettes than light smokers, few researchers have examined the differences between heavy and light smokers, and the available findings have been inconclusive.

"Our group is currently conducting a large-scale trial examining the effectiveness of psychological and pharmacologic approaches to smoking-relapse prevention presented in self-help treatment formats," explained Killen et al (*JAMA* 1988; 260:1581–1585). Prior to participation in this study, subjects had to quit smoking for 48 hours.

The authors collected data on "measures of tobacco dependence and a variety of other psychological and behavioral variables that may influence cessation of smoking or maintenance of the habit." They defined heavy smoking as at least 25 cigarettes smoked per day and light smoking as no more than 15 cigarettes per day. Prior to beginning the study, the authors hypothesized that heavy smokers would find it harder to quit smoking, would exhibit more negative mood states, and would be more dependent on cigarettes, compared to light smokers.

In a baseline telephone interview conducted before the cessation program actually began, interviewers asked the potential participants questions about their smoking history, personal characteristics such as age, gender, and marital status, and medical status, as well as questions

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NEWS BRIEFS are compiled and written by Vicki Glaser.

aimed at assessing the person's dependence on tobacco. These latter questions dealt with times and circumstances in which people smoked, depth of inhalation, and ability to not smoke when it is not allowed.

Following the 48-hour period of abstinence from smoking, the participants completed a survey that assessed their height and weight, tobacco withdrawal symptoms, cravings, and difficulty in abstaining, and included a profile of mood states questionnaire and a family environment scale.

With respect to smoking history, heavy and light smokers did not differ significantly according to the number of years they had smoked, the age at which they had started smoking, the level of nicotine in the type of cigarettes they smoked, and the number of times they had previously tried to quit.

Several physiologic and behavioral differences were found. Heavy smokers weighed more and were more obese than light smokers, reported more dysfunctional mood states, were more dependent on cigarettes, had more difficulty quitting smoking, and experienced more intense cravings and withdrawal symptoms following abstinence.

"Our findings suggest that smoking-cessation programs designed for heavy smokers may need to include strategies enabling them to cope with the effects of physical dependence." Such programs might benefit from aids such as nicotine chewing gum.

How the Canadian health care establishment overcame the tobacco lobby

The Tobacco Products Control Act, recently passed by the Canadian government, bans all tobacco-related advertisements in newspapers and magazines published in Canada. In addition, the new law forbids the use of tobacco product brand names in association with sporting or cultural events. This practice will be phased out over a three-year period. In the future, all cigarette packs must include a description of the health hazards of smoking and the toxic chemicals produced by tobacco smoke (*JAMA* 1988; 260:2480–2481).

Passage of this legislation represents an important victory by the health care establishment and antismoking organizations over the powerful tobacco lobby. A concentrated lobbying effort spearheaded by the Canadian Medical Association (CMA), the Canadian Cancer Society, and the Nonsmokers' Rights Association—a consumer organization—is credited with attaining passage of the law. Individual involvement by thousands of CMA members, via telephone calls to members of the parliament, repeatedly emphasized the public health benefits of the legislation, and successfully countered the heavily funded campaign waged by the affected tobacco and publishing companies.

"Rothmans, Benson and Hedges, Inc, Canada's second largest tobacco company, has asked Canada's federal court to overturn the legislation, claiming the federal government wasn't authorized to pass it and that it violates the right to freedom of expression. The government has not presented 'any credible evidence' that tobacco advertising affects tobacco consumption," according to the attorney representing the company.

However, government officials have cited declines in teenage smoking, for example, in countries such as Iceland that have instituted bans on tobacco advertising. While officials hope that the advertising ban will affect how much people smoke, they must contend with the fact that about 75% of the magazines available in Canada are published in the United States, which permits tobacco advertising.

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Charles Felix Banas, MD, Buffalo. Died September 13, 1988; age 76. Dr Banas was a 1937 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. He was a member of the American Academy of Allergy, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Louis Baron, MD, Stony Brook. Died October 12, 1988; age 78. Dr Baron was a 1938 graduate of the Medizinische Fakultaet Rheinischen, Friedrich Wilhelms Universitaet, Bonn, Germany. He was a Fellow of the American College of Obstetrics and Gynecologists. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Ernest H. Bettmann, MD, White Plains. Died September 22, 1988; age 89. Dr Bettmann was a 1923 graduate of Universitaet Leipzig Medizinische Fakultaet, Saxony, Germany. He was a Diplomate of the American Board of Orthopedic Surgery. His memberships included the American Academy of Orthopaedic Surgery, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Joseph R. Cally, MD, Catskill. Died July 2, 1988; age 64. Dr Cally was a 1952 graduate of the New York Medical College, New York. Dr Cally was a Qualified Fellow of the International College of Surgeons and a Diplomate of the American Board of Surgery. He was a member of the Medical Society of the County of Greene and the Medical Society of the State of New York.

Hugh Peter Davis, MD, Sarasota, Florida. Died August 30, 1988; age 88. Dr Davis was a 1927 graduate of McGill University Faculty of Medicine, Toronto, Ontario, Canada. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Guerrino Deboni, MD, Liverpool. Died October 12, 1988; age 68. Dr Deboni was a 1948 graduate of Facolta di Medicina e Chirurgia dell'Universita di Padova, Padova, Italy. He was a Diplomate of the American Board of Obstetrics and Gynecology. His memberships included the American Fertility Society, the Onondaga County Medical Society, and the Medical Society of the State of New York.

Angelo C. Ferrara, MD, New York. Died October 15, 1988; age 57. Dr Ferrara was a 1958 graduate of the Facolta di Medicina e Chirurgia dell'Universita di Roma, Rome, Italy. He was a Fellow of the American Academy of Pediatrics and a Diplomate of the American Board of Pediatrics. His memberships included the New York Academy of Medicine, the American Academy of Allergy, the American Pediatric Society, the New York County Medical Society, and the Medical Society of the State of New York.

Alton Anthony Germain, MD, Attica. Died October 8, 1988; age 71. Dr Germain was a 1945 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. His memberships included the New York Academy of Medicine, the American Academy of Family Physicians, the Aerospace Medical Association, the American Geriatrics Society, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Joseph Gertner, MD, Brooklyn. Died October 4, 1988; age 84. Dr Gertner was a 1930 graduate of the Faculte de Medecine de l'Universite de Nancy, Nancy, France. His memberships included the American Rheumatism Association, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Leo Golub, MD, Norwalk, Connecticut. Died April 1988; age 61. Dr Golub was a 1973 graduate of the Facultad de Medicina de la Universidad Autonoma de Guadalajara, Guadalajara, Mexico. He was a Fellow of the American College of Physicians. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Edward Calisch Held, MD, Hempstead. Died October 19, 1988; age 87. Dr Held was a 1926 graduate of the Johns Hopkins University School of Medicine, Baltimore, Maryland. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Roy T. Johnson, MD, Jamestown. Died September 21, 1988; age 64. Dr Johnson was a 1948 graduate of the University of Tennessee Center for Health Sciences, Memphis, Tennessee. He was a Fellow of the American Academy of Family Physicians. He was a member of the Medical Society of the County of Chautauqua and the Medical Society of the State of New York.

Leon Linder, MD, Purchase. Died April 13, 1988; age 76. Dr Linder was a 1938 graduate of the Medizinische Fakultaet der Universitaet Wien, Wien, Germany. His memberships included the American Society of Anesthesiologists, Inc, the American Geriatrics Society, the Bronx County Medical Society, and the Medical Society of the State of New York.

Melvyn Robert Miller, MD, Forest Hills. Died June 5, 1988; age 79. Dr Miller was a 1928 graduate of the Facolta di Medicina e Chirurgia dell'Universita di Padova, Padova, Italy. His memberships included the American Psychiatric Association, the Academy of Psychoanalysis, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Isaac Y. Peress, MD, Roslyn. Died September 28, 1988; age 75. Dr Peress was a 1938 graduate of the Medical School, American University of Beirut, Beirut, Lebanon. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

S. Jerome Rauch, MD, Pelham Manor. Died July 3, 1988; age 84. Dr Rauch was a 1929 graduate of the New York

University School of Medicine, New York. He was a Fellow of the American Academy of Family Physicians. His memberships included the New York Cardiological Society, the American Geriatrics Society, the Bronx County Medical Society, and the Medical Society of the State of New York.

Leo Paul Reckford, MD, New York. Died October 19, 1988; age 85. Dr Reckford was a 1928 graduate of the Universitaet Wien, Medizinische Fakultaet, Wien, Austria. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

William F. Regan, Jr, MD, New Rochelle. Died October 25, 1988; age 62. Dr Regan was a 1953 graduate of Tufts University School of Medicine, Boston, Massachusetts. He was a Diplomate of the American Board of Ophthalmology. His memberships included the American Academy of Ophthalmology and Otolaryngology, the Pan American Association of Ophthalmology, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Carl Reich, MD, New York. Died September 29, 1988; age 88. Dr Reich was a 1924 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a Fellow of the American College of Physicians, and a Diplomate of the American Board of Internal Medicine and the American Board of Pathology. His memberships included the New York Academy of Medicine, the New York Pathological Society, the International Society of Hematology, the New York County Medical Society, and the Medical Society of the State of New York.

Maxwell Spring, MD, Bronx. Died October 25, 1988; age 75. Dr Spring was a 1938 graduate of the New York University School of Medicine, New York. He was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. His memberships included the New York State Society of Internal Medicine; the Clinical Society, New York Diabetes Association; the Society of Nuclear Medicine; the American Society of Clinical Hypnosis; the Bronx County Medical Society; and the Medical Society of the State of New York.

Irwin Daniel Stein, MD, Milford, Massachusetts. Died September 13, 1988; age 81. Dr Stein was a 1931 graduate of the New York University School of Medicine, New York. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Earle W. Voorhees, MD, Poughkeepsie. Died October 8, 1988; age 91. Dr Voorhees was a 1922 graduate of the University of Pennsylvania School of Medicine, Philadelphia. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Otolaryngology. His memberships included the American Academy of Ophthalmology and Otolaryngology, the American Academy of Otolaryngology, the Dutchess County Medical Society, and the Medical Society of the State of New York.

Robert Lee Yeager, MD, Pomona. Died October 2, 1988; age 81. Dr Yeager was a 1931 graduate of the University of Pennsylvania School of Medicine, Philadelphia. He was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. His memberships included the New York Society for Thoracic Surgery, the American Public Health Association, the American Thoracic Society, the Medical Society of the County of Rockland, and the Medical Society of the State of New York.

ERRATUM

Based on information provided by the New York County Medical Society, the November issue of the *Journal* incorrectly reported the death of Dr Milton Earle Eisen of Manhattan.

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1988; 88:456-462). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith^2 reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al*. Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83:925-927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics*, ed 12. Philadelphia, WB Saunders Co, 1983, pp 337-338.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Pascal James Imperato, MD, Editor, *New York State Journal of Medicine*, PO Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

FOR MY PATIENTS

NUMBER 6/1989

WHEN I PRESCRIBE A DRUG FOR YOU— HOW TO PREVENT PROBLEMS

Dear Patient,

There are many possible problems that can arise when you take prescription drugs. In order to reduce your chance of having a bad reaction and to make sure the drug will work properly, it is wise to observe the following suggestions carefully.

INTERACTIONS

Before I hand you a prescription for a drug, you and I should discuss any other medications or drugs you may be taking. That may prevent serious interactions that sometimes can occur between certain drugs taken on the same day. For example, antacids can interfere with the absorption of tetracycline antibiotics taken shortly afterward. Some combinations of two different drugs might cause side-effects such as drowsiness.

If you drink alcohol regularly or plan to consume alcohol at an upcoming party, there might be a bad interaction with the drug you will be taking. We should talk about that.

ALLERGIES AND OTHER REACTIONS

I need to know of any drug allergies you have or any kind of drug reactions you may have had in the past; for example, if you ever developed headache, rash, nausea, or diarrhea from any drug.

If you are planning a visit to the beach in the summer or a trip to a sunny vacation area, we should consider whether the drug you will be taking is photosensitizing. That is, will your skin sunburn quickly and severely. For example, drugs containing sulfa compounds or tetracyclines are photosensitizing. I may then advise you to use sunblockers on your skin, or I may prescribe a different drug.

We should discuss what reactions, if any, the drug might cause. Some drugs will have a package insert which you should read to learn about possible reactions. Follow the instructions exactly as written on the label of the bottle. Ask your pharmacist if it can be taken before, during, or after meals.

If you think you may be having a reaction, call me and stop taking the drug immediately. If the reaction seems serious, such as wheezing, dizziness, or faintness, call me right away.

If you experience a major reaction to the drug, I will report it to the drug's manufacturer or to the Food & Drug Administration. If enough reports of serious side-effects are received by them, the drug may be removed from the market.

STORAGE AT HOME

Before storing any unused pills or liquid in your medicine chest, ask your pharmacist to look up the expiration date of the drug, which you should then write on the bottle's label. Otherwise, it is a safe rule to discard all leftover prescription drugs after one year. And remember that it is dangerous to share your prescription drugs with friends or relatives.

PROTECT YOUR CHILDREN

If you have any young children at home, ask your pharmacist to fill all your prescriptions using child-resistant bottle caps. Whenever possible, only buy medicines, such as aspirin, which are sold in bottles with child-resistant caps.

Also, if you have any young children at home, store all drugs and medicines under lock and key or high out of the reach of the young ones. Keep the telephone number of the local Poison Control Center handy in case your child accidentally swallows any of the drugs. Buy a bottle of *syrup of Ipecac* to be used if an emergency arises where the Poison Control Center or I advise you to make the child vomit immediately.

By the way, it's a good idea to fill all your prescriptions at the same pharmacy because they keep a file of all the drugs you are taking and can warn you of any possible interactions between them.

MEDICAL SOCIETY OF THE STATE OF NEW YORK
ORGANIZED 1807

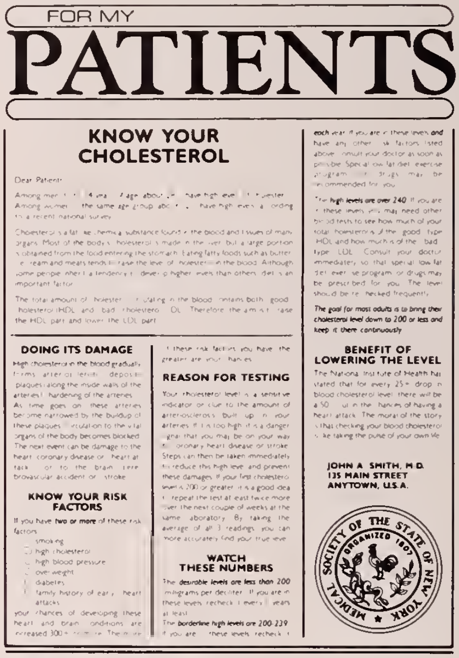
Office Handouts for Your Patients

This issue of the *New York State Journal of Medicine* contains another "For My Patients" letter, which appears on the preceding page.

Use this letter series to keep your patients informed about timely issues concerning diseases, treatments and health maintenance. Simply make photocopies of the page and distribute them to your patients with your newsletter or other correspondence, or place them in your reception area for your patients to read and take home.

MSSNY and the *New York State Journal of Medicine* have waived the copyright for these handouts, so you may duplicate them without permission.

As an added feature, MSSNY will personalize this form and make copies for you, at a nominal charge. See the example below for how these forms would look with your name and address professionally printed on them. Use the order form below, and we'll ship the personalized forms to you as soon as possible.



FOR MY
PATIENTS

KNOW YOUR CHOLESTEROL

Dear Patient:

DOING ITS DAMAGE

KNOW YOUR RISK FACTORS

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- Just one easy-to-swallow caplet q.i.d. may encourage compliance.
- Prompt, effective relief with minimal drowsiness.* Clinical studies have shown that patients respond by the end of 24 hours following initiation of therapy.

Or with an analgesic

- Formulated without an analgesic, for maximum treatment flexibility.
- Can be used with the analgesic of your choice, according to the patient's need for pain relief.
- Scored caplet permits greater dosing flexibility for patients requiring lower daily doses.

*Clinical studies have shown that most patients remain alert when chlorzoxazone-containing products are administered at recommended doses, although drowsiness sometimes occurs.

Please see following page for brief summary of Prescribing Information.

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Parafon Forte DSC
#40
Sig: 1 caplet q.i.d.

The following is a brief summary only. Before prescribing, see complete prescribing information in PARAFON FORTE DSC labeling.
CONTRAINDICATIONS: PARAFON FORTE DSC chlorzoxazone is contraindicated in patients with known intolerance to the drug.
WARNINGS: The concomitant use of alcohol or other central nervous system depressants may have an additive effect. *Usage in Pregnancy.* The safe use of PARAFON FORTE DSC chlorzoxazone has not been established with respect to the possible adverse effects upon fetal development. Therefore, it should be used in women of childbearing potential only when, in the judgment of the physician, the potential benefits outweigh the possible risks.
PRECAUTIONS: PARAFON FORTE DSC chlorzoxazone should be used with caution in patients with known allergies or with a history of allergic reactions to drugs. If a sensitivity reaction occurs such as urticaria, redness, or itching of the skin, the drug should be stopped. If any symptoms suggestive of liver dysfunction are observed, the drug should be discontinued.
ADVERSE REACTIONS: After more than twenty-six years of extensive clinical use of chlorzoxazone-containing products in an estimated thirty-two million patients, it is apparent that the drug is well tolerated and seldom produces undesirable side effects. Occasional patients may develop gastrointestinal disturbances. It is possible in rare instances that chlorzoxazone may have been associated with gastrointestinal bleeding. Drowsiness, dizziness, lightheadedness, malaise, or overstimulation may be noted by an occasional patient. Rarely allergic-type skin rashes, petechiae, or ecchymoses may develop during treatment. Angioneurotic edema or anaphylactic reactions are extremely rare. There is no evidence that the drug will cause renal damage. Rarely, a patient may note discoloration of the urine resulting from a phenolic metabolite of chlorzoxazone. This finding is of no known clinical significance. Approximately thirty-six patients have been reported in whom the administration of chlorzoxazone-containing products was suspected as being the cause of liver damage. In one case, the jaundice was subsequently considered to be due to a carcinoma of the head of the pancreas rather than to the drug. In a second case, there was no jaundice but an elevated alkaline phosphatase and BSP retention. In this patient there was a malignancy with bony and liver metastases. The role of the drug was difficult to determine. A third and fourth case had cholelithiasis. Diagnosis in a fifth case was submassive hepatic necrosis possibly due to abusive use of the drug for approximately one year. The remaining cases had a clinical picture compatible with either a viral hepatitis or a drug-induced hepatitis. In all these latter cases, the drug was stopped, and with one exception, the patients recovered. It is not possible to state that the hepatitis in these patients was or was not drug-induced.
Full directions for use should be read before administering or prescribing.
For information on symptoms and treatment of overdosage, see full prescribing information.

12/16/87

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952

(continued from p 6A)

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Feb 18–25. **Sixth Annual Office Based Sports Medicine Conference.** 25 Cat I Credits. Sun Valley. Contact: Extended Programs in Medical Education, School of Medicine, University of California, San Francisco, CA 94142-0742. Tel: (415) 476-4251.

KENTUCKY

Feb 19–24. **20th Family Medicine Review—Session I.** Hyatt Regency Hotel, Lexington. Contact: Joy Greene, Continuing Medical Education, 132 College of Medicine Office Building, University of Kentucky, Lexington, KY 40536-0086. Tel: (606) 233-5161.

LOUISIANA

Feb 25–26. **Second Annual Congress of the North American Society of Phlebology.** Fairmont Hotel, New Orleans. Contact: North American Society of Phlebology, 3960 Park Blvd, Suite E, San Diego, CA 92103. Tel: (619) 296-1070.

MASSACHUSETTS

Feb 27–Mar 3. **Postgraduate Course in Clinical Pharmacology, Drug Development, and Regulation.** Lafayette Hotel, Boston. Contact: Chuck McNelly, Center for the Study of Drug Development, Tufts University, 136 Harrison Ave, Boston, MA 02111. Tel: (617) 956-0073.

TEXAS

Feb 9–11. **Gynecologic Surgery and Urogynecology.** St Anthony Intercontinental Hotel, San Antonio. Contact: American College of Obstetricians and Gynecologists, 409 12th St, SW, Washington, DC 20024-2188. Tel: (800) 533-8811.

Feb 23–26. **Seventh Annual International Symposium on Man and His Environment in Health and Disease.** Sheraton Park Central Hotel, Dallas. Contact: Kim Rice, American Environmental Health Foundation, Inc, 8345 Walnut Hill Lane, Suite 205, Dallas, TX 75231. Tel: (214) 324-1731.

UTAH

Feb 19–24. **Diagnostic Imaging Update.** Shadow Ridge, Park City. Contact: Radiology Postgraduate Education, Registration Office, University of California, San Francisco, CA 94143-0628. Tel: (415) 476-5731.

VERMONT

Feb 7–10. **Ob/Gyn Update '89 Conference.** Stratton Mountain Inn, Stratton. *Feb 22–23.* **Clinical Update in Emergency Medicine.** Stratton Mountain Inn, Stratton. Contact: Diane M. Martin, Office of Continuing Medical Education, Albany Medical College, 47 New Scotland Ave, Albany, NY 12208. Tel: (518) 445-5828.

AROUND THE WORLD

CANADA

Feb 23–25. **Basic and Advanced Colposcopy.** Hotel Chateau Mont Sainte-Anne, Beaupre, Quebec. Contact: American College of Obstetricians and Gynecologists, 409 12th St, SW, Washington, DC 20024-2188. Tel: (800) 533-8811.

MEXICO

Feb 27–Mar 4. **Eighth Annual Comprehensive Update Body Imaging, Mammography and Neuroradiology.** 30 Cat I Credits. Acapulco Princess, Acapulco. Contact: Office of Continuing Education, SUNY Health Science Center at Syracuse, 750 E Adams St, Syracuse, NY 13210. Tel: (315) 473-4606.

VIRGIN ISLANDS

Feb 13–15. **Screening, Diagnosis, and Management of Breast Disease and Gynecologic Cancer.** *Feb 16–18.* **Current Trends in Obstetric Practice.** Frenchman's Reef, St Thomas. Contact: American College of Obstetricians and Gynecologists, 409 12th St, SW, Washington, DC 20024-2188. Tel: (800) 533-8811. □

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1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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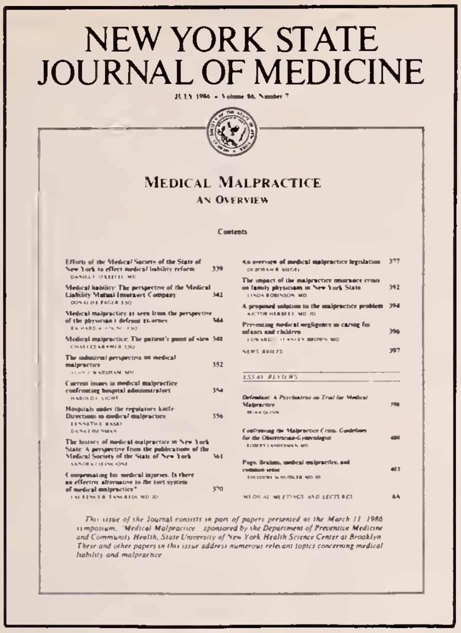
NEW YORK STATE
JOURNAL OF MEDICINE
JULY 1986 • Volume 86, Number 7

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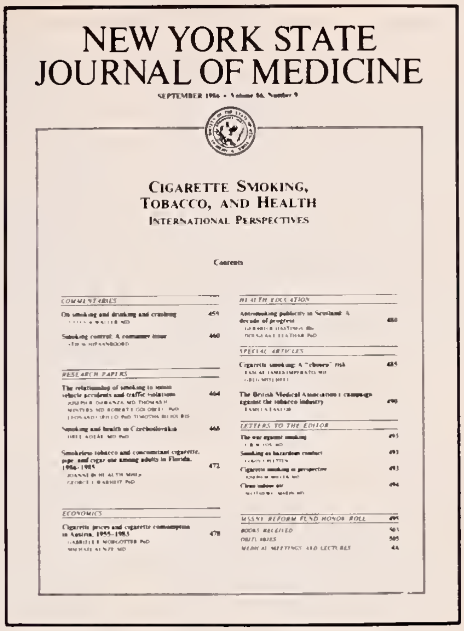
Contents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SEPTEMBER 1986 • Volume 86, Number 9

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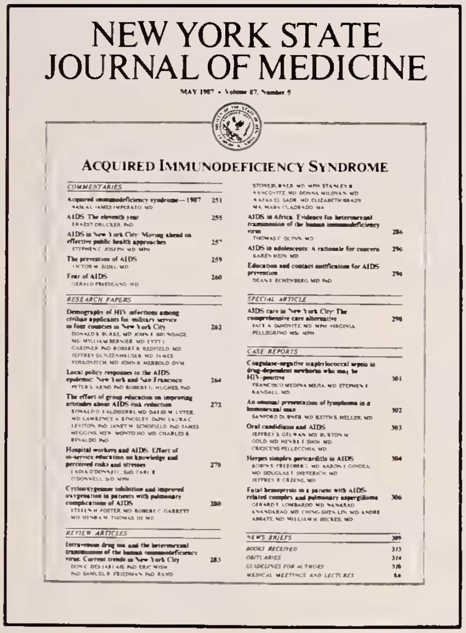
INTERNATIONAL PERSPECTIVES

Contents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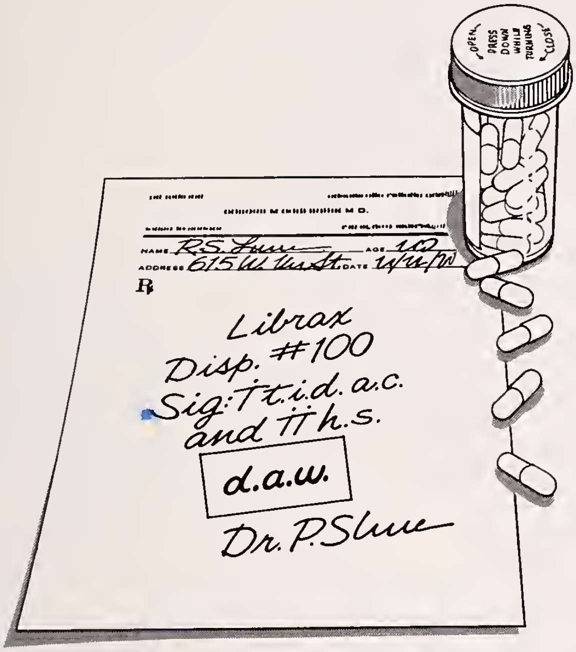

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* **Indications:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows: "Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.
Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

> ***Usage in Pregnancy:*** **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur.

Withdrawal symptoms of the barbiturate type have occurred after discontinuation of benzodiazepines (see Drug Abuse and Dependence).

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established. Inform patients to consult physician before increasing dose or abruptly discontinuing this drug.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

Drug Abuse and Dependence: Withdrawal symptoms similar to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of chlordiazepoxide; more severe seen after excessive doses over extended periods; milder after taking continuously at therapeutic levels for several months. After extended therapy, avoid abrupt discontinuation and taper dosage. Carefully supervise addiction-prone individuals because of predisposition to habituation and dependence.

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Please see summary of prescribing information on adjacent page.

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Contents

COMMENTARIES

The challenge of change 55
SAMUEL M. GELFAND, MD

Reflections on the New York do-not-resuscitate law 57
PHILLIP T. SWENDER, MD

Typhlitis 58
JAMES M. KUNKEL, MD

RESEARCH PAPERS

Hospital costs, resource characteristics, and the dynamics of death for patients with a primary diagnosis of congestive heart failure 60
ERIC MUÑOZ, MD, MBA; DONALD CHALFIN, MD; ELISA BIRNBAUM, MD; KATHERINE MULLOY; HOUSTON JOHNSON, MD; LESLIE WISE, MD

Variations in the delivery of medical care in Central New York 63
GERALD M. RICHMOND, JR, MRP, JOHN T. PRIOR, MD

REVIEW ARTICLES

The presumption of consent in New York State's do-not-resuscitate law 69
ROBERT N. SWIDLER, JD

The living will: Does it protect the rights of the terminally ill? 72
ROBERT F. RIZZO, PhD

COMMITTEE REPORT

Fetal therapy and surgery: Fetal rights versus maternal obligations 80
FRED ROSNER, MD; ALLEN J. BENNETT, MD; ERIC J. CASSELL, MD; PETER B. FARNSWORTH, MD; ALLISON B. LANDOLT, MD; LAURENCE LOEB, MD; PATRICIA J. NUMANN, MD; FERNANDO V. ONA, MD; HERMAN M. RISEMBERG, MD; PHILIP H. SECHZER, MD; PETER P. SORDILLO, MD

SPECIAL ARTICLE

Himself, Mrs Plucker, and the rites of spring 85
PASCAL JAMES IMPERATO, MD

CASE REPORTS

Typhlitis occurring in a myelodysplastic syndrome 89
JASBIR S. SRA, MD, MICHAEL R. OWENS, MD

***Campylobacter fetus* as a pulmonary pathogen in man: Case report of post-pneumonectomy empyema** 90
EDDIE L. HOOVER, MD; ANUKWARE KETOSUGBO, MD; HWEI-KANG HSU, MD; HUELDINE WEBB, MD; BRUCE TOPOROFF, MD

Enterococcal meningitis: A subacute presentation 92
DARRON R. BROWN, MD, RICHARD C. REICHMAN, MD

LETTERS TO THE EDITOR

Temporary insanity and premenstrual syndrome 94
SALLY K. SEVERINO, MD, MARGARET MOLINE, PhD

Empty lifeboat 95
JULIET GILL, MD
HERMAN R. NAYER, MD

Adult day care centers for elderly patients 96
WAYNE A. MYERS, MD

ESSAY REVIEW

Life and the law 97
JONATHAN D. MORENO, PhD

AN APPRECIATION

Our referees 100

LEADS FROM EPIDEMIOLOGY NOTES 102

BOOK REVIEWS 105

NEWS BRIEFS 108

OBITUARIES 110

GUIDELINES FOR AUTHORS 112

HOUSE OF DELEGATES

Sessions, Officers, Councilors, and Trustees; Members; Nominating and Reference Committees; Report of the House Committee on Bylaws 113

MEDICAL MEETINGS AND LECTURES 13A

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Contraindications: VASOTEC® (Enalapril Maleate, MSD) is contraindicated in patients who are hypersensitive to this product and in patients with a history of angioedema related to previous treatment with an ACE inhibitor

Warnings: *Angioedema* Angioedema of the face, extremities, lips, tongue, glottis, and/or larynx has been reported in patients treated with ACE inhibitors, including VASOTEC. In such cases, VASOTEC should be promptly discontinued and the patient carefully observed until the swelling disappears. In instances where swelling has been confined to the face and lips, the condition has generally resolved without treatment, although antihistamines have been useful in relieving symptoms Angioedema associated with laryngeal edema may be fatal. **Where there is involvement of the tongue, glottis, or larynx likely to cause airway obstruction, appropriate therapy, e.g., subcutaneous epinephrine solution 1:1000 (0.3 mL to 0.5 mL), should be promptly administered.** (See ADVERSE REACTIONS.)

Hypotension: Excessive hypotension is rare in uncomplicated hypertensive patients treated with VASOTEC alone Heart failure patients given VASOTEC commonly have some reduction in blood pressure, especially with the first dose, but discontinuation of therapy for continuing symptomatic hypotension usually is not necessary when dosing instructions are followed, caution should be observed when initiating therapy. (See DOSAGE AND ADMINISTRATION.) Patients at risk for excessive hypotension, sometimes associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death, include those with the following conditions or characteristics: heart failure, hyponatremia, high-dose diuretic therapy, recent intensive diuresis or increase in diuretic dose, renal dialysis, or severe volume and/or salt depletion of any etiology It may be advisable to eliminate the diuretic (except in heart failure patients), reduce the diuretic dose, or increase salt intake cautiously before initiating therapy with VASOTEC in patients at risk for excessive hypotension who are able to tolerate such adjustments. (See PRECAUTIONS, *Drug Interactions* and ADVERSE REACTIONS.) In patients at risk for excessive hypotension, therapy should be started under very close medical supervision and such patients should be followed closely for the first two weeks of treatment and whenever the dose of enalapril and/or diuretic is increased Similar considerations may apply to patients with ischemic heart disease or cardiovascular disease in whom an excessive fall in blood pressure could result in a myocardial infarction or cerebrovascular accident. If excessive hypotension occurs, the patient should be placed in supine position and, if necessary, receive an intravenous infusion of normal saline. A transient hypotensive response is not a contraindication to further doses of VASOTEC, which usually can be given without difficulty once the blood pressure has stabilized If symptomatic hypotension develops, a dose reduction or discontinuation of VASOTEC or concomitant diuretic may be necessary

Neutropenia/Agranulocytosis. Another ACE inhibitor, captopril, has been shown to cause agranulocytosis and bone marrow depression, rarely in uncomplicated patients but more frequently in patients with renal impairment, especially if they also have a collagen vascular disease Available data from clinical trials of enalapril are insufficient to show that enalapril does not cause agranulocytosis at similar rates. Foreign marketing experience has revealed several cases of neutropenia or agranulocytosis in which a causal relationship to enalapril cannot be excluded Periodic monitoring of white blood cell counts in patients with collagen vascular disease and renal disease should be considered

Precautions: *General: Impaired Renal Function:* As a consequence of inhibiting the renin-angiotensin-aldosterone system, changes in renal function may be anticipated in susceptible individuals. In patients with severe heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with ACE inhibitors, including VASOTEC, may be associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death

In clinical studies in hypertensive patients with unilateral or bilateral renal artery stenosis, increases in blood urea nitrogen and serum creatinine were observed in 20% of patients These increases were almost always reversible upon discontinuation of enalapril and/or diuretic therapy In such patients, renal function should be monitored during the first few weeks of therapy

Some patients with hypertension or heart failure with no apparent preexisting renal vascular disease have developed increases in blood urea and serum creatinine, usually minor and transient, especially when VASOTEC has been given concomitantly with a diuretic. This is more likely to occur in patients with preexisting renal impairment Dosage reduction and/or discontinuation of the diuretic and/or VASOTEC may be required

Evaluation of patients with hypertension or heart failure should always include assessment of renal function. (See DOSAGE AND ADMINISTRATION.)

Hyperkalemia. Elevated serum potassium (> 5.7 mEq/L) was observed in approximately 1% of hypertensive patients in clinical trials. In most cases these were isolated values which resolved despite continued therapy Hyperkalemia was a cause of discontinuation of therapy in 0.28% of hypertensive patients. In clinical trials in heart failure, hyperkalemia was observed in 3.8% of patients, but was not a cause for discontinuation

Risk factors for the development of hyperkalemia include renal insufficiency, diabetes mellitus, and the concomitant use of potassium-sparing diuretics, potassium supplements, and/or potassium-containing salt substitutes, which should be used cautiously, if at all, with VASOTEC. (See *Drug Interactions.*)

Surgery/Anesthesia: In patients undergoing major surgery or during anesthesia with agents that produce hypotension, enalapril may block angiotensin II formation secondary to compensatory renin release If hypotension occurs and is considered to be due to this mechanism, it can be corrected by volume expansion

Information for Patients:

Angioedema Angioedema, including laryngeal edema, may occur especially following the first dose of enalapril. Patients should be so advised and told to report immediately any signs or symptoms suggesting angioedema (swelling of face, extremities, eyes, lips, tongue, difficulty in swallowing or breathing) and to take no more drug until they have consulted with the prescribing physician.

Hypotension: Patients should be cautioned to report lightheadedness especially during the first few days of therapy If actual syncope occurs, the patients should be told to discontinue the drug until they have consulted with the prescribing physician.

All patients should be cautioned that excessive perspiration and dehydration may lead to an excessive fall in blood pressure because of reduction in fluid volume Other causes of volume depletion such as vomiting or diarrhea may also lead to a fall in blood pressure, patients should be advised to consult with the physician

Hyperkalemia. Patients should be told not to use salt substitutes containing potassium without consulting their physician.

Neutropenia Patients should be told to report promptly any indication of infection (e.g., sore throat, fever) which may be a sign of neutropenia.

NOTE As with many other drugs, certain advice to patients being treated with enalapril is warranted This information is intended to aid in the safe and effective use of this medication It is not a disclosure of all possible adverse or intended effects.

Drug Interactions.

Hypotension. Patients on Diuretic Therapy Patients on diuretics and especially those in whom diuretic therapy was recently instituted may occasionally experience an excessive reduction of blood pressure after initiation of therapy with enalapril The possibility of hypotensive effects with enalapril can be minimized by either discontinuing the diuretic or increasing the salt intake prior to initiation of treatment with enalapril. If it is necessary to continue the diuretic, provide close medical supervision after the initial dose for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and DOSAGE AND ADMINISTRATION.)

Agents Causing Renin Release: The antihypertensive effect of VASOTEC is augmented by antihypertensive agents that cause renin release (e.g., diuretics).

Other Cardiovascular Agents: VASOTEC has been used concomitantly with beta-adrenergic-blocking agents, methyldopa, nitrates, calcium-blocking agents, hydralazine, prazosin, and digoxin without evidence of clinically significant adverse interactions.

Agents Increasing Serum Potassium: VASOTEC attenuates potassium loss caused by thiazide-type diuretics Potassium-sparing diuretics (e.g., spironolactone, triamterene, or amiloride), potassium supplements, or potassium-containing salt substitutes may lead to significant increases in serum potassium Therefore, if concomitant use of these agents is indicated because of demonstrated hypokalemia, they should be used with caution and with frequent monitoring of serum potassium Potassium-sparing agents should generally not be used in patients with heart failure receiving VASOTEC

Lithium: A few cases of lithium toxicity have been reported in patients receiving concomitant VASOTEC and lithium and were reversible upon discontinuation of both drugs. Although a causal relationship has not been established, it is recommended that caution be exercised when lithium is used concomitantly with VASOTEC and serum lithium levels should be monitored frequently

Pregnancy—Category C. There was no fetotoxicity or teratogenicity in rats treated with up to 200 mg/kg/day of enalapril (333 times the maximum human dose). Fetotoxicity, expressed as a decrease in average fetal weight, occurred in rats given 1200 mg/kg/day of enalapril but did not occur when these animals were supplemented with saline. Enalapril was not teratogenic in rabbits However, maternal and fetal toxicity occurred in some rabbits at doses of 1 mg/kg/day or more Saline supplementation prevented the maternal and fetal toxicity seen at doses of 3 and 10 mg/kg/day, but not at 30 mg/kg/day (50 times the maximum human dose).

Radioactivity was found to cross the placenta following administration of labeled enalapril to pregnant hamsters.

There are no adequate and well-controlled studies in pregnant women. VASOTEC® (Enalapril Maleate, MSD) should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers Milk in lactating rats contains radioactivity following administration of ^{14}C enalapril maleate. It is not known whether this drug is secreted in human milk Because many drugs are secreted in human milk, caution should be exercised when VASOTEC is given to a nursing mother.

Pediatric Use Safety and effectiveness in children have not been established

Adverse Reactions: VASOTEC has been evaluated for safety in more than 10,000 patients, including over 1000 patients treated for one year or more. VASOTEC has been found to be generally well tolerated in controlled clinical trials involving 2987 patients.

Hypertension. The most frequent clinical adverse experiences in controlled trials were: headache (5.2%), dizziness (4.3%), and fatigue (3%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in controlled clinical trials were: diarrhea (1.4%), nausea (1.4%), rash (1.4%), cough (1.3%), orthostatic effects (1.2%), and asthenia (1.1%).

Heart Failure. The most frequent clinical adverse experiences in both controlled and uncontrolled trials were: dizziness (7.9%), hypotension (6.7%), orthostatic effects (2.2%), syncope (2.2%), cough (2.2%), chest pain (2.1%), and diarrhea (2.1%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in both controlled and uncontrolled clinical trials were: fatigue (1.8%), headache (1.8%), abdominal pain (1.6%), asthenia (1.6%), orthostatic hypotension (1.6%), vertigo (1.6%), angina pectoris (1.5%), nausea (1.3%), vomiting (1.3%), bronchitis (1.3%), dyspnea (1.3%), urinary tract infection (1.3%), rash (1.3%), and myocardial infarction (1.2%).

Other serious clinical adverse experiences occurring since the drug was marketed or adverse experiences occurring in 0.5% to 1% of patients with hypertension or heart failure in clinical trials in order of decreasing severity within each category:

Cardiovascular: Myocardial infarction or cerebrovascular accident, possibly secondary to excessive hypotension in high-risk patients (see WARNINGS, *Hypotension*); cardiac arrest, pulmonary embolism and infarction, rhythm disturbances, atrial fibrillation; palpitation.

Digestive: Ileus, pancreatitis, hepatitis or cholestatic jaundice, melena, anorexia, dyspepsia, constipation, glossitis.

Nervous/Psychiatric: Depression, confusion, ataxia, somnolence, insomnia, nervousness, paresthesia.

Urogenital: Renal failure, oliguria, renal dysfunction (see PRECAUTIONS and DOSAGE AND ADMINISTRATION), prostate hypertrophy

Respiratory Bronchospasm, rhinorrhea, asthma, upper respiratory infection.

Skin. Herpes zoster, pruritus, alopecia, flushing, photosensitivity

Other Muscle cramps, hyperhidrosis, impotence, blurred vision, taste alteration, tinnitus.

A symptom complex has been reported which may include fever, myalgia, and arthralgia, an elevated erythrocyte sedimentation rate may be present Rash or other dermatologic manifestations may occur These symptoms have disappeared after discontinuation of therapy.

Angioedema Angioedema has been reported in patients receiving VASOTEC (0.2%) Angioedema associated with laryngeal edema may be fatal If angioedema of the face, extremities, lips, tongue, glottis, and/or larynx occurs, treatment with VASOTEC should be discontinued and appropriate therapy instituted immediately (See WARNINGS.)

Hypotension: In the hypertensive patients, hypotension occurred in 0.9% and syncope occurred in 0.5% of patients following the initial dose or during extended therapy Hypotension or syncope was a cause for discontinuation of therapy in 0.1% of hypertensive patients In heart failure patients, hypotension occurred in 6.7% and syncope occurred in 2.2% of patients Hypotension or syncope was a cause for discontinuation of therapy in 1.9% of patients with heart failure. (See WARNINGS.)

Clinical Laboratory Test Findings.

Serum Electrolytes Hyperkalemia (see PRECAUTIONS), hyponatremia

Creatinine, Blood Urea Nitrogen: In controlled clinical trials, minor increases in blood urea nitrogen and serum creatinine, reversible upon discontinuation of therapy, were observed in about 0.2% of patients with essential hypertension treated with VASOTEC alone Increases are more likely to occur in patients receiving concomitant diuretics or in patients with renal artery stenosis (See PRECAUTIONS.) In patients with heart failure who were also receiving diuretics with or without digitalis, increases in blood urea nitrogen or serum creatinine, usually reversible upon discontinuation of VASOTEC and/or other concomitant diuretic therapy, were observed in about 11% of patients Increases in blood urea nitrogen or creatinine were a cause for discontinuation in 1.2% of patients.

Hemoglobin and Hematocrit Small decreases in hemoglobin and hematocrit (mean decreases of approximately 0.3 g % and 1.0 vol %, respectively) occur frequently in either hypertension or heart failure patients treated with VASOTEC but are rarely of clinical importance unless another cause of anemia coexists. In clinical trials, less than 0.1% of patients discontinued therapy due to anemia

Other (Causal Relationship Unknown): In marketing experience, rare cases of neutropenia, thrombocytopenia, and bone marrow depression have been reported

Liver Function Tests: Elevations of liver enzymes and/or serum bilirubin have occurred

Dosage and Administration: *Hypertension:* In patients who are currently being treated with a diuretic, symptomatic hypotension occasionally may occur following the initial dose of VASOTEC The diuretic should, if possible, be discontinued for two to three days before beginning therapy with VASOTEC to reduce the likelihood of hypotension (See WARNINGS.) If the patient's blood pressure is not controlled with VASOTEC alone, diuretic therapy may be resumed.

If the diuretic cannot be discontinued, an initial dose of 2.5 mg should be used under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, *Drug Interactions.*)

The recommended initial dose in patients not on diuretics is 5 mg once a day Dosage should be adjusted according to blood pressure response. The usual dosage range is 10 to 40 mg per day administered in a single dose or in two divided doses. In some patients treated once daily, the antihypertensive effect may diminish toward the end of the dosing interval. In such patients, an increase in dosage or twice-daily administration should be considered If blood pressure is not controlled with VASOTEC alone, a diuretic may be added.

Concomitant administration of VASOTEC with potassium supplements, potassium salt substitutes, or potassium-sparing diuretics may lead to increases of serum potassium (see PRECAUTIONS).

Dosage Adjustment in Hypertensive Patients with Renal Impairment. The usual dose of enalapril is recommended for patients with a creatinine clearance >30 mL/min (serum creatinine of up to approximately 3 mg/dL). For patients with creatinine clearance ≤30 mL/min (serum creatinine ≥3 mg/dL), the first dose is 2.5 mg once daily The dosage may be titrated upward until blood pressure is controlled or to a maximum of 40 mg daily

Heart Failure: VASOTEC is indicated as adjunctive therapy with diuretics and digitalis. The recommended starting dose is 2.5 mg once or twice daily After the initial dose of VASOTEC, the patient should be observed under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, *Drug Interactions.*) If possible, the dose of the diuretic should be reduced, which may diminish the likelihood of hypotension. The appearance of hypotension after the initial dose of VASOTEC does not preclude subsequent careful dose titration with the drug, following effective management of the hypotension. The usual therapeutic dosing range for the treatment of heart failure is 5 to 20 mg daily given in two divided doses. The maximum daily dose is 40 mg Once-daily dosing has been effective in a controlled study, but nearly all patients in this study were given 40 mg, the maximum recommended daily dose, and there has been much more experience with twice-daily dosing In addition, in a placebo-controlled study which demonstrated reduced mortality in patients with severe heart failure (NYHA Class IV), patients were treated with 2.5 to 40 mg per day of VASOTEC, almost always administered in two divided doses. (See CLINICAL PHARMACOLOGY, *Pharmacodynamics and Clinical Effects.*) Dosage may be adjusted depending upon clinical or hemodynamic response (See WARNINGS.)

Dosage Adjustment in Heart Failure Patients with Renal Impairment or Hyponatremia. In heart failure patients with hyponatremia (serum sodium <130 mEq/L) or with serum creatinine >1.6 mg/dL, therapy should be initiated at 2.5 mg daily under close medical supervision (See DOSAGE AND ADMINISTRATION, *Heart Failure*, WARNINGS, and PRECAUTIONS, *Drug Interactions.*) The dose may be increased to 2.5 mg b.i.d., then 5 mg b.i.d. and higher as needed, usually at intervals of four days or more, if at the time of dosage adjustment there is not excessive hypotension or significant deterioration of renal function. The maximum daily dose is 40 mg

For more detailed information, consult your MSD representative or see Prescribing Information Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, PA 19486.

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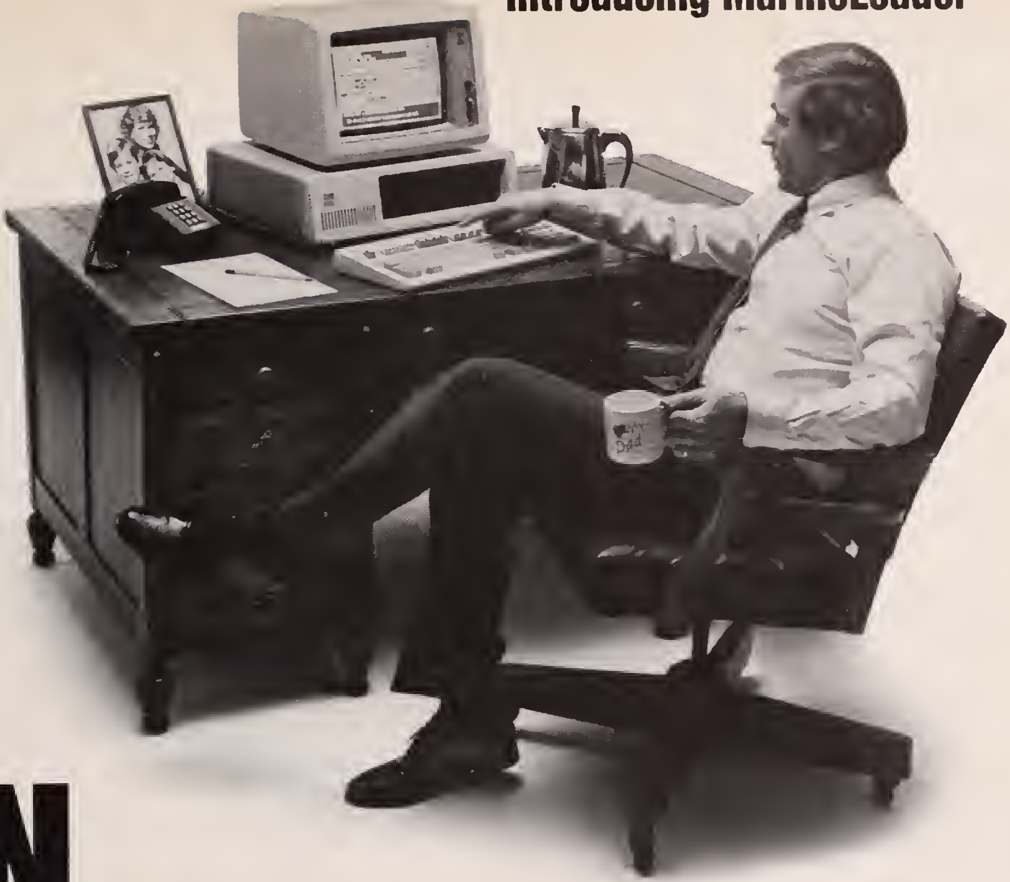

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*Clinical studies have shown that most patients remain alert when chlorzoxazone-containing products are administered at recommended doses, although drowsiness sometimes occurs.

Please see following page for brief summary of Prescribing Information.

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Parafon
Forte DSC
#40
Sig: 1 caplet
q.i.d.

The following is a brief summary only. Before prescribing, see complete prescribing information in PARAFON FORTE DSC labeling.
CONTRAINDICATIONS: PARAFON FORTE DSC chlorzoxazone is contraindicated in patients with known intolerance to the drug.
WARNINGS: The concomitant use of alcohol or other central nervous system depressants may have an additive effect. *Usage in Pregnancy.* The safe use of PARAFON FORTE DSC chlorzoxazone has not been established with respect to the possible adverse effects upon fetal development. Therefore, it should be used in women of childbearing potential only when, in the judgment of the physician, the potential benefits outweigh the possible risks.
PRECAUTIONS: PARAFON FORTE DSC chlorzoxazone should be used with caution in patients with known allergies or with a history of allergic reactions to drugs. If a sensitivity reaction occurs such as urticaria, redness, or itching of the skin, the drug should be stopped. If any symptoms suggestive of liver dysfunction are observed, the drug should be discontinued.
ADVERSE REACTIONS: After more than twenty-six years of extensive clinical use of chlorzoxazone-containing products in an estimated thirty-two million patients, it is apparent that the drug is well tolerated and seldom produces undesirable side effects. Occasional patients may develop gastrointestinal disturbances. It is possible in rare instances that chlorzoxazone may have been associated with gastrointestinal bleeding. Drowsiness, dizziness, lightheadedness, malaise, or overstimulation may be noted by an occasional patient. Rarely allergic-type skin rashes, petechiae, or ecchymoses may develop during treatment. Angioneurotic edema or anaphylactic reactions are extremely rare. There is no evidence that the drug will cause renal damage. Rarely, a patient may note discoloration of the urine resulting from a phenolic metabolite of chlorzoxazone. This finding is of no known clinical significance. Approximately thirty-six patients have been reported in whom the administration of chlorzoxazone-containing products was suspected as being the cause of liver damage. In one case, the jaundice was subsequently considered to be due to a carcinoma of the head of the pancreas rather than to the drug. In a second case, there was no jaundice but an elevated alkaline phosphatase and BSP retention. In this patient there was a malignancy with bony and liver metastases. The role of the drug was difficult to determine. A third and fourth case had cholelithiasis. Diagnosis in a fifth case was submassive hepatic necrosis possibly due to abusive use of the drug for approximately one year. The remaining cases had a clinical picture compatible with either a viral hepatitis or a drug-induced hepatitis. In all these latter cases, the drug was stopped, and with one exception, the patients recovered. It is not possible to state that the hepatitis in these patients was or was not drug-induced.
Full directions for use should be read before administering or prescribing.
For information on symptoms and treatment of overdosage, see full prescribing information.

12/16/87

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BE/BC, two adult neurologist, one pediatric neurologist for large neurological group in Southeast. Salary and benefits including malpractice and relocation. Clinical experience in EEG, EMG, evoked response, doppler ultrasound. Send resume to SEI Health Services, 7725 Little Avenue, Charlotte, North Carolina 28226 or call James Hacker at (704) 542-7100.

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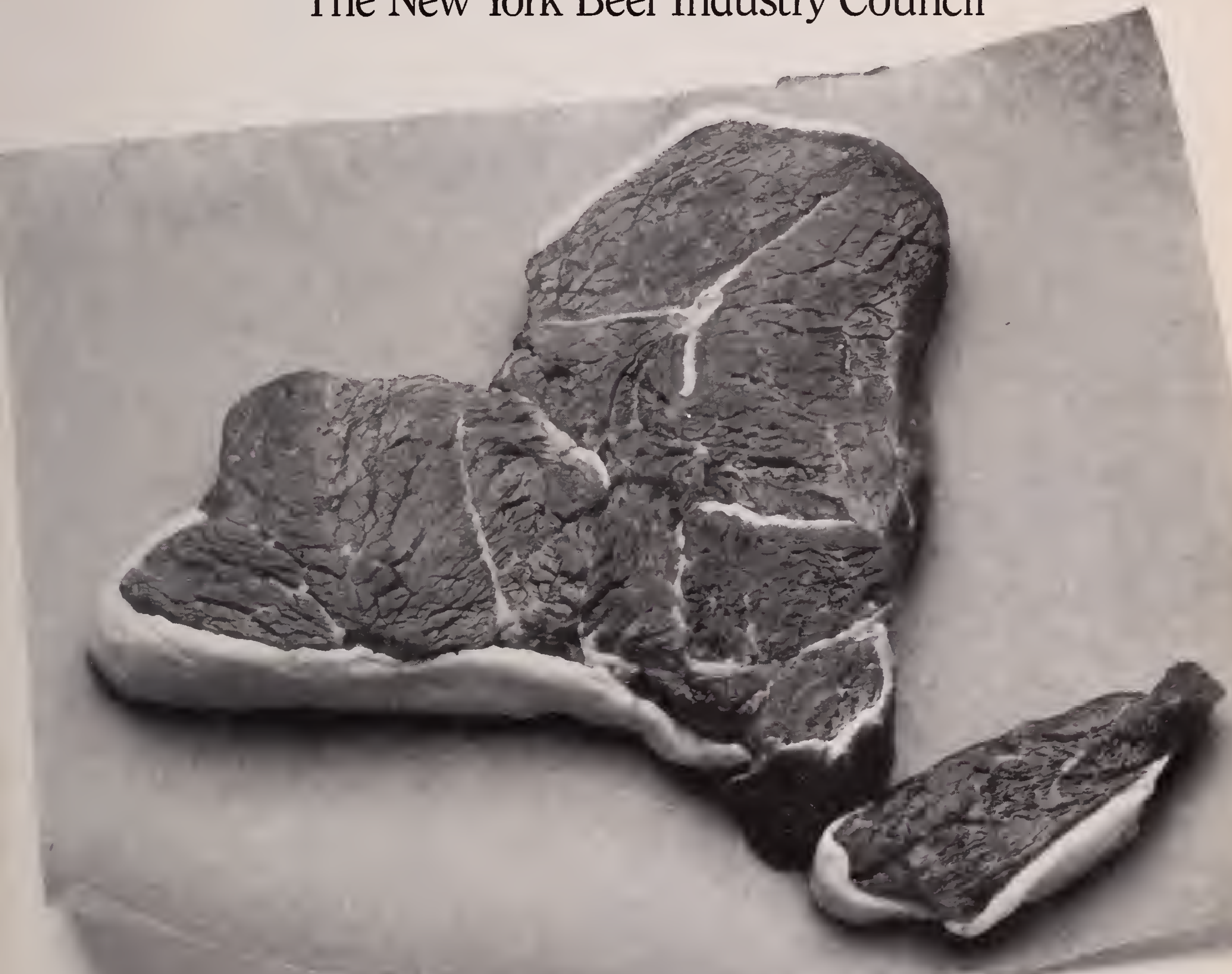

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WEDNESDAY, MARCH 15TH:
Occupational Inhalation Lung Diseases
Moderator: Morton C. Gluck, M.D.
Arthroscopy: Its Use and Potential; Its Relationship to Legal and Compensation Medicine
Moderator: Arnold Illman, M.D.

THURSDAY, MARCH 16TH:
Fraudulent and Abusive Activities in Health Care
Moderator: Robert Katz, M.D.
Mock Trial
Moderator: Peter Aldin, M.D.

***For information and application please write:* Mathew H. M. Lee, M.D., F.A.C.P., Program Chairman, American Academy of Legal & Industrial Medicine, Inc., P.O. Box 10, Island Station, Roosevelt Island, NY 10044. Phone: (212) 750-6781.**

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In irritable bowel syndrome,* intestinal discomfort will often erupt in tandem with anxiety—launching a cycle of brain/bowel conflict. Make peace with Librax. Because of possible CNS effects, caution patients about activities requiring complete mental alertness.

*Librax has been evaluated as possibly effective as adjunctive therapy in the treatment of peptic ulcer and IBS.

Specify Adjunctive

LIBRAX®

Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium bromide.

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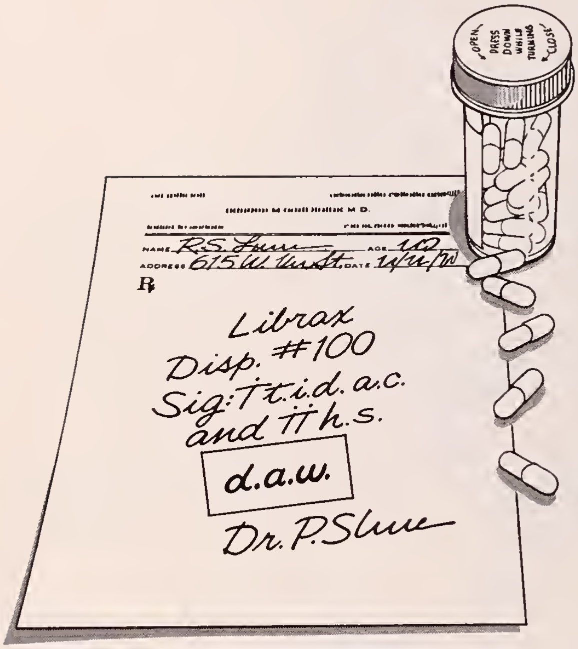
Please see summary of prescribing information on adjacent page.

For the brain/bowel conflict of IBS*



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THE PEACEMAKER.

Antianxiety
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Antispasmodic

Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium bromide.

Please consult complete prescribing information, a summary of which follows:

* **Indications:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows: "Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.
Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur.

Withdrawal symptoms of the barbiturate type have occurred after discontinuation of benzodiazepines (see Drug Abuse and Dependence).

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established. Inform patients to consult physician before increasing dose or abruptly discontinuing this drug.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

Drug Abuse and Dependence: Withdrawal symptoms similar to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of chlordiazepoxide; more severe seen after excessive doses over extended periods; milder after taking continuously at therapeutic levels for several months. After extended therapy, avoid abrupt discontinuation and taper dosage. Carefully supervise addiction-prone individuals because of predisposition to habituation and dependence.

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MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices. Information must be submitted at least three months prior to the event.*

MARCH 1989

AROUND THE STATE

ALBANY

Mar 16. **Geriatrics Conference.** *Mar 30.* **Occupational Medicine Conference.** Albany Medical College, Albany. Contact: Diane M. Martin, Office of CME, Albany Medical College, 47 New Scotland Ave, Albany, NY 12208. Tel: (518) 445-5828.

BRONX

Mar 29. **Sex Crimes.** Lehman College. Contact: Mark L. Taff, MD, President, New York Society of Forensic Sciences, 130 Gold Pl, Malverne, NY 11565. Tel: (516) 887-4691.

MANHATTAN

Mar 3–5. **Bellevue Hospital Center Psychiatry Board Review.** 23½ Cat I Credits. NYU Medical Center. *Mar 18–23.* **Anesthesiology Comprehensive Review IX.** 46 Cat I Credits. Waldorf Astoria Hotel. *Mar 27–31.* **Seminar in Advanced Rheumatology.** 28 Cat I Credits. Contact: NYU Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

Mar 9. **Symposium on Nutrition, Children and Health.** New York Academy of Medicine. Contact: Committee of Public Health of the New York Academy of Medicine, 2 E 103rd St, New York, NY 10029. Tel: (212) 876-8200, ext 252.

Mar 17–28. **Fracture Fixation: The Next Challenge.** Mt Sinai Medical Center. Contact: Elton Strauss, MD, Chief of Orthopaedic Trauma and Reconstructive Surgery, Mt Sinai Medical Center, 1 Gustave L. Levy Pl, New York, NY 10029. Tel: (212) 505-9911.

Mar 9–10. **Fourth Dr. Robert K. Match**

(*continued on p 18A*)

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think of it first

For respiratory tract infections due to susceptible strains of indicated organisms.

Summary.
Consult the package literature for prescribing information.

Indication: Lower respiratory infections, including pneumonia, caused by *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Streptococcus pyogenes* (group A β-hemolytic streptococci).
Contraindication: Known allergy to cephalosporins.
Warnings: CECLOR SHOULD BE ADMINISTERED CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS PENICILLINS AND CEPHALOSPORINS SHOW PARTIAL CROSS-ALLERGENICITY POSSIBLE REACTIONS INCLUDE ANAPHYLAXIS

Administer cautiously to allergic patients

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics It must be considered in differential diagnosis of antibiotic-associated diarrhea Colon flora is altered by broad-spectrum antibiotic treatment, possibly resulting in antibiotic-associated colitis

Precautions:
- Discontinue Ceclor in the event of allergic reactions to it.
- Prolonged use may result in overgrowth of nonsusceptible organisms
- Positive direct Coombs' tests have been reported during treatment with cephalosporins.
- Ceclor should be administered with caution in the presence of markedly impaired renal function. Although dosage adjustments in moderate to severe renal impairment are usually not required, careful clinical observation and laboratory studies should be made
- Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.
- Safety and effectiveness have not been determined in pregnancy, lactation, and infants less than one month old. Ceclor penetrates mother's milk Exercise caution in prescribing for these patients

Adverse Reactions: (percentage of patients)

Therapy-related adverse reactions are uncommon. Those reported include
- Gastrointestinal (mostly diarrhea) 2.5%
- Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment.
- Hypersensitivity reactions (including morbilliform eruptions, pruritus, urticaria, and serum-sickness-like reactions that have included erythema multiforme [rarely, Stevens-Johnson syndrome] and toxic epidermal necrolysis or the above skin manifestations accompanied by arthritis/arthralgia, and frequently, fever). 1.5%, usually subside within a few days after cessation of therapy Serum-sickness-like reactions have been reported more frequently in children than in adults and have usually occurred during or following a second course of therapy with Ceclor No serious sequelae have been reported Antihistamines and corticosteroids appear to enhance resolution of the syndrome
- Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy
- As with some penicillins and some other cephalosporins, transient hepatitis and cholestatic jaundice have been reported rarely
- Rarely, reversible hyperactivity, nervousness, insomnia, confusion, hypertonia, dizziness, and somnolence have been reported
- Other. eosinophilia, 2%; genital pruritus or vaginitis, less than 1%, and, rarely, thrombocytopenia

Abnormalities in laboratory results of uncertain etiology
- Slight elevations in hepatic enzymes
- Transient fluctuations in leukocyte count (especially in infants and children).
- Abnormal urinalysis, elevations in BUN or serum creatinine
- Positive direct Coombs' test
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COMMENTARIES

The challenge of change

During my professional career, I have seen changes in the patterns of practice, health care delivery, and financing, along with inconceivable dramatic advances in diagnostic and therapeutic modalities. The practice of medicine has changed from predominantly solo practitioners delivering most of the medical care in the patient's home to single and multispecialty groups rendering care primarily in hospitals, private offices, and clinics.

At one time the payment for health care was made directly by the recipients of the care or their families. Today, with government sponsored programs, employee fringe benefits, and private insurance, the vast majority of health care is paid for indirectly through some third party mechanism.

Early in the 20th century, a physician's armamentarium consisted of care, compassion, concern, and limited diagnostic and therapeutic tools. Now, antibiotics, chemotherapy, respirators, CAT scanning, magnetic resonance imaging, cardiac catheterization, implantable pacemakers and defibrillators, open heart surgery, organ transplants, and more are everyday elements of our practice. It is common to discuss research projects relating to artificial hearts, genetic manipulations, in vitro gestation, etc.

Yet, to date, many old and new problems remain unsolved, such as cancer, AIDS, major trauma, burns. We can approach the present and future unsolved problems with the confidence that anything imaginable can be conquered if research is not discouraged, restricted, or stymied by extra-professional forces.

The changes in medical technology and the methods of health care delivery in the past several decades have accelerated from a normal evolutionary to a rapid revolutionary pace. High costs, the responsibility for payment of these costs, the role of government in health care policy, the extent of legislative and regulatory controls, the effects of increasing professional liability costs in a highly litigious society, and mounting bioethical considerations are but a few of the issues we will have to address as these changes carry us into the 21st century.

What will the role of the physician be in directing the changes and what will his or her role be in the new health care system? How will this affect pre- and postgraduate education in forming the foundation for a physician's professional career? We must ask ourselves these critical questions in developing answers geared toward the preservation of the basic fundamentals for maintaining an accessible, affordable, quality health care system which presently is in a turmoil of confusion.

Health care costs are escalating because of new, expensive technology and improved longevity. The fastest growing age group in our population by percentage is over age 85, and costs are subject to inflationary increases.

Under the third party mechanism of payment, those responsible for paying the bills are finding they have committed themselves to purchasing more health care than they can afford or desire to pay for. As a result, in futile efforts to reduce costs, various means of cost containment have been instituted. These range from price freezes, reduced reimbursement, withheld reimbursement, and delayed payments and now are extending beyond reimbursement into dictating the extent and mode of permissible practice. The latest government consideration is a cap on health care expenditures.

This policy of over-regulation and under-reimbursement can only lead to increased rationing of resources for care, research, and educational assistance. Under the diagnosis related groups system of reimbursement for hospitals, 177 hospitals in this country closed in 1987 due to fiscal insolvency. Prestigious nursing schools are closing because of a diminished applicant pool. This country is facing a shortage of nurses approaching crisis proportions. Some medical schools have lowered their class size to maintain admission standards due to a decreased applicant pool. Others have been forced to lower their admission standards to maintain enrollment.

Government policy must change to allow health care careers to again become attractive and competitive with other career opportunities. Otherwise, in the not-too-distant future, we may face a catastrophic personnel shortage drastically affecting the ability to deliver adequate quality health care.

Educational standards were set in the early 1900s after the Flexner Report[1] and training and practice requirements are outlined by the specialty boards. The setting and evaluation of standards of quality have always been established and managed within the profession as the need developed and this should continue.

As physicians, we must preserve our freedom to exercise our clinical judgment. In the wake of economically motivated bureaucratic rules and regulations, the delivery of quality health care must not succumb to financial constraints.

We cannot allow transient elected and appointed officials to scar the health care system and destroy quality care for many generations to come, long after they are gone, by ill-conceived, even if well-intended, efforts to balance budgetary blunders on the back of the medical profession. These officials allow shortsighted vision to replace careful long range planning when seeking a quick fix to control escalating costs, too often at the expense of access and quality. Public officials need to carefully address the many factors generating high costs. They can no longer hide this shortsightedness behind a facade of cliches, such as quality control.

There is a constant concern that a professional interface with a patient could become a future adversarial relationship in a liability lawsuit. This forces the physician into a position of having to order tests and perform services defensively to establish an objective, well-documented record, which add greatly to the cost of care. We can no longer tolerate and the patient can no longer afford these extraneous manipulations of our clinical decisions and practices. The bonds of trust between the doctor and the patient must be preserved.

The professional liability problems plaguing us must be resolved. The solution requires revamping an antiquated and inadequate tort system, which at present does little more than facilitate astronomical jury awards more reflective of a legal lottery than a just system of compensation. A mechanism for adjudicating legitimate complaints of liability in a fair, equitable, and timely manner for all concerned must be instituted.

As physicians, we should become more knowledgeable about medical care costs, and limit orders, services, and procedures to those absolutely necessary, demonstrating that the profession can manage a cost effective system rather than be forced to ration resources in a cost containment system. We are the patients' advocates, always have been, and must always remain so.

Today the national annual health care expenditure exceeds $465 billion. This encourages ambitious local and national entrepreneurs to pursue huge profits by insuring low risk groups and limiting services. This leaves the tasks of caring for the elderly, the poor, the high risk groups, and the chronic and catastrophically ill to others.

The health care dollars wastefully expended by overregulation and excessive administrative costs and skimmed by fast buck profiteers must be redirected to the delivery of quality health care to those in need of it. Health care policy and practices cannot be decided in corporate boardrooms and courtrooms.

Medicine must remain a noble profession dedicated to maintaining availability, accessibility, and affordability of quality health care for all; dedicated to continued research seeking preventive and curative solutions to the many unanswered problems facing us now and in the future; and dedicated to passing our knowledge and experience on to younger physicians through adequate undergraduate and graduate teaching programs.

Preservation of these principles to which we are dedicated requires putting aside our personal vested interests and looking at the larger picture. We cannot lose sight of the forest for the trees. We must stop interspecialty, intersociety, and regional bickerings and unify our efforts toward the achievement and preservation of our dedicated goals. As President Abraham Lincoln stated, quoting the Bible, "A house divided against itself cannot stand."[2(p635)] We must look to the future and continue in the leadership role of setting the agenda for this nation's health care system. We must preserve the recognition, respect, and public trust of medicine as a profession. In his wisdom, Sir Winston Churchill[2(p921)] aptly stated before the House of Commons: "If we open a quarrel between the past and the present, we shall find that we have lost the future."

Health care planners are stripping us of our recognition as physicians. They would lump us into a broad category of health care providers rather than deal with us as an independent profession. They would further subdivide and fragment the delivery of health care into the hands of many who are granted qualifications and credentialed by legislative fiat rather than by education and training. The ultimate responsibility for patient care must remain in the hands of qualified physicians.

There are some within our system who are motivated to exploit it for personal gain at the expense of others and of the whole profession. There are a few among us practicing in a manner that falls below acceptable standards. We must more diligently encourage them to mend their ways or eliminate them from our ranks. The media exploit these anecdotal instances, portraying them as the usual physician performance tainting the entire profession. Medicine will survive as a respected and trusted profession, but only if we put our concerted efforts toward preserving it. Physicians and future physicians must become acquainted with the socioeconomic issues that are threatening their future and professional freedom. They are all involved because whatever evolves will affect all of them. It is far better to be actively and prospectively involved, having input in directing the changes and future course, than to stand by passively and retrospectively complain about undesirable changes after the fact, when it is too late.

Today, there is confusion about which direction health care delivery will take and what the role of physicians will be. We must not regard these challenges as a dark cloud of gloom but rather as the sunshine of opportunity for us to take the lead in setting the new direction and formulating the new ground rules. We must seize this opportunity to set the course. We have been blessed with tremendous advances in knowledge and technology, giving us the ability to deal with illness as never before imagined. This can supplement but can never replace care, compassion, and concern.

There are still many unanswered questions and unsolved problems. We have the best health care system in the world. We physicians have the responsibility of improving our excellent health care system. We cannot rest on our laurels. Adversity and setbacks cannot discourage us, but should only rekindle our energies to act for success in fulfilling this responsibility. We must meet these challenges and move forward. As Babe Ruth said when being congratulated on his great record, "Yesterday's home runs won't win today's ballgame."

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Reflections on the New York do-not-resuscitate law

For perhaps more years than I originally intended, I have taken care of children with chronic and often terminal conditions. I have sought to provide care that is compassionate, sensitive, and technically appropriate, striving to offer patients and their families some sense of control and determination in their lives and at the time of death. Well intended legislation and regulation have, in the past few years, made care which I believe is morally right, difficult if not impossible. While we in medicine acknowledge that reasonable regulation and oversight are necessary and even desirable, these legislative and regulatory intrusions have done irreparable harm to my relationships with families and patients, and have seriously impaired my efforts to help children with terminal illness experience life with meaning and death with dignity, unalone and unafraid.

Diagnosis related groups, prior approval requirements, Medicaid regulations, prepaid health plans, as well as a host of other circumstances and regulations, all implemented with good intent, have eroded the humane considerations involved in care of the chronically ill. Each of these deserves its own discussion. However, the dilemma has been brought into sharp focus with the implementation of the New York State do not resuscitate (DNR) law. While I applaud its attention to the necessity for documentation and its efforts to protect patients from arbitrary and unreasonable decisions, I believe its inflexibility may accomplish the opposite of its intent.

I am now required, by written or verbal consent, obtained in front of witnesses, to reflect my patients' wishes regarding resuscitation. This is not new. Most of us have been doing this for years. I must in effect ask the patient and family for an unequivocal acknowledgment of the finality of terminal illness. This is difficult, but reasonable when the impending death is imminent, but often it is not. I must now also reaffirm these wishes every three days presumably with the patient's continued consent, and I must repeat this process at every admission, even with a patient who for the last ten years has told me "when it comes time, Doc, I don't want any machines," and who during that time has had dozens of admissions. I have been told by different sources that the "every three days" provision is both a reaffirmation of the appropriateness of the DNR order which does not require renewed consent, and a means of guaranteeing the opportunity for patients and families to reconsider their decision. I'm confused. Surely I would be irresponsible if I were to renew an order with such profound implications without consulting my patient. At the same time, it would be tragic if a patient with a valid DNR order in place were denied the full application of life saving measures when he or she choked on a piece of steak at dinnertime, the event unrelated to the illness which prompted the DNR order. Patients and families should have the opportunity to reconsider and the records should reflect the continued appropriateness of DNR orders. However, circumstances should dictate the interval and nature of those deliberations, not an arbitrary legal standard.

The new law also makes passive consent a thing of the past. I can no longer say, "Mrs Jones, your son has lived as long as he can; if you have no objections, I'm going to stop now." I must add to Mrs Jones' agony, by making her an active instrument of her child's death. Some could say that such passive consent is risky and may lead to later reconsideration with the inherent risks of litigation and even prosecution. I have unwittingly taken these risks because I believed that for some patients and families it was most humane. Apparently I was wrong. It has been suggested that if active consent adds anguish, it is more than offset by release from the ambiguity of passive consent. I would suggest that uncertainty and doubt always accompany such decisions. To a sensitive and compassionate caregiver, feelings and wishes are rarely ambiguous, whether or not they are documented in some arbitrarily required manner or interval. Such compliance does at least in part provide freedom from liability. I am told that the law is needed to protect patients, physicians, and hospitals. From each other? How unfortunate.

What should I do when, in the middle of my discussion, the 25-year-old patient says, "I really don't want to hear all of this. I trust you, Doc. When it's time, do what you think is best." Or the mother who, with tears staining the consent form, says, "I know you're right, but I can't say yes to my own child's death. Please help me!"

I recently refused to use the proper forms in ordering DNR for a 19-year-old patient. My reasons included the fact that the subject has been one of discussion between us for years, and I was well aware of the young man's wishes. I also believed that the choice of agendas for our discussions at this most difficult and final time should be his, not mine, nor the hospital's, nor the State of New York's. As it happened, he chose to talk about not his death, but rather things of life, including the New York Yankees and the shapeliness of his current student nurse. Were I to comply with the letter of the law, I would have had to put aside these moments of humor and hope, to be sure that the witnessed DNR order was in place, and every three days to remind him and myself of his hopelessness. Must I extinguish those weakly flickering flames of hope by such a constant reminder of mortality for the promise of freedom from the risk of liability and prosecution? Apparently so.

I agree that charts should accurately reflect the wishes of families and patients regarding resuscitation and should demonstrate that orders not to resuscitate are periodically reconsidered, and are not written out of ignorance or bias with regard to age, ethnicity, or handicap. I do not believe that the new law will, however, guarantee any of these things, but rather will accomplish two important things. First, physicians unfamiliar or infrequently involved with DNR cases will be reluctant to write such orders; and second, it will lead to increased numbers of "sham" resuscitations where we "walk, not run," to the code and "squirt the meds into the pillow." How sad. It is to avoid these things that the law was written, but I believe that its rigidity and cumbersome nature will lead to the opposite of its intended effect.

After I wrote the DNR order on the 19-year-old man, I returned later to find his chart adorned with a blue star, apparently indicating his "no-code" status. I am told that most hospitals have something akin to a blue star. It stood out among a rack of unstarred charts of other adolescent patients. I asked that it be removed because I felt that it was a stigmatizing and powerful symbol, which would significantly alter interactions with my patient. My other patients on the unit knew exactly what a blue star meant. I was told that the star was necessary so that other hospital personnel unfamiliar with the patient would not initiate resuscitation. I then asked a number of hospital employees what a blue star meant. Most had no idea. I then asked some staff members familiar with the patient whether or not they would feel compelled to initiate resuscitation if I did not "properly" write the DNR order. Most felt that they would need to do something, but not very aggressively or seriously. What, then, is the point? Thank goodness the choice of stars wasn't yellow. Perhaps a skull and crossed bones would be more effective.

When I haltingly ask the question, "Do we need a law at all?" the usual response is a litany of anecdotal misuses of DNR orders and a perceived need to protect the physician and the hospital from liability and prosecution. I reluctantly acknowledge that there is a need. I believe, however, that the law as written is insensitive and inflexible and will in the final analysis be a disservice to dying patients.

I have not chosen to discuss the sections of the law relating to surrogate decision making and the process of arbitration when disagreements occur. These are problems in themselves.

In summary, despite having its origins in the deliberations of moral people, I do not believe the law as written will assure that DNR decisions will be made morally or with sensitivity, nor will it protect against later reconsiderations with the inherent risk of legal action. I am not convinced that it is reasonable or necessary to require documentation beyond a physician's note, a written order for non-resuscitation, and demonstration of periodic reaffirmation. While I believe that the subject should be discussed intensely in a variety of public forums, I also believe that the relationship between a dying patient and his or her caregivers is as intimate as any human relationship can be and deserves freedom from unreasonable regulatory intrusion and governmental scrutiny.

I am aware that many, including a number of my colleagues, will not agree with this point of view. I hope the dialogue will lead to a more reasonable consideration of this subject. The efforts of the New York State Task Force on Life and the Law are laudable and courageous. I must, however, raise my voice in protest against this particular legislation as written and request thoughtful reconsideration of this law.

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Typhlitis

Typhlitis, also known as neutropenic enterocolitis or the ileocecal syndrome, is a well described entity of bowel necrosis that can complicate the treatment of hematologic malignancies and can arise spontaneously in certain hematologic disorders.[1] In several large autopsy series of patients with leukemia, the incidence of this syndrome has ranged from 10% to 46%.[2-5] Cases have also been reported in association with lymphomas, multiple myeloma, aplastic anemia, and cyclic neutropenia. The report by Sra and Owens[6] in this issue of the *Journal* is notable for being the first reported case of cecal necrosis in a patient with the myelodysplastic syndrome classification of refractory anemia with excess blasts.

The pathologic process in typhlitis is generally limited to the terminal ileum, cecum, vermiform appendix, and ascending colon, hence the terminology "ileocecal" syndrome. Specific histopathologic findings consist of bowel wall necrosis—more often mucosal and submucosal than full thickness—that is associated with bacterial or fungal invasion as well as ulceration and intramural hemorrhage and edema. There is a conspicuous absence of mesenteric vascular occlusion and only occasional neoplastic infiltrations in these lesions.[3] This suggests that neither ischemia nor necrosis of neoplastic infiltrates plays a major role in the disease process. While the pathogenesis of neutropenic enterocolitis is not clear, there are a few common denominators. All the patients are neutropenic and most are thrombocytopenic. They usually have undergone recent therapy with both antineoplastic agents and broad spectrum antibiotics.

Granulocytopenia represents a deficit of an important internal host defense against infection. This can be the result of either marrow infiltration, aplasia, or myelosuppression induced by chemotherapy. Thrombocytopenia caused by similar factors predisposes to intramural hemorrhage. Antineoplastic agents can alter the normal repair process of bowel mucosa predisposing to local ulceration. Systemic antimicrobial therapy alters the bowel flora, which may contribute to bowel wall invasion by nonpathogenic or saprophytic organisms. In a recent review of the literature, the development of neutropenic enterocolitis could not be attributed to any specific regimen of chemotherapy. Nevertheless, the use of 6-mercaptopurine, vincristine, cytosine arabinoside, and steroids was common.[1] Similarly, while there was no association with a specific microorganism, *Clostridia*, *Pseudomonas*, *Escherichia coli*, and *Candida* frequently were found in the bowel wall lesions and/or blood cultures of these patients.[1] In most of the reported cases, this condition has occurred as a complication of the treatment of hematologic malignancies; however, it has been well documented that this condition can arise spontaneously in aplastic anemia[4,7,8] and cyclic neutropenia.[9-11] Cases have also been reported in patients who are neutropenic from other causes.[12,13] Neutropenia appears to be the sine qua non of this syndrome.

Unfortunately, most cases of neutropenic enterocolitis have been diagnosed post mortem. Because the symptoms are often relatively nonspecific, the diagnosis is frequently missed. Symptoms initially consist of nausea, vomiting, abdominal discomfort, and diarrhea, sometimes bloody diarrhea. A high temperature is seen, usually in the range of 102°–104°F. Most patients eventually develop abdominal pain that localizes in the right lower quadrant. When the syndrome is recognized, aggressive surgical management has proved to be very successful among those patients in which the neutropenia ultimately resolves. With persistent neutropenia, the syndrome tends to be fatal despite appropriate surgical intervention. In a review of patients with typhlitis undergoing right hemicolectomy or total abdominal colectomy, there were 16 long term survivors and six fatalities. All survivors had return of normal levels of circulating neutrophils; the nonsurvivors did not.[1] Hence, the prognosis depends on the recognition of the condition, appropriate surgical intervention, and the prompt return of normal levels of circulating neutrophils.[14]

An interesting feature of the case report by Sra and Owens[6] is the fact that the patient's absolute neutrophil count was 900 at its lowest point, which does not represent severe neutropenia. The authors suggest that this lack of neutropenia raises some questions about the proposed pathophysiology of typhlitis. An alternative explanation might be based on the qualitative rather than quantitative abnormalities of granulocytes that have been documented in the myelodysplastic syndromes. The qualitative abnormalities of these cell lines are believed to result from ineffective hematopoiesis. Morphologic abnormalities in granulocytes can be seen in the peripheral blood as well as in the bone marrow, and include cytoplasmic abnormalities as well as abnormalities in nuclear segmentation.[15]

Another consideration is that the absence of severe neutropenia raises some questions about the diagnosis of typhlitis in this case. While the patient demonstrated a clinical course very similar to many other reported cases of typhlitis, there are, of course, other entities such as appendicitis, Ogilvie syndrome, ischemic colitis, and pseudomembranous enterocolitis that can cause bowel necrosis with perforation and abscess formation in patients with hematologic malignancies.[16] Although in this case there apparently was no clinical evidence to support any of these other diagnostic possibilities, a postmortem examination would have been of interest.

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Hospital costs, resource characteristics, and the dynamics of death for patients with a primary diagnosis of congestive heart failure

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ABSTRACT. **Although substantial changes have been recommended in the diagnosis related group (DRG) prospective hospital payment system related to very expensive care for some patients, no major change in payment for these patients has been implemented by the US Congress. Both the Health Care Financing Administration and the Prospective Payment Assessment Commission continue to study issues related to DRG stratification along the lines of severity of illness, outcome (lived or died), or complications and/or comorbidities. We analyzed hospital resource consumption for 599 patients with a primary diagnosis of congestive heart failure (CHF) by outcome (ie, survivors vs mortalities). The 68 mortalities had a much greater intensity of hospital resource utilization, and a substantial financial risk under DRG pricing schemes, compared to the 531 survivors. Only mortalities within one week of admission to the hospital were profitable under DRGs. A long hospital length of stay (LOS) for mortalities was very unprofitable (mortalities with a greater than 60-day LOS generated a $42,028 loss per patient). Emergency patients who died tended to have a shorter hospital LOS and less financial risk under DRGs, compared to patients who died who were not admitted as emergencies.**

These data suggest significant inequities in the DRG prospective payment system vis-a-vis CHF mortality. Variables predictive of greater hospital resource utilization for mortalities included longer hospital lengths of stay and urgent admission. Health policy leaders should be encouraged to further stratify DRG hospital resource consumption for appropriate variance in hospital costs.

(NY State J Med 1989; 89:60–63)

Both the Health Care Financing Administration (HCFA) and the Prospective Payment Assessment Commission (ProPAC) provide ongoing analyses of the federal Medicare diagnosis related group (DRG) prospective hospital payment system.[1] The DRG prospective hospital payment system has been criticized for inaccurate resource prediction.[2] Supporters of the current DRG scheme claim that any system that groups ten million annual Medicare hospitalizations into less than 500 mutually exclusive categories is based on "average prediction" at best, and that one key component for fairness is providing adequate hospital reimbursement at the institutional level (ie, hospital specific). Although many issues regarding improvement to the DRG system have been raised, recent federal regulations have proposed no major change to the system.[3]

DRGs are stratified according to the principal diagnosis or operative procedure, the presence of a comorbidity (a condition present on admission to the hospital) or complication (a condition that occurs after admission to the hospital) and, occasionally, by outcome. Hospitals thought to treat a more severely ill or expensive patient, such as teaching hospitals and public hospitals, receive additional components to their DRG payment for "teaching costs" and caring for indigent patients. This is known as disproportionate share adjustment.[4,5]

The DRG hospital payment system, as it was conceived, contained no major division along the lines of outcome, making no distinction between survivors and mortalities. Currently, none of the 250 surgical DRGs are stratified by outcome; a few medical DRGs are stratified by outcome, however. There has been evidence to suggest that mortalities and patients with multiple complications and comorbidities generate financial risk under DRG pricing schemes.[6,7] The purpose of this study was to analyze hospital resource consumption for a large group of mortalities with congestive heart failure by the following factors: route of admission (emergency vs urgent), age, and hospital length of stay.

PATIENTS AND METHODS

The Long Island Jewish Medical Center (LIJMC) is an 805-bed teaching hospital in suburban New York City. All adult patients with congestive heart failure (CHF) admitted during a two-year period (1/1/85–12/31/86) were analyzed regarding a number of resource characteristics for survivors (those discharged alive from the hospital) and mortalities (those who died in the hospital). Patients were audited who had congestive heart failure as the primary ICD-9-CM diagnosis code as their reason for admission to the hospital.

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Several parameters were measured for each patient: age (in years); hospital length of stay (LOS) in days; mean total hospital cost; mean total hospital cost per day; profit or loss under the Medicare DRG reimbursement scheme and standard New York State DRG methodology (LIJMC is an urban teaching hospital); outliers; route of admission (emergency, admitted through the emergency room, vs non-emergency or urgent); the number of ICD-9-CM diagnosis codes; the number of ICD-9-CM procedure codes; the total number of ICD-9-CM codes (as a proxy for severity of illness); and certain clinical variables, such as the presence of intensive care unit (ICU) admission and the need for blood and/or plasma products. Although the total number of ICD-9-CM codes is not a completely accurate method to assess for severity of illness, this measure was available on our database, (as opposed to other severity measures such as the Horn Index, which we did not have available). A number of hospital characteristics were used in combination to serve as proxies for severity of illness for survivors vs mortalities; these included outcome, clinical characteristics (such as route of admission, ICU, and blood utilization) and the total number of ICD-9-CM diagnostic and procedure codes.

Hospital cost per patient (as a proxy) was computed per patient by hospital service area using patient-specific hospital charge data and converting to costs using our hospital's cost to charge ratio. Patients who died were analyzed in a number of ways: survivors vs mortalities; hospital lengths of stay (1–7 days, 8–14 days, 15–21 days, 22–30 days, 30–60 days, and more than 60 days); source of admission (ER vs non-ER); and age (18–24 years, 25–34, 35–44, 45–54, 55–64, 65–69, 70–74, 75–79, 80–85, and more than 85 years). The Student's *t*-test and the analysis of variance (ANOVA) were used to test statistical differences between groups of patients.

RESULTS

The 68 mortalities had a higher mean age compared to the 531 survivors (Table I). Mortalities had a 0.6% lesser mean DRG weight index, but they had a 50.9% greater hospital length of stay and a 27.1% greater number of diagnoses, a 162.2% greater number of procedures, and a 48.8% greater severity of illness, compared to survivors. Mean total hospital cost per patient was 105.1% greater for mortalities compared to survivors; hospital cost per day was 55.3% greater. Mortalities generated an $8,078 loss per patient under DRG payment, compared to a $398 loss per patient for survivors. ICU utilization and blood requirements were much higher for mortalities compared to survivors. The greatest number of deaths occurred during the first week of the hospital stay (Fig 1). The mean hospital cost per day for mortalities dropped as the hospital length of stay increased (Fig 2).

TABLE I. Resource Characteristics* for Survivors and Nonsurvivors with Congestive Heart Failure

Characteristic	Survivor (N = 531)	Nonsurvivor (N = 68)
Age (yr)	70.9[†]	73.8
DRG weight index (mean)	1.1656[†]	1.1582
Hospital length of stay (days)	11.0[‡]	16.6
No. diagnoses (ICD-9-CM codes/patient)	4.21[†]	5.35
No. procedures	0.82[†]	2.15
Total severity	5.04[†]	7.50
Total hospital cost	$8,002[†]	$16,411
Hospital cost per day	$780[†]	$1,211
Mean profit/loss (−)	−$398[†]	−$6,078
Emergency room admissions	76.6%[†]	70.6%
ICU admissions	22.2%	44.1%
Blood transfused	11.3%[†]	30.9%

* Mean per patient.

[†] Statistically significant difference from the nonsurvivor group ($p < 0.0001$).

[‡] Statistically significant difference from the nonsurvivor group ($p < 0.006$).

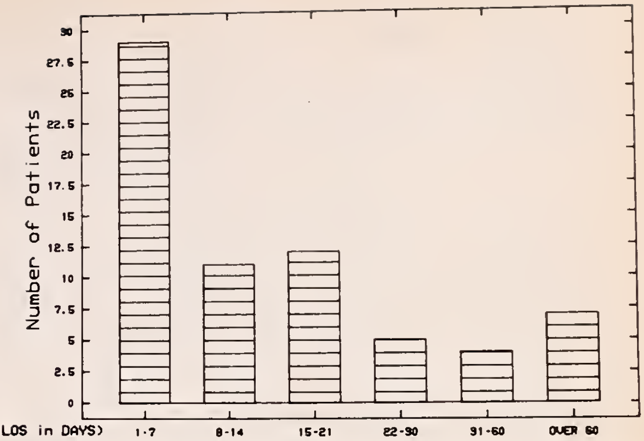

FIGURE 1. Frequency of mortality by length of stay for CHF patients.

There were some significant differences in resource characteristics for deaths based on the hospital length of stay (LOS) (Table II). Mean age and DRG weight index per patient generally did not differ significantly by hospital LOS groups; however, total hospital cost per patient, losses under DRGs, and outliers increased as hospital length of stay increased. The only profitable category for mortalities included those patients that died within seven days of admission to the hospital.

Route of admission (emergency vs urgent) was usually predictive of the rapidity of death and the level of aggregate hospital resource consumption (Table III). Patients admitted through the emergency room (ER) who died tended to have shorter hospital lengths of stay, a similar number of diagnoses, procedures, and severity of illness, and similar hospital costs per day compared to urgent patients who died. Financial risk was less for emergency mortalities, as measured by total hospital costs, losses under DRGs, and outliers. These data suggest that emergency admissions who became mortalities tended to have a shorter hospital stay, compared to urgent patients with concomitant lower aggregate hospital resource consumption. The percentages of ICU admissions and those requiring blood were similar for emergency and urgent mortalities.

Resource characteristics by age are shown in Table IV. The largest age group of mortalities was 75–79 years of age (29.7%). The longest hospital length of stay per mortality was highest for patients 70–74 years of age (26.3 days). The highest mean DRG weight index was for mortalities 55–64 years of age (1.6345). The mean total hospital cost per mortality by age group ranged from $2,761 to $28,600; the highest was for mortalities 80–85 years of age. Most age groups of mortalities generated losses under DRG payment; the greatest losses were for patients 80–85 years of age, with an $18,164 loss per patient. The percent outliers ranged from 0% to 37.5%.

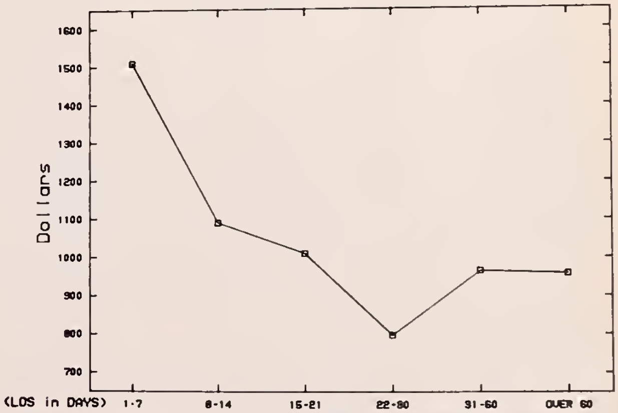

FIGURE 2. Cost per day for mortalities by length of stay for CHF patients.

TABLE II. Resource Consumption* for Nonsurvivors (Congestive Heart Failure) by Hospital Length of Stay (LOS)

Hospital Length of Stay (days)	Age (years)	Mean DRG Weight Index	Per Patient Hospital Costs	Per Patient Mean Profit/ Loss (−)	Outliers	Total ICD-9-CM	Non-ER Admissions
1–7	69.0†	1.0967‡	$3,875‡	$3,327‡	0‡	6.3‡	27.6%
8–14	81.3	1.0300	$10,875‡	−$4,099‡	0‡	6.8	27.3%
15–21	79.0	1.1334	$17,671†	−$10,374†	0‡	7.6	33.3%
22–30	73.2	1.6300	$19,822	−$13,046	4.8%‡	10.0‡	20.0%
30–60	72.8	1.7621†	$41,425†	−$28,544‡	75.0%‡	12.8‡	75.0%
60+	73.9	1.4029	$58,154‡	−$42,028‡	100.0%‡	13.5‡	14.3%
All patients	73.8	1.1582	$16,411	−$8,078	7.4%	7.5	29.4%

* Mean per patient.
† Statistically different from the mean, $p < 0.05$.
‡ Statistically different from the mean, $p < 0.01$.

TABLE III. Source of Admission (Emergency vs Urgent) and Resource Consumption* for Nonsurvivors of Congestive Heart Failure

Characteristic	Emergency Room (N = 48)	Urgent (N = 220)
Age (yr)	76.2†	68.0
Mean DRG weight index	1.1141	1.2638
Hospital length of stay (days)	11.3	29.5
No. diagnoses (ICD-9-CM codes/patient)	5.44	5.15
No. procedures	2.08	2.30
Total severity	7.52	7.45
Hospital cost/patient	$11,262	$28,768
Hospital cost/day	$1,277	$1,053
Mean profit/loss (−)	−$3,909	−$18,083
Outliers	2.1%	20.0%
ICU admissions	47.9%	35.0%
Blood transfused	31.3%	30.0%

* Mean per patient.
† Statistically significant difference from the urgent group ($p < 0.001$).

DISCUSSION

The federal DRG hospital payment system has been the most major change in hospital reimbursement in the last 20 years. Analyses of the effects of DRGs during the first five years of the payment system have demonstrated no significant adverse effects on the American health care system.[1] The American hospital industry contends, however, that the Medicare program is systematically underpaying for in-hospital care, with serious deleterious effects on American hospitals.[8] We analyzed hospital resource consumption for patients with congestive heart failure who died in the hospital. Mortalities consumed a disproportionate share of hospital resources compared to survivors; mortalities also generated tremendous financial risk under DRG prospective pricing schemes. The greatest number of mortalities occurred during the first week in the hospital; the mean hospital cost per day decreased as length of stay per patient increased for mortalities. Only mortalities who died within one week of admission to the hospital were profitable under DRGs; mortalities with long hospital lengths of stay generated a great deal of financial risk for the hospital. Financially, patients with a shorter length of stay and whose mortality occurred within less than seven days were more profitable to the hospital under the DRG system.

Our group has generated a substantial number of studies showing how hospital resource consumption differs by the way patients enter the hospital (ie, route of admission).[9-11] Route of admission (emergency vs urgent) predicted certain resource characteristics for CHF mortalities. Emergency CHF admissions who died tended to have a shorter length of stay in the hospital and less financial

TABLE IV. Resource Consumption* by Age for Nonsurvivors (Congestive Heart Failure)

Age (years)	Percent Admissions	Hospital Length of Stay (days)	Mean DRG Weight Index*	Mean Total Hospital Cost	Mean Profit/ Loss (−)	Outliers (%)	ER Admissions (%)
18–24	1.6	1.0	1.0300‡	$2,761	$4,015	0.0	100.0
25–34	—	—	—	—	—	—	—
35–44	—	—	—	—	—	—	—
45–54	4.7	7.3	1.0300	$3,555	$3,220	0.0	100.0
55–64	7.8	19.8	1.6345†	$19,175	−$9,350	0.0	80.0
65–69	10.9	9.9	1.0300	$7,658	−$883	0.0	42.9
70–74	12.5	26.3	1.0418‡	$18,356	−$9,906	37.5†	50.0
75–79	15.6	10.5	1.1634	$11,021	−$3,573	0.0	90.0
80–85	29.7	25.7	1.2444	$28,600	−$18,164	10.5	63.2
85 +	20.3	9.9	1.0445	$10,734	−$3,886	0.0	92.3†
All Patients	100.0	16.8	1.1601	$16,559	−$8,202	7.4	71.6

* Mean per patient.
† Statistically different from the mean, $p < 0.01$.
‡ Statistically different from the mean, $p < 0.05$.

risk compared to urgent mortalities. Urgent CHF mortalities generated substantially more financial risk under DRGs compared to emergency mortalities. There did not appear to be significant differences in resource consumption by the age of the CHF mortality. Our study outlined characteristics for mortalities with CHF using the framework of the DRG classification scheme. Previous study by our group and others has suggested that mortalities would generate financial risk under DRGs, and that some modification of the current DRG system along the lines of outcome for some groups of patients should be considered.[12,13] The health policy options regarding improvement of DRG equity along these lines involve stratification by severity of illness, outcome, and/or complications and comorbidities. In addition, the size, or total dollars, of the outlier pool could be increased to defray some of the financial risk associated with costly patients.

This study demonstrated several new findings regarding mortality for patients with CHF. Emergency mortalities looked more favorable compared to urgent mortalities; thus emergency admission identifies a mortality with lower hospital resource consumption. Work by our group has demonstrated significantly higher resource utilization (on average) for the emergency hospital admission.[14,15] There is growing interest in patient outcome data for hospitals. Data have suggested that hospital adjusted mortality may provide information, within constraints, as a proxy for the quality of medical care.[16] Physicians and hospitals will increasingly be asked to scrutinize their mortalities vis-a-vis costs and quality. This study demonstrated that mortalities with CHF displayed a number of characteristics regarding hospital resource consumption. Physicians must now undertake further study involving severity of illness systems with resource utilization, and must utilize such studies to advocate changes that will allow hospitals to receive adequate reimbursement and ensure a high quality of care for patients.

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Variations in the delivery of medical care in Central New York

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ABSTRACT. This paper presents an application of the Wennberg small area analysis methodology to the experience of a metropolitan county in upstate New York. It illustrates how these techniques can be used cooperatively by physicians and business to address purchaser concerns such as the impact of ambulatory surgery, the potential need for preadmission review and second opinion programs, and the contribution of high cost procedures to overall utilization and cost. The study found that use rates for most diagnoses and procedures were substantially below statewide norms, representing an annual savings of more than $30 million. It also found ambulatory surgery was effectively used as an alternative to hospitalization, and that high use rate areas involved high cost procedures targeted by second opinion and preadmission review programs.

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Many people associate the interest in geographic variations in medical care with the formation of the Professional Standards Review Organizations, which were created in 1972 to monitor the medical care utilization patterns of

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federally financed beneficiaries. It should be pointed out, however, that nearly 50 years ago, Glover[1] studied differences in tonsillectomy rates in various geographic areas in England. In the United States, Lembcke[2] documented variations in appendectomy rates during the 1950s and observed that higher appendectomy rates were associated with higher, not lower, mortality rates from appendicitis and stated that "considerably more operations of this type are done than are necessary." In the 1970s, John Wennberg[3-6] and others[7-10] began to expand the study of variations to include medical as well as surgical causes of admission and to explore causal factors influencing variations in utilization. While many studies seem to agree that practice style and the supply side of medical care are responsible for variations, there is less consensus about their meaning or the extent to which they are influenced by different factors that can affect practice style.[11-15] Such factors include defensive medicine, financial incentives, the supply of physicians, patient demand, specialty training, perceptions of medical efficacy, uncertainties in clinical judgment and decision making, and knowledge of the cost of care.

The impetus for this study came about as the result of the Central New York Health Systems Agency's examination of geographic variations in hospital utilization and the interest of the Syracuse Business Group on Health and its Physician Workgroup in the impact of physician behavior on utilization and cost. This workgroup was composed of 12 members, half of whom represented leading area industries and half of whom were physicians. The latter were chosen on the basis of specialty (including family practice) and county medical society affiliation. The initial concern of the group was that the future of the business environment in the Syracuse area could be greatly influenced by the delivery of quality care in the most cost-effective manner. A variety of issues were discussed in the initial meetings. These included the rationale for second opinions and preadmission review, liability associated with both preadmission and second opinion programs, the relationship between medical education and physician practice style, and geographic variations in practice patterns. In connection with the latter, it was recognized that potential cost savings could be achieved by focusing on diagnoses and procedures for which local use rates exceeded state norms.

The data presented to the Physician Workgroup were developed using the small area analysis technique that identifies differences in practice patterns by examining variations in inpatient utilization among communities for different "products" or types of care. The workgroup's analyses focused on Onondaga County, the county of residence and hospital use for the majority of employees whose corporations belong to the Syracuse Business Group on Health.

Onondaga County is a medium-sized metropolitan county located in the heart of Central New York. It covers 784 square miles and has a population of approximately 460,000 people. Its health care system, which also functions as the region's major referral center, includes four non-federal hospitals with 1,468 beds, a medical school, and approximately 1,200 non-federal physicians who provide direct patient care. The county is not a typical New York county since its overall use rates for hospital care are among the lowest in the state.

METHODS

The methods of small area analysis, which borrow from the principles of epidemiology, involve the calculation, comparison, and analysis of population-based use rates for specific procedures, diagnoses, and types of care rendered in hospital settings. These rates, which are similar to incidence rates used in epidemiology, provide a more accurate measure of resource consumption by populations than do institutional-based measures used in traditional analyses of hospital utilization.

The products examined represent diagnostic groups of patients who consume similar amounts of hospital services or have similar clinical conditions or treatment regimens. One hundred and seventeen "small area analysis" diagnostic and procedure groups (SAA groups) representing individual diagnosis related groups (DRGs) or clusters of DRGs were used. The groups reflect the modified DRGs used by Wennberg and his associates.[16]

As in epidemiologic studies, the use rates employed in small area analysis studies are age adjusted to permit consistent comparisons across geographic areas. The indirect method of age adjustment is generally used, since its computation factors—ie, observed and expected values—are also used as other statistical indicators in small area analysis. In this study, the age adjustment was based on New York State resident use rates for 18 five-year age cohorts.

In addition, cost estimates were developed using a modified version of the national DRG formula. Factors built into this computation included average national costs for labor and nonlabor services, DRG service intensity weights, regional wage differentials, and capital cost add-ons. Although not a true measure of cost, these estimates provide a consistent measure of relative value reflecting current reimbursement principles. The average cost per case for Onondaga County in 1985 was computed to be $3,188, or $411 per patient day.

The study used 1985 data from the New York State Statewide Planning and Research Cooperative System (SPARCS). SPARCS is responsible for collecting and maintaining data on all patients hospitalized in New York State, and has been in operation since 1980. Data elements collected by SPARCS include the following: age, sex, diagnoses, surgical procedures, DRG and major diagnostic category (MDC), payment source, admitting status, disposition, length of stay, alternate care stay, and patient residence by county and zip code.

The following terms are used in this article:

Actual usage: Observed utilization for county residents as reported in the SPARCS data base.

NYS norm: Computed or expected utilization for a geographic area based on age-specific NYS resident use rates.

Age-adjusted use: Actual or expected utilization adjusted to reflect the demographic characteristics of the state.

Cost: A computed value based on DRG case prices.

Variation from NYS norm: The percent difference between actual usage and the NYS norm.

O/E ratio: The ratio of actual or "observed" utilization to the NYS norm, ie, "expected" utilization.

The study was designed to provide information about the performance of Onondaga County relative to other counties in New York State and to statewide norms with respect to number of patients hospitalized; consumption of hospital services; cost of care; and responsiveness to benefit design issues such as ambulatory surgery, second opinions and preadmission review, and volume of high cost procedures.

The data selected were not intended to serve as an absolute measure of performance. Instead, their purpose was to provide a tool for promoting additional discussion and dialogue regarding reasons for variations and whether the variations identified op-

TABLE I. Overall Utilization

	Reported or Actual	Expected Based on NYS Norms	Difference
Discharges	46,023	58,790	−12,767
Patient days	356,670	499,128	−142,458
Estimated cost (million dollars)	146.7	177.4	−30.7

portunities for improving the utilization experience of the community through promotion of ambulatory surgery; targeting second opinion and/or preadmission review programs; reducing the volume of "high cost" admissions; changing the service delivery patterns for high volume admissions; and developing new programs to meet unmet needs.

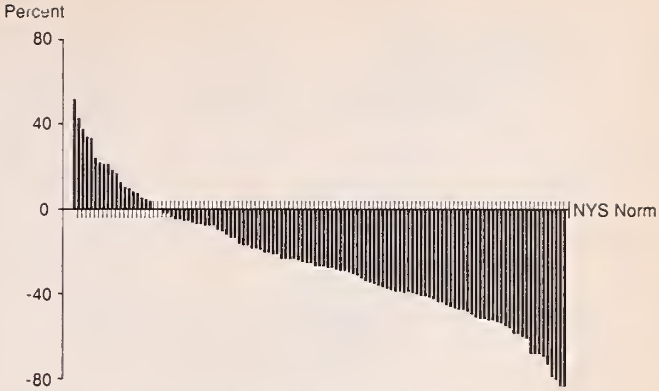

FIGURE 1. Overall utilization patterns, percent variation from state norm by SAA group, Onondaga County, 1985.

RESULTS

Overall Utilization Patterns. Table I summarizes the overall results for Onondaga County. It indicates that Onondaga County residents used substantially less hospital care than those in most other counties in New York State. Discharges were 22% below state norms, and patient days were 29% below state norms. The cost savings of this lower level of usage was estimated to be almost $31 million or $70 per capita. This lower level of utilization was evident in almost all 117 diagnostic and procedure groups (Fig 1). The county was below state norms for 80% of the groups and was more than 25% below state norms for more than half the groups. The diagnostic and procedure groups where discharge rates exceeded state norms are presented in Table II.

Potential for Expanded Ambulatory Surgery. Table III summarizes the results of the analysis of procedure groups that have a high potential for being performed on an ambulatory surgery basis. This analysis, which was done at the DRG level, indicates that Onondaga County residents made effective use of ambulatory surgery as an alternative to hospitalization. Discharges for DRGs in which there is substantial potential for ambulatory surgery were 44% below state norms. The cost savings of this lower level of usage was estimated to be approximately $2 million. As Figure 2 shows, the impact of ambulatory surgery was broad-based, as usage was substantially below state norms in almost all DRGs examined. The identification of DRGs that have a high potential for ambulatory surgery was based on an analysis conducted by the New York State Health Department of 470 specific procedures for which the stays were less than three days. The DRGs selected were those for which the proportion of procedures that met the above criteria exceeded 20%. These DRGs accounted for 4% of all discharges in Onondaga County and were estimated to cost approximately $1,300 per case.

Second Opinion/Preadmission Review. Table IV summarizes the analysis of diagnoses and procedures that are frequently targeted by second opinion and preadmission review programs (eg, major joint operations, cholecystectomy, hysterectomy, hernia operations, tonsillectomy, lens operations, etc). It indicates that although the county compares favorably with state norms, there may be potential for achieving additional cost savings through such programs. The total number of discharges for diagnostic and procedure groups included in this analysis was 4% below state norms. This difference was substantially less than that found in the general analysis of all causes of hospitalization (22% below state norms).

In almost half the diagnostic and procedure groups examined, dis-

TABLE II. Diagnostic and Procedure Groups That Exceed State Norms

Small Area Analysis Group	Actual Discharges	Expected Discharges	O/E Ratio: Actual to Expected	Age Adjusted Use Rate per 10,000 Population	Percent Variation from NYS Norm	Chi-Square Significance Level (*p*)
Major joint operations	458	303	1.51	10.80	51.3	<0.001
Nervous system operations	680	479	1.42	15.42	42.1	<0.001
Back and neck operations	379	279	1.37	8.58	37.3	<0.001
Knee operations	550	411	1.34	11.78	33.7	<0.001
Acute adjustment reaction	88	66	1.33	1.95	32.8	<0.01
Female reproductive system operations	140	113	1.24	3.22	23.5	<0.05
Hysterectomy	810	666	1.22	18.92	21.5	<0.001
Gall bladder disease with cholecystectomy	730	605	1.21	16.65	20.7	<0.001
Respiratory system operations	265	220	1.21	6.07	20.6	<0.01
Other musculoskeletal system operations	1218	1035	1.18	26.48	17.6	<0.001
Other maternal diagnoses	1548	1327	1.17	31.51	16.6	<0.001
Other female laparoscopic operations	259	232	1.12	5.48	11.8	<0.1
Hand operations except ganglion	246	223	1.10	5.34	10.3	—
Inguinal and femoral hernia operations	750	687	1.09	17.12	9.1	<0.05
Chest pain	268	249	1.08	6.22	7.7	—
Vaginal delivery	5415	5066	1.07	110.38	6.9	<0.001
Major cardiovascular operations	547	520	1.05	12.68	5.1	—
Cesarean section	1951	1951	1.05	34.43	4.7	<0.1
Major small and large bowel operations	434	417	1.04	10.12	4.0	—
Gastrointestinal hemorrhage	381	380	1.00	8.86	0.2	—
Soft tissue operations	132	132	1.00	2.90	0.1	—

TABLE III. Ambulatory Surgery Potential

	Reported or Actual	Expected Based on NYS Norms	Difference
Discharges	1,861	3,300	−1,439
Estimated Cost (million dollars)	2.5	4.5	−2.0

TABLE IV. Second Opinion/Preadmission Review Potential

	Reported or Actual	Expected Based on NYS Norms	Difference
Discharges	9,704	10,159	−455
Estimated Cost (million dollars)	34.1	31.5	2.6

TABLE V. High Cost Discharges

	Reported or Actual	Expected Based on NYS Norms	Difference
Discharges	17,168	19,298	−2,130
Estimated Cost (million dollars)	87.0	94.3	−7.6

TABLE VI. Characteristics of High Cost Diagnostic and Procedure Groups That Exceed State Norms

Small Area Analysis Group	Estimated Cost Per Case	Targeted by Second Opinion/ Preadmission Review Programs
Major joint operations	$7,501	X
Nervous system operations	$5,580	X
Back and neck operations	$5,141	X
Knee operations	$3,306	X
Hysterectomy	$3,398	X
Gall bladder disease with cholecystectomy	$4,927	X
Respiratory system operations	$7,701	—
Other musculoskeletal system operations	$3,765	—
Major cardiovascular operations	$13,022	—
Major small and large bowel operations	$8,108	X

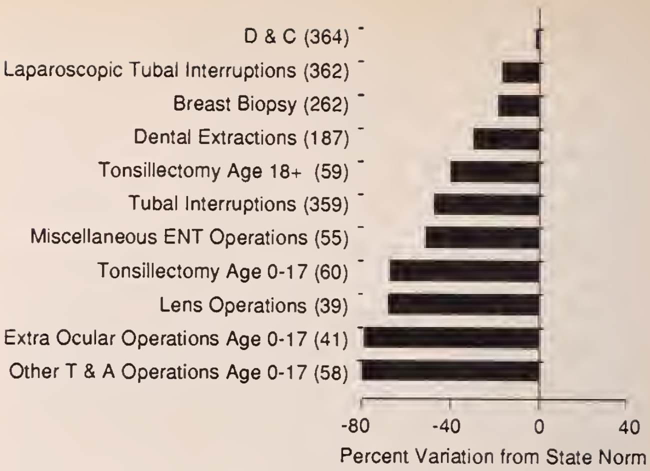

FIGURE 2. Ambulatory surgery procedures, percent variation from state norm by DRG, Onondaga County, 1985.

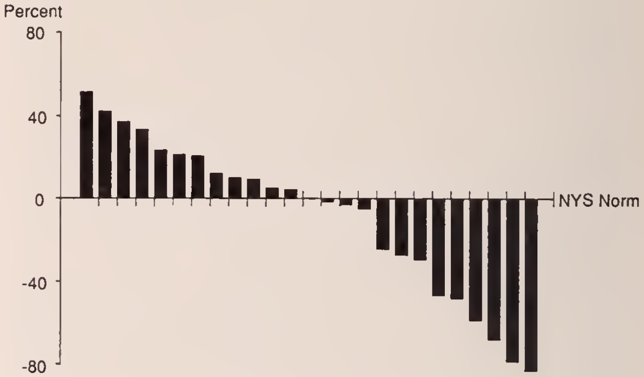

FIGURE 3. Second opinion/preadmission review, percent variation from state norm by SAA group, Onondaga County, 1985.

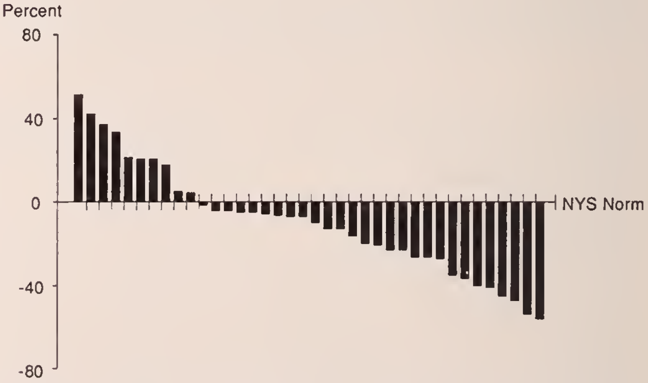

FIGURE 4. High cost discharges, percent variation from state norm by SAA group, Onondaga County, 1985.

charges exceeded state norms, in some cases by as much as 50% (Fig 3). If the level of usage for these groups had been equal to state norms, the cost savings would have been $5 million. The identification of diagnostic and procedure groups for this analysis was based on a review of lists developed by insurance companies, professional review organizations, and other payors. These groups accounted for 21% of all discharges in Onondaga County and were estimated to cost approximately $3,500 per case.

High Cost Procedures. Table V summarizes the analysis of high cost diagnoses and procedures. It also indicates that although the county compares favorably with state norms, there may be potential for achieving additional cost savings by reducing the level of high cost discharges. The total number of discharges for high cost diagnostic and procedure groups was 11% below state norms. Although this difference is less than that found for all causes of hospitalization (22% below state norms), its cost impact is significant, since these discharges were estimated to be responsible for 59% of total cost but only 37% of total discharges. The potential for savings is most likely to be found in ten high cost diagnostic and procedure groups in which usage exceeded state norms (Fig 4), in some cases by more than 30%. If the level of usage for these ten groups had been equal to state norms, the cost savings would have been $5.9 million.

These savings, however, would not have been additive to those previously cited, because there is substantial overlap between high cost diagnoses and procedures and those targeted for second opinion or preadmission review. Seven of the ten high cost diagnostic and procedure groups that exceeded state norms were also included in the second opinion/preadmission review analysis (Table VI).

The identification of high cost procedures was based on DRG service intensity weights. The diagnostic and procedure groups selected were those with service intensity weights greater than one, a level which in Onondaga County was estimated to represent approximately $3,300 per case. They accounted for 37% of all discharges in the county. Their cost per case was estimated to average $5,100 but range as high as $13,000.

DISCUSSION

This paper presents an illustration of how the small area analysis technique can be applied to address purchaser concerns such as the impact of ambulatory surgery on hospital utilization, the potential need for preadmission review and second opinion programs, and the contribution of high cost discharges to overall utilization and cost. It should be noted, however, that the county examined, Onondaga, is not a typical New York county since its overall use rates for hospital care are among the lowest in the state. The analysis confirmed this relative position and illustrated the impact that longstanding efforts in the county to promote ambulatory surgery as an alternative to hospitalization have had on hospital care. It also provided evidence for why certain procedures continue to be targeted by preadmission review and second opinion programs. The vast majority of the diagnoses and procedure groups that exceeded state norms in this county were those targeted by such programs.

As a follow-up to its preliminary analysis of practice patterns, the Physician Workgroup has begun to focus on individual specialty areas in which utilization rates exceed state norms. Its approach has been to examine the specialty area in greater detail by profiling overall utilization patterns (Fig 5) and comparing the study area's experience for specific diagnostic and procedure groups to that of similar geographic areas across the state (Fig 6). It has also begun to enter into a dialogue with specialty leaders in the local medical community. It is hoped that the educational value of these exchanges, combined with efforts to fine-tune benefit packages, will effect more uniform professional decision making, cost savings, and clinical efficacy. The initial focus of these follow-up efforts has been on orthopedics and obstetrics/gynecology.

In the final analysis, it is this exchange of information and the potential for reexamining behavior that is the ultimate benefit and goal of the small area analysis methodology. Physicians will need to become increasingly aware of this method for examining behavior during the next two to five years as purchasers, planners, hospital administrators, medical staff committees, utilization review organizations, and others begin to apply its techniques to document variations for different dimensions of utilization and cost (eg, discharge rates, length of stay, hospital charges, physician fees, etc) and to develop a better understanding of physician practice patterns across the state.

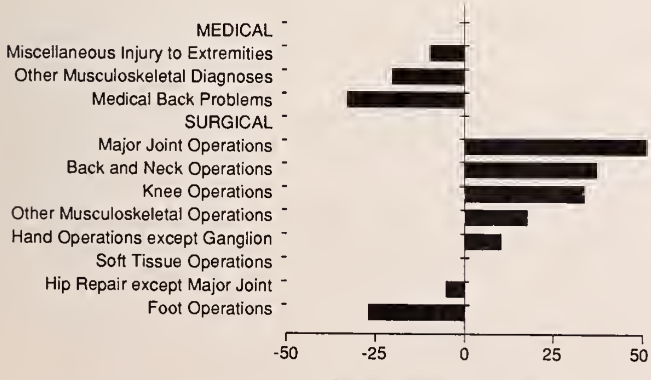

FIGURE 5. Orthopedic utilization, percent variation from state norm by SAA group, Onondaga County, 1985.

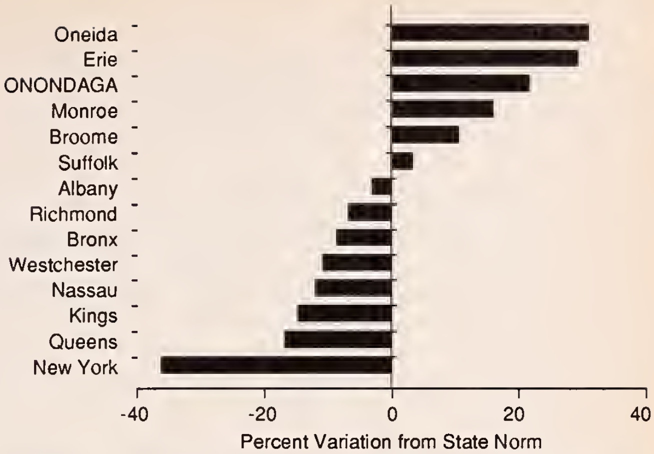

FIGURE 6. Hysterectomy and female reproductive system, reconstructive operations, percent variation from state norm by county, Onondaga versus other metropolitan referral centers, 1985.

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Special Issue: Women Treating Women

The New York State Journal of Medicine, in conjunction with a special committee of the Nassau Chapter of the American Medical Women's Association, is planning a special thematic issue of the *Journal* for the Fall of 1989. This issue will deal with the current status of women physicians, as well as other women health professionals and their female patients, and will be entitled "Women Treating Women." The *Journal* welcomes submissions from readers who may be doing current research in this area, as well as review articles, clinical material, and "think" pieces. We would also welcome letters to the editor, classified advertising of particular interest to women in medicine, and other items of interest. The deadline for manuscripts is May 15, 1989. Correspondence should be addressed to Naomi Bluestone, MD, Consulting Editor, *New York State Journal of Medicine*, 420 Lakeville Rd, Lake Success, NY 11042.

The presumption of consent in New York State's do-not-resuscitate law

ROBERT N. SWIDLER, JD

April 1, 1988 marked the implementation of an innovative law governing the issuance of do-not-resuscitate (DNR) orders in New York State hospitals, nursing homes, and mental health facilities.[1] The law strives to clarify the rights and responsibilities of patients, family members, and health care professionals in decisions about cardiopulmonary resuscitation (CPR). It responds to dual, mirror-image problems that had persisted in health care facilities largely as a result of legal uncertainties: the covert issuance of DNR orders, without the consent of the patient or patient's family, and the failure to issue DNR orders when they are medically appropriate and sought by the patient or patient's family. The law is based on a 1986 proposal by the New York State Task Force on Life and the Law, although it differs from the task force proposal in certain respects.[2]

The DNR law establishes comprehensive standards and procedures for issuing DNR orders, but its provisions can be distilled into two essential principles. First, for patients who have decisional capacity, a DNR order is lawful if issued with the consent of the patient. Second, for patients who lack decisional capacity, a DNR order is lawful if the patient's condition is sufficiently poor to warrant a DNR order, and if the order is issued with the consent of an appropriate surrogate decision maker. Considerable statutory attention is given to the procedures for implementing these principles (eg, determining a patient's lack of capacity), and to addressing the major problems that may arise (eg, disagreements among family members, or between a family member and the physician).

In these first few months of experience with the DNR law, an important problem has emerged concerning the statutory presumption of a patient's consent to CPR.[3] One widely held reading of the presumption is that it requires CPR to be performed on every patient for whom a DNR order has not been written, even if it would clearly be futile or detrimental to the patient.[4] Some medical personnel have expressed grave concern about this inflexible rule; some even contend that it compels them to violate their oath to "do no harm."

The view that the presumption of consent constitutes an absolute duty to resuscitate is at odds with what the statute says, what the drafters and legislature intended, and what good medical practice requires. The Department of Health has already taken steps to correct this misinterpretation by a health facilities memorandum.[5] This article suggests the proper, narrower reading of the presumption of consent: the presumption merely signifies that the non-DNR patient is deemed to *permit* resuscitation. (For the purpose of this article, "non-DNR patient" means a patient for whom a DNR order has not been entered pursuant to the DNR law). The presumption does not, however, define the duty to provide resuscitation to that patient; such duty continues to be based on the applicable standard of care that governs the conduct of health care professionals and facilities.

LANGUAGE AND INTENT OF THE CLAUSE

Section 2962.1 of the DNR law states that:

> Every person admitted to a hospital shall be presumed to consent to the administration of cardiopulmonary resuscitation in the event of cardiac or respiratory arrest, unless there is a consent to the issuance of an order not to resuscitate as provided in this article.[3,6]

The clause plainly does not address the duty of health care professionals to perform CPR, nor does it mandate resuscitation of every non-DNR patient. Instead, it speaks only about the patient's *consent*. The clause simply restates a well-established principle of law: When emergency medical treatment is indicated and the patient is unable to consent expressly (and had not previously objected) to such treatment, the patient's consent is presumed—ie, the patient is deemed to permit the treatment.[7-9] Thus, there can be no liability, based on a claim of lack of consent, for providing CPR to a non-DNR patient who arrests.

The presumption of consent clause was part of the original legislative proposal by the Task Force on Life and the Law. In its report, the task force explicitly described the legal principle upon which the presumption is based:

> Where emergency treatment is necessary, the patient's consent to treatment is presumed. Hence, unless proper consent to a DNR order has been obtained, the failure to provide CPR *when it is medically indicated* may subject a physician to civil liability, professional sanctions and, if sufficiently egregious, criminal penalties.[2(p13),10]

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Subsequently, in its discussion of the proposed law, the report makes clear that the presumption of consent clause was designed to track, not modify, existing law:

> In the absence of a decision by the patient to refuse CPR, the proposed legislation explicitly affirms the presumption *under existing law* that all patients, in the event of cardiac or respiratory arrest, consent to CPR [emphasis added].[2(p22)]

Legislators were made aware of this limited function of the presumption of consent. A copy of the task force report was provided to every legislator. Moreover, an informational memorandum distributed during the Senate debate on the bill contained the following question and answer:

> **Question:** Does this bill create a new obligation to perform CPR that did not previously exist? Will nursing homes have to start providing CPR?
>
> This bill does not expand or contract the health care professional's duty to provide CPR in the absence of a DNR order.
>
> The bill creates a presumption that a patient consents to resuscitation in the absence of a DNR order. That presumption just means that the physician is not required to obtain consent, and can provide CPR without fear of liability. It does not create any new duty to provide CPR. The question of whether a hospital or nursing home has an obligation to provide CPR depends on the applicable standard of care—an evolving common law standard.[11]

ANALYSIS OF THE PRESUMPTION OF CONSENT

The clause presumes that a patient who does not have a DNR order consents to CPR—that is, the patient would *permit* it. That presumption does not create any new obligation to provide CPR. Consent to a treatment cannot alone establish a duty to provide the treatment. For instance, a patient might expressly or implicitly consent to the removal of any cancerous tumor found during exploratory surgery; that consent does not define the scope of the physician's duty to remove an inoperable tumor. Or a patient might consent to an organ transplant; that consent does not bind the physician to go forward with the procedure if he or she determines that the donated organ would not be viable.

Instead, the obligation to perform CPR has been and continues to be defined by the same sources that define the obligation to perform other medical procedures: the evolving common law standard of care, professional guidelines, and, when applicable, health department regulations and rulings. Currently, the only regulatory standards on providing CPR are set forth in a health department memorandum relating to emergency medical technicians—ambulance personnel.[12]

The standard of care in New York may be stated in general terms: A physician is obligated to possess and exercise that degree of knowledge, care, and skill expected of the average physician in the locality and of the same class of physicians to which he or she belongs, and to use his or her best judgment and reasonable care in applying that knowledge, care, and skill.[13,14] A health care facility, acting through its physicians, nurses, and other professionals, is subject to a similar standard of care.[15] The issue in a malpractice action is whether what the doctor or facility did or failed to do in treating the patient constituted a departure from this standard of care.[16]

The precise application of this standard to resuscitation—or for what matter, to any medical treatment—is necessarily inexact and fact-based. The outer boundaries of the duty, though, are clear. On one hand, a professional who, though able, fails to resuscitate a non-DNR patient who has a reasonable chance to recover from the arrest could be liable for malpractice—not because of any new duty imposed by the DNR law, but because the professional would always have been liable under those circumstances. He or she failed to do what a physician of ordinary skill and training would have done under the circumstances, and that omission caused the patient's injury.[17]

Conversely, if a physician makes an on-the-scene decision to withhold CPR from an arresting patient who does not have a DNR order because of a well-grounded judgment that CPR at that juncture would be medically futile, the physician will not be liable for malpractice or subject to other sanction: He or she did not omit a measure that an ordinary doctor would provide. (The existence of a hospital policy mandating CPR for all non-DNR patients might alter this analysis by establishing a hospital-wide standard of care.) Nor did he or she violate any requirement of the DNR statute—the presumption of patients' consent does not define the scope of the physician's duty to non-DNR patients.

It could be argued that because the law permits a DNR order to be entered on the ground of "futility" under two specified circumstances—where there is surrogate consent;[18] and where no surrogate is available and two physicians authorize the order[19]—that it implicitly mandates CPR in all other instances of arrest, even if CPR would be futile. That inference should be rejected. The two statutory references to "futility" relate to the grounds and procedures for entering a DNR order—an instruction, in advance of a patient's arrest, not to attempt CPR. They do not relate to or logically govern the separate issue: how physicians must respond to a non-DNR patient's arrest.

While the DNR law does not define the duty of health care professionals to respond to a non-DNR patient's arrest, it has cast a spotlight on the response of health care facilities and may cause them to reassess their CPR policies for such patients. In that sense the law may affect the standard of care. Moreover, as discussed below, the law enhances the obligation of physicians to *talk* to those patients who ought to have a DNR order, or to their family members, before an emergency arises. But disputes about the duty to provide CPR to the non-DNR patient will ultimately turn, not on any provision in the DNR law, but on how a knowledgeable, careful, and skillful physician would respond under the circumstances.

THE NEW YORK STATE DEPARTMENT OF HEALTH MEMORANDUM

In a Health Facilities Memorandum issued on March 18, 1988, the New York State Department of Health expressly endorsed the limited reading of the presumption of consent. The memorandum states as follows:

> *No New Duty to Resuscitate*
>
> Article 29-B imposes no new duty on facilities or staff to resusci-

> tate patients in arrest where such a duty did not exist before the law. Patients are presumed to consent to resuscitation absent a contrary order, but facilities without the capability of providing CPR are not required to develop such a capability. Also, the authority of a physician to make a medical judgment that resuscitation measures would be futile in a particular case has not changed, but decisions made on this basis cannot become so common as to constitute a policy of unwritten DNR orders.[5]

The memorandum reflects the Health Department's view that the presumption of consent does not constitute an absolute duty to resuscitate. However, the final sentence reveals the department's legitimate concern with an implication of its interpretation: Physicians who feel burdened by the requirements of the DNR law may be tempted to avoid writing appropriate DNR orders, and opt instead to await the patient's arrest or impending arrest, at which time the physician can, if available, direct the withholding of resuscitative efforts on grounds of futility. This practice would obviously thwart a key objective of the law—to place the patient, or the patient's family, at the center of DNR decisions, and to plan in advance of a crisis. The Health Facilities Memorandum thus correctly warns physicians against practices or patterns along these lines.

The DNR law, then—and more explicitly the Health Facilities Memorandum—identifies an emerging element in the physician's duty toward the severely ill patient: In situations where a physician concludes that a patient is not an appropriate candidate for CPR, and where there is an opportunity to enter a DNR order pursuant to the statute (ie, to obtain the patient's or surrogate's consent), the physician should follow the procedures for entering a DNR order, and not await an arrest and invoke futility or other grounds for nonresuscitation.

THE "EMERGENCY EXCEPTION" ANALOGY

The physician's discretion to withhold CPR from a non-DNR patient who arrests may be characterized as an "emergency exception" to the DNR law, similar to the emergency exception to the general requirement of obtaining a patient's consent. In general, a physician obtains a patient or surrogate decision-maker's consent prior to an invasive treatment—surgery, for instance. In a true emergency, when the patient's consent cannot reasonably be obtained, the physician may treat without that consent (unless the patient's objection is known). However, it constitutes poor medical practice to ignore a patient's foreseeable need for surgery, and the opportunity to obtain the patient's decision about that procedure, in order to provide treatment on the basis of the patient's implied consent when an emergency occurs.

So, too, with DNR orders. In general, a physician should obtain a patient or surrogate's decision about a DNR order prior to an actual crisis. In some cases, the physician may be confronted with an arrest or an impending arrest before he or she has contemplated the resuscitation issue or completed the DNR procedure. In some of these emergency situations, the physician's best judgment will be to withhold, or direct the withholding of, resuscitative efforts. The DNR law does not impede that exercise of judgment. But here, too, public policy must guard against those who exhibit a pattern of waiting for emergencies to occur, then dispense with the consent requirement and withhold resuscitation.

The analogy may be carried a step further. A physician may not invoke the emergency exception to the general informed consent requirement for a patient who, prior to the emergency, had objected to treatment.[20] For similar reasons, a physician should not invoke the "emergency exception" to the DNR consent requirement if the patient previously objected to the entry of a DNR order, or if a surrogate objected on the patient's behalf. The notion that a patient or surrogate decision-maker can object to a DNR order—in effect, insist upon CPR—has recently been criticized by some commentators.[21,22] However, the DNR law embodies the premise that most patients and surrogates, when provided with a realistic assessment of the potential benefits and burdens of CPR under the circumstances, and with sound advice, will make rational decisions. This view has been advanced by other commentators.[23-25]

IMPLICATIONS

The proper, more limited reading of the presumption of consent signifies that physicians retain the ability to exercise good medical judgment in their response to the non-DNR patient's arrest. This has important implications for resuscitative decisions in the emergency room and the delivery room, where physicians often do not, as a practical matter, have the opportunity to comply with all the procedures of the DNR law. As explained in this article, when full compliance with the DNR statute is not reasonably practicable, emergency room and delivery room physicians can and should follow accepted medical practice in making resuscitation decisions, notwithstanding the statutory presumption that the patient consents to CPR. The lawfulness of withholding CPR under such circumstances will depend, as it always has, on whether it conforms to the applicable standard of care.

Similarly, in other hospital locations, when a code is called on a non-DNR patient, the DNR law does not preclude a responding physician from exercising judgment about whether to start resuscitation, any more than it precludes him or her from deciding what resuscitative measures to utilize and when to stop. (The definition of "cardiopulmonary resuscitation" in the Health Department regulations specifies several resuscitative measures, including electrical defibrillation and open chest cardiac massage.[26] The function of that definition, though, is not to specify the measures that must be performed when resuscitating a patient, but rather to identify the measures that *must not* be performed on a patient who has a DNR order.[2(p20),27]

Moreover, if a patient's arrest appears imminent and, in the physician's supportable judgment, resuscitation would be futile, the physician may, as an interim measure until an appropriate DNR order may be entered, direct the staff to withhold resuscitative efforts. Again, the legitimacy of this interim directive will depend on whether it conforms to the applicable standard of care. At least in cases where a reasonable physician would deem resuscitation futile, where the physician conformed to the DNR law to the extent possible under the circumstances (eg, by attempting to complete the determination of incapacity or to locate a surrogate decision-maker), and where there has been no express decision *against* a DNR order by the patient or surrogate, that standard of care will be met. (Again, the hospital policy will be an additional important factor.)

FUNCTION OF THE DNR LAW

Why, then, the DNR law? What role does it play? Quite simply, the law governs the issuance of DNR orders—orders that embody an advance decision not to resuscitate a patient in the event of an arrest. The law reflects an ethical consensus that such orders should be documented, and should be based on the consent of the patient or an appropriate surrogate decision-maker. DNR orders written on this basis are expressly authorized by law, and the health care professionals who write or honor them are afforded statutory immunity from criminal and civil liability and administrative sanctions. Professionals acting within this "safe harbor" need not argue the contours of the applicable standard of care. Conversely, the entry of secret no-codes, or the deliberate exclusion of the patient or surrogate from the DNR decision, are now manifestly against public policy.

As stated previously, a key objective of the DNR law was to place the patient or the patient's family at the center of DNR decision-making to the extent possible. To achieve this objective, enormous efforts are needed to induce physicians to act in advance of crisis—to identify patients for whom a DNR order appears to be appropriate, and to talk to those patients or their surrogate decision-makers in order to ascertain and implement their decision.

But crises will occur. It is essential for health care professionals, administrators, and counsel to understand that, in such crises, if there is no reasonable opportunity to complete a proper DNR order, the physician's hands are not tied by the DNR law: The law does not increase, decrease, or alter existing standards about when, in the absence of a DNR order, CPR must be provided.

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The living will: Does it protect the rights of the terminally ill?

ROBERT F. RIZZO, PHD

Thirty-eight states and the District of Columbia have passed legislation that recognizes the rights of self-determination of the legally competent person to execute a directive or declaration indicating the kind of treatment desired in a terminal condition. However, because these laws vary in wording and provisions, the question whether such a directive, called the "living will," protects the rights of the terminally ill cannot be answered simply. A study of these laws establishes that legislatures have had difficulty in effectively allowing the person to exercise the right of determining medical treatment by a written declaration which is to become effective in the event of incompetency during terminal illness. While seeking to avoid the neces-

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sity of recourse to the courts for decisions when the patients are incompetent, these statutes have complicated the decision making process by being too narrowly or inadequately worded.

FOCUS ON FUNDAMENTAL POINTS

In a legal guide to medical treatment decisions, Concern for the Dying[1] (an educational council) succinctly states a number of fundamental points that should be kept in focus as the living will legislation is examined. The basic right of self-determination in regard to medical treatment has been upheld in the legal tradition by a number of judicial rulings that support it in terms of either a constitutional right of privacy or the common law right of bodily integrity. Because no right is absolute in the sense that it may be exercised at any time and under any condition, it must be weighed against compelling interests of the state: namely, the preservation of life, the protection of innocent third parties, the prevention of suicide, and the safeguarding of professional integrity. However, in rulings of appellate courts, the exercise of the right by a competent adult has generally taken precedence over the state's interests, the only exception being when the exercise of one's autonomy to refuse medical treatment has been seen to threaten the life or health of another person.

The refusal of a competent adult to accede to medical treatment has not been viewed as suicidal or as a threat to the integrity of the medical profession, nor, if it is carried out and the patient dies, as negligence for which the physician would be criminally or civilly liable.[2] In the case of an incompetent patient, there is a consensus in the judicial rulings that the determining factor in medical decisions should be the expressed wishes of the patient while competent. In the event the wishes are not clear, the "substituted judgment" principle has been applied by most courts. According to this principle, the one responsible for the decision attempts to interpret the wishes of the patient, drawing upon lifestyle and other evidence for an indication. If no evidence is available, the decision is based on the "objective test," which consists in asking what a prudent person would decide in the same circumstances. This amounts to determining what is in the best interest of the patient in view of the diagnosis and prognosis related to therapeutic options. Nevertheless, there are instances in which a dilemma has been created by medical technology that can sustain organic functioning and prolong the life of a terminally ill patient. Some physicians may feel the imperative to use such technology, but at the same time they are obliged to act in accordance with the wishes of the patient. A final point, which will be one of the major concerns of the following analysis, is the fact that the legal tradition is rooted in the moral tradition. Therefore, an understanding of the latter is required if law is to evolve properly in the area of legal directives and protection of rights.

OBJECTIVES OF THE ANALYSIS

The first objective of this analysis is to pinpoint the critical issues raised and addressed by these laws and what improvements need to be made on crucial points. If the primary intent of legislation is to recognize the legal force of the written declaration of a competent person in regard to treatment which is to be followed should that person become incompetent, such legislation should be characterized by ease of comprehension and use, accessibility to the public, and the prospect of being followed by physicians.[3] A number of commentators have noted that generally the statutes fall short of these characteristics. The present legislation turns out to be too complex or confusing in wording of key terms and principles, without proper sanctions for refusal to follow the directives, and needlessly demanding in regard to provisions for execution. Ironically, in the present atmosphere of anxiety about litigation, the failure of a patient to execute a living will may prompt physicians to avoid effective and humane decision making, which may now be deferred until the patient dies or a court decides.

The second objective is to evaluate recommendations made by various groups for reforms in the legislation and, after examination of their proposals, to offer some conclusions as to what type of legislation would best serve the interests of the patient.

STATUTORY PROBLEMS

The statutory provisions of the various living will laws present a variety of problems, including a lack of uniformity; the danger that the patient's right to forgo treatment may be ignored if a directive is not executed; the vague and narrow wording of the declaration and its definitions; the absence of strong sanctions to ensure enforceability of the directive; and the overly strict requirements for execution.

Each problem puts into question the usefulness of the living will; taken together, they cripple the effectiveness of present laws. The lack of uniformity is a significant problem since it may complicate the decision making for a patient whose will was executed according to the requirements of another state. This issue was addressed by the National Conference of Commissioners on Uniform State Laws (hereinafter the National Commissioners) in 1985. The difficulties created by specific provisions sometimes offset the benefits intended. They reside not only in the procedures designed as safeguards which in practice can become obstacles in the implementation of the wills, but also in the vagueness and imprecision of the terminology regarding key concepts.

A research monograph[4] prepared for the Oregon legislature in 1985 pinpoints the limitations of these laws. There is the danger that patients may lose rights already existing under common law. The failure to execute a directive may result in the presumption that the patients have decided to use all possible means to sustain life regardless of their condition. This may happen in those states that have no provisions for alternative decision making for incompetent patients without living wills. At least 11 states[5] (Arkansas, Connecticut, Florida, Iowa, Louisiana, New Mexico, North Carolina, Oregon, Texas, Utah, and Virginia) have provided for an alternative by formally recognizing in law the right of the family to make medical decisions in behalf of an incompetent adult patient who is "terminally ill" or in a "terminal condition."

The fact that many living will laws are not backed by strong sanctions puts their enforceability into question. A number of states require physicians to transfer patients if they refuse to follow the directives (for example, Alabama, California, Colorado, Florida, Georgia, Illinois, Kansas, Louisiana, New Mexico, Texas, Vermont, Wisconsin) and in a few states (for example, California, Colorado, Kansas, Texas, Wisconsin) physicians who do not transfer can face charges of unprofessional conduct. The most troublesome feature of these statutes is the language which needlessly restricts the application of the directives. Laws that stipulate that in order for the directive to be effective death must be "imminent" or "death must occur regardless of means being used at that time" impose problems of interpretation, especially in view of the fact that they give no clear definition of "imminent" or precise understanding of the relation of the treatment to the prospect of death.

Of the 38 declarations researched for the following analysis, the vast majority create problems in this area, and the others may be faulted for being too vague. The Indiana, South Carolina, and Hawaii declarations are the notable exceptions in avoiding some of the major pitfalls resulting from the use of such wording as "my death is imminent whether or not life-sustaining procedures are utilized" or "death will occur unless life-sustaining procedures are used." An additional problem is created by a few laws which require that there be a diagnosis of a terminal condition before the directive can be executed (California, Idaho, and Texas). George J. Annas and Leonard H. Glantz[6] sum up the legal picture in these words:

> As presently drafted, what these statutes primarily do is provide that if a patient is terminally ill, and if the physician can do nothing to sustain the patient's life, and if the patient does not want his life sustained, and the doctor agrees with the patient's decision, then the doctor *may* (but does not have to) follow the patient's desire, and be assured criminal and civil immunity for his actions.

This is hardly a reassuring view of the effectiveness of these statutes in protecting the rights of the patient.

THE NATIONAL COMMISSIONERS' CRITIQUE AND RECOMMENDATIONS

The National Conference of Commissioners on Uniform State Laws[7] expressed their concern for these problems when they stated that the purpose for the "Uniform Rights of the Terminally Ill Act" (hereinafter the act) was to present a model law that would eliminate the shortcomings of the statutes. They attempted to do so by focusing on the declaration and its key concepts. Though their critique is valid on a number of points, their own recommendations fall short of their goal.

The National Commissioners[7] cited the problems created by the definitions of "life-sustaining" and "terminal condition" in the statutes. The first problem was that "terminal condition" was defined in terms of "life-sustaining" and "life-sustaining" in terms of "terminal condition." Most laws define "terminal condition" to mean that death must occur or be imminent "whether or not" or "regardless of life sustaining treatment" and also define "life-sustaining" treatments or procedures as those which not only prolong dying but also fail to prevent death. This means that the two concepts cannot be separated because they are defined in terms of each other. If the definition of "life-sustaining" demands that death be inevitable even with the use of such treatment, then why consider withdrawing it at all? It is precisely because it is used that the dying process leading to clinical death is prolonged and the event of clinical death is delayed. Obviously the physician would face the question whether death will occur in spite of the treatment which is sustaining vital functions and postponing the event of clinical death. Such a question may not be answered in time to implement humanely the intent expressed by the living will.

The second problem is the notion that death must be imminent regardless of the treatment employed. This makes the living will difficult to apply to concrete cases because it is the very use of medical technology that prolongs the dying process and makes clinical death more remote. The lack of precise meaning of the word "imminent" in the laws is coupled with a variety of professional interpretations to make the provision hardly useful to one who desires to have life-sustaining treatment withheld or withdrawn when it only serves to prolong one's dying.

In a survey conducted one year after the adoption of the California Natural Death Act (1976), just over 83% of the physicians surveyed interpreted "imminent" to mean one week or less and 91% to mean two weeks or less.[8] If death must be expected to occur within one week or two weeks even with the use of an array of medical procedures, it does not seem likely that the living wills with such a stipulation will be useful in preventing the artificial prolongation of the dying process. To require that death be "imminent" despite the use of medical procedures is to make the decision on the cessation of aggressive medical treatment superfluous and living wills unnecessary. The probability that this language will promote discord rather than consensus can be concluded from the consideration that, as the President's Commission for the Study of Ethical Problems in Medical and Biomedical and Behavioral Research[9] observes, predictions about the time of death in the final stage of life are rather unreliable. It can be expected, then, that the prognosis of imminent death will vary from physician to physician, even if they should agree on the meaning of imminent, and correspondingly the determination of treatment will also vary.

The National Commissioners attempted to improve on other legal definitions and eliminate confusion by clarifying the meaning of "terminal condition" and "life-sustaining treatment," which they regard as crucial to the proper interpretation of their declaration. The declaration reads as follows:

> If I should have an incurable or irreversible condition that will cause my death within a relatively short time, and I am no longer able to make decisions regarding my medical treatment, I direct my attending physician, pursuant to the Uniform Rights of the Terminally Ill Act of this State, to withhold or withdraw treatment that only prolongs the process of dying and is not necessary to my comfort or to alleviate pain.[7]

By using the word "condition," the commissioners signify a state which could be caused by an accident as well as by an illness which involves the inevitable process leading to death. However, the definition of terminal condition is a narrower concept than illness or disease because it contains the notion of "a short time" and the feature of irreversibility, aspects not necessarily implied by terminal illness. They also separate the definition from that of life-sustaining treatment by noting that, without the use of life-sustaining treatment, death will occur within a relatively short time. The determination of a terminal condition, therefore, is made apart from the use of medical procedures and treatment. The insertion of the words "relatively short time" includes the restrictions of "imminent," but it avoids the problem of assessing a fixed period which the commissioners regard as too artificial since different disorders and diagnostic evaluations can lead to varying estimates even in similar cases or even in regard to the same person. According to their view,[7] the requirements that the condition be irreversible as well as incurable and that life-sustaining treatment serve only to prolong the dying process "exclude certain types of disorders, such as kidney disease requiring dialysis, and diabetes requiring continued use of insulin," since the treatment can reverse the disorder and does not serve "*only* to prolong the dying process." In this way, they hope to prevent an overly broad interpretation of "terminal condition." But unfortunately their own explanation does not harmonize with the wording of the definition, which regards a terminal condition as "an incurable or irreversible condition."

A CRITIQUE OF THE ACT

In an analysis of the act, Thomas J. Marzen[10] makes several criticisms which cut to the heart of the National Commissioners' purposes. The most pertinent criticism relates to the commission's analysis of what constitutes terminal condition. The commissioners really confuse matters when they insert "or" between the words "incurable" and "irreversible" in the definition. If one reads the comment, as Marzen notes, the explanation really warrants the conjunctive "and" by virtue of the explanation and examples given in regard to insulin and dialysis. In the view of the commissioners, kidney disease and diabetes are not regarded as terminal conditions if they can be reversed to some extent. If it were the intention of the commissioners to demand both incurability and irreversibility, Marzen asks why they did not state so in the definition, instead of leaving the interpretation to specific circumstances as to whether it be "or" or "and." Moreover, their comment on irreversibility contradicts the purpose of the act and other living will laws which would allow for the withholding or withdrawing of treatment which may sustain life indefinitely regardless of the overall condition of the patient. To follow their logic, one would have to conclude that any treatment—like insulin, dialysis, artificial hydration, artificial feeding, mechanical heart, respirator—which could keep specific vital functions going indefinitely may not be withheld or withdrawn if the patient would not die within a short time. For example, such treatment may not be withdrawn or withheld from a person in a "persistent vegetative state" as long as it can reverse, to some degree, the deterioration of specific functions. This conclusion follows logically from the commissioners' analysis of irreversibility. However, it is in direct contradiction to the purpose of the act and the recognition that it is precisely the new medical technology with its capability of prolonging life indefinitely which has prompted the living will legislation. This puzzling inconsistency is caused by the difficulty of translating moral concepts and principles into law. It may also reside in an inadequate understanding of these very concepts and principles which are not the sole possession of one religious tradition but now are widely shared in our society.

TRANSLATION OF MORAL CONCEPTS INTO LAW

The moral tradition has led the way to resolving the issues of withholding or withdrawing treatment in terminal cases. It has relied on the distinction between "ordinary" and "extraordinary" means as morally defined. It has applied the principles that one is not obliged to use extraordinary means to preserve one's life and that in the event of the patient's incompetence the next of kin has a primary role in making the decision in behalf of the patient in line with his or her wishes or best interests.

In the 1950s, Gerald Kelly[11] addressed the same issues addressed by the National Commissioners but with much more insight by making a clear moral distinction between the overall condition of the patient and the specific effects of the disorder. Perhaps the National Commissioners glimpsed the distinction when they insisted that what can be omitted are life-sustaining treatments which serve *only* to prolong the dying, but they failed to explain it clearly. The person with a diabetic or kidney disorder benefits from the treatment in such a way that one's living is enhanced; it is not merely that the dying is prolonged. The progress of the disease is significantly halted or reversed so that the benefits to the total condition outweigh the risks and burdens of the treatment. Kelly would have considered these "ordinary means" in the moral sense that they offer reasonable hope of benefit not outweighed by excessive pain, suffering, or other grave consequences including financial loss. The cases cited by Marzen to call into question the National Commissioners' analysis are such that, though the specific problem caused by the disorder may be remedied somewhat, the benefits to the patient's total condition are not significant enough to outweigh the disadvantages of the treatment. The overall condition, which is a result of various factors, remains terminal because the dying process continues and cannot be reversed but only prolonged.

Kelly addressed this issue after stating a moral principle traced back to the 16th century in the Roman Catholic tradition: One is not morally obliged to use "extraordinary means," that is, "all medicines, treatments, and operations, which cannot be obtained or used without excessive expense, pain or other inconveniences, or which, if used, would not offer a reasonable hope of benefit."[11] Kelly made clear that in the evaluation of "extraordinary" the primary concern is the effect of the treatment on the total well-being of the whole person and not its isolated effect on a particular disorder or its routine use. For example, he stated that "it is one thing to use oxygen to bring a person through a crisis; it is another to use it merely to prolong life when hope of recovery is practically negligible." [12] In his opinion, the evaluation of "extraordinary" could apply to the use of insulin for a diabetic cancer patient in the final stage of terminal illness because the focus should be on benefits to the total condition of the patient and not on the effects on a specific disorder. In line with the moral tradition, Kelly upheld the role of the family in making decisions to omit the use of extraordinary means in the best interest of the patient who is incapable of doing so. The moral concepts, principles and moral evaluation of Kelly's analysis were substantially restated by Pope Pius XII[13] in a speech to physicians and anesthesiologists on November 24, 1957. These were reiterated in the Vatican's "Declaration on Euthanasia"[14] on May 5, 1980. The declaration restates the moral evaluation by noting that, whether the older terminology relating to "extraordinary" is used or the means are discussed in terms of "proportionate" and "disproportionate," the moral evaluation of means amounts to the same considerations:

> . . . studying the type of treatment to be used, its degree of complexity or risk, its cost and possibilities of using it, and comparing these elements with the result that can be expected, taking into account the state of the sick person and his or her physical and moral resources.[14]

In commenting on this "burden-benefit" calculus, Richard McCormick[15] makes a significant point in stating that the quality of life or continued existence is a proper factor to be calculated in the evaluation. He correctly notes that the president's commission agrees with this point in defining the patient's best interest to "take into account such factors as the relief of suffering, the preservation or restoration of functioning, and the quality as well as the extent of the life sustained."[15] There are also two recent court decisions, *In re Conroy*,[16] and *Barber v Superior Court of State of California*,[17] which McCormick cites as having taken this approach.

Some of the more recent decisions[18-22] have also expanded the more narrow approach of a ruling of the New York State Court of Appeals, *In re Storar*,[23] in regard to what constitutes invasive medical treatment and what could be considered morally extraordinary in a given case. Anthony Storar was a mentally incompetent patient who was expected to die within six months from bladder cancer, and was in increasingly severe pain from the condition. He was receiving transfusions, and the majority ruled that the transfusions should not be stopped at the request of the mother because such treatment is ordinarily mandated by the courts in similar cases (eg, a child of Jehovah's Witness parents) and is tantamount to feeding. The minority opinion disputed the majority's interpretation of what constitutes extraordinary treatment, concluding that the transfusions were merely prolonging the painful dying process.

The interpretation of many living wills and the position of the National Commissioners seem to focus too narrowly on the effects of treatment on a disorder or disease without taking a broader view of the total condition despite the original intention to do so. If legislators understood the moral concepts, principles, and reasoning in the moral traditions, the living will statutes that attempt to incorporate them would be more practical and cause fewer legal complications.

TIME ELEMENT

The National Commissioners also addressed the time element in the declaration by using the concept of "relatively short time" as a substitute for "imminent," which they view as too restrictive and subjective. Though their intentions are good, they do not provide a remedy to the problem of subjectivity. Marzen[10] notes that physicians do not use such terminology and that there is no precise way of knowing what it means, nor is it clear what the short time is relative to. This difficulty makes the phrase as ambiguous as "imminent" is in the same context and subject to various interpretations. One could argue that the time reference should be left out of the declaration provided there is an adequate definition of "terminal condition." The interpretation of "relatively short time" can vary considerably in terms of hours, days, weeks, or months. If this is the case, and if "imminent" is too restrictive, then why not just leave out the time reference altogether? The difficulty of assigning a time period even to "terminal" is seen in the fact that some consider it to mean six months or less and others one year.[9,24] There are, therefore, sound reasons to exclude a time reference from the definitions and the declaration.

CENTRAL DEFINITIONS

There is a need for precise and uncomplicated definitions of both "terminal condition" and the nature of the treatment to be withheld or withdrawn under the living will. A "terminal condition" involves an irreversible dying process leading to clinical death. The condition is characterized by the irreversibility of the process, regardless of treatment. The time of clinical death may be proximate or remote, but such a consideration is not directly relevant to the establishment of the terminal condition. Once the irreversibility of the dying process is diagnosed, the terminal condition is established. What has plagued the other definitions is the failure to distinguish between the dying process and the event of clinical death, which is the irreversible cessation of all spontaneous functions of the brain or circulatory and respiratory functions.[25] One need not predict the latter in order to determine that the patient is in a terminal condition.

The usefulness of such a prognosis is questionable in view of the variables affecting predictions. It distracts one from focusing on the major question, which is whether the treatment is benefiting or burdening the patient. It does not foster clear thinking and may lead to some unusual conclusions, for example, that a patient who is irreversibly comatose with loss of neocortical functioning but with brain stem activity is not in a terminal condition because he or she can survive more than six months or a year. Before artificial life supports were used, how long would such a patient have survived? Now, with artificial feeding by nasogastric tube and hydration and other life supports to prevent failure of organ systems, such patients can survive many months or years. Yet a narrow legalistic approach, as, for example, under the Colorado living will, could lead to the conclusion that a person in a persistent vegetative state would not qualify for removal of medical treatment because that person "may be expected to 'live' for several years and thus is not in a terminal condition."[8] Is it conceivable that a person who has suffered the irreversible cessation of spontaneous integrated functioning of the neocortex and who is artificially maintained in an irreversible comatose state is not in an irreversible process of dying and therefore in a terminal condition? If the patient is not in a terminal condition, then what are we to call it? It is obvious that the preoccupation with the time element distracts from the real issues.

The definition of "life sustaining treatment" also needs revamping. The first step is to speak of "medical treatment," omitting "life-sustaining" because of its limiting connotation. The latter designation is superfluous because the main focus should be on the weighing of benefits and burdens of any medical treatment in regard to the total condition of the patient. The Legal Advisory Committee of Concern for Dying recommends a definition which fulfills this objective:

> "Medical procedure or treatment" shall mean any action taken by a physician or health care provider designed to diagnose, assess, or treat a disease, illness, or injury. These include, but are not limited to, surgery, drugs, transfusions, mechanical ventilation, dialysis, resuscitation, artificial feeding, and any other medical act designed for diagnosis, assessment or treatment.[1(p57)]

These measures are to be distinguished from "palliative care" which is intended for the comfort of the patient. The declaration which presupposes such a definition need only state that in a terminal condition one wishes withheld or withdrawn medical procedures or treatments which prolong the dying process and whose benefits, if any, are outweighed by the burdens imposed by prolongation of the dying process. The assessment of benefits and burdens of medical procedures and treatment should be made in reference to the total condition of the patient, and then there should be a weighing of whether the benefits are proportionate to the burdens. As stated above, one should avoid focusing on individual disorders and their respective treatment and make this evaluation in regard to the impact on the overall condition of the patient. According to this analysis, it is clear that, although use of insulin or dialysis in specific terminal cases may provide some benefits in terms of the specific conditions, the benefits are outweighed by the prolongation (sometimes painful) of the dying process which cannot be remedied or reversed by such treatment. In terms of the overall condition, the burdens outweigh the benefits.

If the National Commissioners had followed this line of reasoning, they could have clarified their position in regard to artificial feeding and hydration. Controversy has surrounded the issue whether these are medical treatments which can be omitted in certain cases where they help to prolong the dying without offering a proportionate benefit of relieving pain or discomfort. The commissioners[7] allow for the withdrawal or withholding in such cases (for example, patients in persistent vegetative state), as have several court decisions.[18–22] In fact, they allow a person to take the initiative in explicitly stating his or her wishes in regard to such treatment and regard these wishes as "controlling." Their position on this issue would have been clearer if they had made the distinctions discussed above. Ambiguity with regard to such treatments as dialysis or insulin could have been avoided. The commissioners could also have better explained why existing statutes are too limiting in language and usefulness.

THE NEW YORK STATE TASK FORCE ON LIFE AND THE LAW

In response to the obstacles faced by patients in the clinical situation and in an attempt to avoid the problems raised by current legislation, the New York State Task Force on Life and the Law set forth its recommendations in a report entitled *Life-Sustaining Treatment: Making Decisions and Appointing a Health Care Agent.*[26] The task force makes a strong recommendation for a law that will allow for the appointment of a surrogate, which it prefers to call a "health care agent," through a document called a "health care proxy." In its view, though the living will is useful, it cannot take into account all the various circumstances and alternatives of choice involved in real-life situations. In addition, the terminology of "extraordinary" or "heroic" measures is difficult to apply in concrete decisions because what may be extraordinary or heroic to one patient may not be to another. The appointment, therefore, of a health care agent best serves to resolve two major issues: the party who has responsibility to make medical decisions for the incompetent patient and the grounds on which the decision is to be made.

The task force concludes that the individual should be allowed by law to appoint an agent and have the opportunity to direct the agent either in writing or orally concerning decisions on treatment or nontreatment, medical personnel, access to and disclosure of medical information, and use of funds for treatment. Hence, under the guidance of the patient's general and more specific instructions, the health care agent is in the position to make decisions in regard to the patient's prognosis, treatment alternatives, and possible consequences in response to changing events and information.

These recommendations make eminent sense when compared to the vagueness and limited utility of the majority of statutes. However, several observations are in order. While the appointment of a health care agent, backed by general and specific guidance from the patient, is an ideal way for the patient to maintain control over health care decisions in the event of incompetency, it still does not eliminate the need for the statute to include a clearly worded standard declaration. There are three obvious reasons for this need. The first is that many patients will not have the opportunity to appoint a surrogate. The second is that many people find it difficult to write directives and need some guidance. A general declaration will provide a structure and help them to begin the process. It can be useful and instructive to individuals who are uncomfortable writing any instruction (consider the difficulties in writing wills) regarding their death or dying. The individual will thus have some support in facing issues which are psychologically difficult to confront. The declaration should be carefully and clearly worded, so that anyone can understand its meaning.

The third reason for a declaration is that the health care agent may not be available to substitute his or her judgment for the patient's. Without a commonly understood and legally supported declaration, there is the possibility that the meaning of a personally drafted directive may not be clear, especially in the absence of the duly appointed surrogate to interpret it.

REVISED DECLARATION AND DURABLE POWER OF ATTORNEY

In light of the aforementioned definitions and evaluations, a revised declaration would read as follows:

> If I should be in a terminal condition, a state which involves the irreversible process of dying resulting inevitably in clinical death, and I am no longer able to make decisions regarding my medical treatment, I direct my attending physician to withhold or withdraw treatments which prolong the process of dying and whose benefits are outweighed by the burdens they create for me during the process of dying. I desire only that care which is necessary to provide me comfort and alleviate my pain.

At this point, in accordance with the recommendations of Concern for Dying,[1] optional specific provisions could be included, relating, for example, to such treatment or procedures as nasogastric tube feeding in the event of an incapacity to take nourishment naturally because of paralysis or comatose state; or cardiopulmonary resuscitation in the event of cardiac arrest. However, these specific provisions may not be necessary if the wording of the aforementioned declaration is properly understood. Because specific provisions may not cover all circumstances, it would be a good idea to incorporate into the living will document an option for the durable power of attorney for medical decision making. This allows one to designate someone else to serve as one's proxy or surrogate (health care agent, sometimes referred to as attorney-in-fact) if one is unable to make the decision. This recommendation is extended by Concern for Dying to appointing more than one proxy and prioritizing one's preferences.

Despite the shortcomings in the definitions and wording of the declaration, some statutes have made a significant step in sanctioning living wills or directives that include the option in regard to artificial nutrition and hydration (for example, Alaska, California, Hawaii, Illinois, New Mexico, Oklahoma). Others have allowed in the directive an option for the durable power of attorney (Delaware, Louisiana, Wyoming). Other state statutes include both options in the directive (Florida, Texas).

In order to complement the living will, some states have passed separate laws sanctioning the durable power of attorney specifically for medical treatment decisions (for example, California, Colorado, Illinois, Nevada, Oklahoma, Virginia). As stated above, the durable power of attorney provides a way of resolving issues on which the declaration does not express clearly the person's wishes. However, it is preferable that the option for the durable power of attorney be incorporated into the same document as the living will. Such a document would serve two purposes. It would help to remedy the procedural problems raised by overly complex separate durable power of attorney laws, such as California's, and by strict provisions for witness qualification.[6(p138),27] It would also help the public to understand more readily the relation between the two and may facilitate the execution of both at the same time.

The individual designated to be the surrogate should be in a position to know deeply the wishes and concerns underlying the directive. Having the best interests of the patient at heart and the directive in mind, he or she should be able to interpret what that person would decide in regard to treatment in a particular circumstance. In the event that the surrogate is unavailable to make the decision, the wishes stated in the directive would be decisive in regard to treatment.

Some legal commentators[28] and legislators support only the durable power of attorney law. The legislation proposed in Michigan[29] is well-intentioned in attempting to avoid the notion that it is merely "right-to-die" legislation since in that scheme the surrogate, following the wishes of the patient, may decide for continuation of treatment, even experimental, as well as for cessation. But the directive on the part of the patient is still needed both as a guide to the surrogate and as a check on the surrogate's decisions. It would be appropriate to know whether the surrogate is reflecting the basic wishes of the patient. The written declaration provides this criterion as well as a legal basis for decision making in the absence of the surrogate.

The living will document should be witnessed and notarized by a notary public. Various states' statutory requirements have excluded a number of parties from being witnesses: spouse, blood relative, beneficiary, physician, physician's employee, employee of patient's health facility, financial supporters of the patient's health care.[10] The National Commissioners[7] have omitted any qualifications for witnesses in the act for two reasons: so that the legal requirements are not so complicated as to prevent execution in difficult circumstances, and to relieve physicians of the legal burden of determining whether the requirements are fulfilled. In their view, abuse can be adequately prevented by the fact that criminal penalties safeguard the proper execution, revocation, and implementation of the directive and by the fact that health care professionals, particularly the physician who records the declaration in the medical record, will be aware of any abuses through discussion with patient and family. This may be unrealistic, especially in regard to the elderly and mentally ill patients who have no family or are susceptible to institutional exploitation. The laws should provide for an advocate or ombudsman who will diligently look after the best interests of such patients.

One hopes that frank discussion between all concerned parties will take place before the directive is executed and during the course of the terminal condition. There is the danger, however, that the execution of the will may become just a formality that substitutes for communication. This risk points up why it makes good sense to have the durable power of attorney part of the directive so that there will be someone there to continue the dialogue throughout the process.

Any suggestion that the law allow one to delegate decision making to the surrogate when one is still competent is misguided. Such a proposal would facilitate the withdrawal of the patient from the process. This result would be the very opposite of what the legislation intends to promote, namely, the patient's maintaining control over the decisions relating to health care. The signing of the directive which expresses one's wishes in regard to treatment and appointment of a surrogate should never be viewed as a surrender to the "fate" of a terminal condition or an abdication of personal responsibility but rather as an expression of freedom and dignity. Viewed in this way, it can provide great comfort and a psychological support in facing a life-threatening condition. It can give one a sense of confidence that one's final days will not be taken from one's control.

CONCLUSION

The living will is a proper instrument for protecting the rights of the terminally ill person provided that it is carefully drafted in the crucial parts of the declaration and the provisions for execution, revocation, and implementation are uncomplicated. At the present state of the law, this is not universally the case. The statutes are in need of revision in all areas, but particularly in regard to the declaration. The directive should include both the living will declaration, unencumbered by vague and conflicting terminology and backed by clear definitions, and also an option to appoint a surrogate or proxy as a way of complementing and interpreting the intentions and wishes of the patient. The durable power of attorney provision would be helpful in protecting the rights of the patient and promoting communication between physician and family. It should not be viewed as a substitute for a well-written living will because of the possibility that the surrogate may not be available for decision making. The states should revise their living will laws to achieve uniformity, which is needed to protect the rights of individuals as they travel or move from state to state. What we want to avoid are laws that are so demanding or confusing in their requirements and so rigidly interpreted that they stifle compassionate decision making and actually prevent humane decisions which would have been made if the laws had not been passed.

The New York State law on do not resuscitate orders,[30] approved though restricted to decisions involving cardiopulmonary resuscitation, provides a recent example of legislation that is well intentioned but rather complex in its provisions. It covers everything from decision making of the adult with capacity to surrogate decision making, decision making on behalf of a minor patient, revocation, physician review, interinstitutional transfer, dispute mediation, judicial review, and judicial approval. The law has many good features and is thorough in covering essential issues. However, if followed to the letter, it could actually prolong needless resuscitation and the dying process. In one particular provision, for example, there is a requirement that, in the absence of a duly witnessed request of the patient or a surrogate, "the attending physician determines, in writing, that, to a reasonable degree of medical certainty, resuscitation would be futile, and another physician selected by a person authorized by the hospital to make such selection, after personal examination of the patient, reviews and concurs in writing with such determination."[30] A delay in fulfilling this provision could force the staff to continue the resuscitation efforts against their best judgment until the written order is received and confirmed in writing.[31] Hopefully New York State's living will legislation will be designed to avoid the necessity for complex procedures and documentation that can thwart compassionate decision making. The protection which the living will can provide is ultimately only as good as the understanding legislators incorporate into the legislation.

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Fetal therapy and surgery

Fetal rights versus maternal obligations

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On June 16, 1987, a judge of the District of Columbia Superior Court made a decision, upheld by the Court of Appeals, that elicited enormous debate in legal and medical circles. A 28-year-old married woman was approximately 25 weeks pregnant when she learned that her cancer, which she thought was in remission, had spread to her lungs. One week later, with death apparently imminent, the patient and her doctors decided against an emergency cesarean section. In response to a request by the hospital for a judicial decision, the judge overruled the wishes of the patient, her family, and her physicians. When told that the judge had ordered the surgery, the patient said twice, "I don't want it done." Her attorney requested a hearing before the local appeals court, which held a hearing by telephone and refused to issue a stay. The baby died two hours after the operation, and the patient died two days later.

Strongly critical of the judicial decision, Annas[1] stated that the judges

> treated a live woman as though she were already dead, forced her to undergo an abortion, and then justified their brutal and unprincipled opinion on the basis that she was almost dead and her fetus's interests in life outweighed any interest she might have in her own life or health.

To Annas, it is clear that the patient's wishes should have been honored. The patient's attorney[2] commented that his client's case is a good example of how the system failed even at a highly respected academic institution. "This case was one that cried out for intervention from a hospital ethics committee." On November 24, 1987, 39 organizations, including the American Medical Association, asked the District of Columbia Court of Appeals to reconsider its ruling that a gravely ill pregnant woman could be forced to undergo a cesarean delivery against her wishes.[3]

COURT-ORDERED CESAREAN SECTIONS

A recent national survey[4] reported that court orders have been obtained for cesarean sections in 11 states, for hospital detentions in two states, and for intrauterine transfusions in one state. Among 21 cases in which court orders were sought, the orders were obtained in 86%, mostly within six hours. The authors concluded that court-ordered obstetrical procedures represent an important and growing problem that evokes sharply divided responses from obstetricians; such procedures are based on dubious grounds and may have far-reaching implications for obstetrical practice and maternal and infant health. In an accompanying editorial, Annas[5] stated that before birth we have access to the fetus only through its mother and, in the absence of her informed consent, can do so only "by treating her as a fetal container, a nonperson without rights to bodily integrity."

The article and editorial generated considerable discussion. One writer[6] suggested that mothers should be held criminally or civilly liable after birth for behavior that seriously injures their offspring, since pregnant women have no moral right to injure children who would otherwise be born healthy. Another writer[7] castigated Annas for categorically voiding the obligation of the obstetrician to the fetus.

In recent years, courts and state legislatures have increasingly granted rights to fetuses traditionally enjoyed by persons. Some of these fetal rights conflict with a pregnant woman's constitutional rights to liberty, privacy, and equal protection.[8] Does a brain dead pregnant woman not have a right to be buried promptly? Yet such women have been artificially ventilated and maintained for weeks or months to allow a viable baby to be born.[9] Is it legitimate to use the body of a newly deceased woman to serve the interests of her fetus?[10] Why should her organs not be used (with consent) for transplantation to save the life of other persons with "full rights" rather than maintaining her to save her fetus who has only "partial rights?"

FETAL TISSUE TRANSPLANTATION

Another situation of potential conflict between mother and fetus is fetal tissue transplantation and embryo research. In Sweden[11] and Mexico,[12] patients with Parkinson disease were treated with adrenal medullary autografts to the brain. Because of the need in such transplants to perform both laparotomy and craniotomy in elderly pa-

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tients, investigators turned to the use of fetal tissue transplants for patients with Parkinson disease.[13] Such fetal grafts have already been performed in the People's Republic of China, Mexico, and Sweden[14] and raise many ethical questions: Should doctors be allowed to use tissue from intentionally aborted fetuses to alleviate an otherwise hopeless condition? Some women have suggested that they become pregnant for the purpose of providing fetal tissue to treat themselves (eg, with fetal pancreatic cells for maternal diabetes) or a family member (eg, a father with Alzheimer disease). Are women to become "fetal factories" to provide organs or tissues for transplantation? Is the killing and dissection of the fetus an abuse of a developing or potential human being? Is the risk to the recipient negligible? What are the social consequences of the medical use of human fetal tissue? The personhood of an unborn fetus is a subject of great moral and legal debate.

The scientific, social, and ethical perspectives of the fetus as a transplant donor are extensively described by McCullagh.[15] Detailed ethical discussions of fetal tissue transplants include those of Fine[16] and Mahowald and colleagues.[17] Fine opines that "fetal tissue transplantation circumscribed by medical and moral limits will not erode important ethical values, but the pace of scientific research must not preempt public debate and a verdict consistent with societal values." Mahowald et al state that whether or not abortion is morally justified, use of human fetal tissue for research or therapy is justified in certain circumstances. The rationale, both for permitting transplantation of fetal tissue and for limitations in exercising the technology, continue these authors, is "based on the same set of ethical principles that supported restrictive legislation in the past: respect for autonomy and a balancing of harms and benefits that gives priority to those most affected."

In spite of the above-cited ethical justification, the Department of Health and Human Services on March 22, 1988, denied a National Institutes of Health request to perform experiments on patients with Parkinson disease using brain tissue from aborted fetuses[18] because "the proposal raises a number of questions—primarily ethical and legal—that have not been satisfactorily addressed, either within the Public Health Service or within society at large."

The moratorium on research funded by the US Public Health Service utilizing human fetal tissue obtained from induced abortions for therapeutic transplantations does not apply to therapeutic research using human fetal tissue from spontaneous abortions or stillbirths. The director of the NIH was asked to convene an ad hoc advisory committee to consider the implications of human fetal tissue transplantation research and to examine the appropriate circumstances for the therapeutic use of human fetal tissue obtained from induced abortions.

Tissue and organ transplants from anencephalic fetuses and newborns are discussed by us elsewhere.[19] Although tissue transplants from fetuses and anencephalic neonates are a topic of active debate in the medical and lay press, the issue of research on embryos 14 days or younger has not yet been resolved. In 1985, the United State Congress imposed a three-year moratorium on funding from the National Institutes of Health for embryo research.[20] In October 1988, the NIH advisory panel found the use of fetal tissue acceptable for medical research, provided certain safeguards are in place to protect the fetus from "ulterior motives" in a woman's decision to seek abortion. A draft executive order from the White House, however, would continue the moratorium on federal funding for human fetal research.

The American Medical Association has published guidelines for fetal research[21] but has no position relating specifically to fetal tissue transplants or the use of anencephalic fetuses or neonates as organ or tissue donors.

FETAL THERAPY

The technique of amniocentesis was developed in 1952 to measure amniotic fluid bilirubin to evaluate the severity of fetal Rh isoimmunization.[22] Intrauterine transfusion for severe Rh hemolytic disease has been practiced since 1963[23] with considerable success, although the disease has practically disappeared since the widespread use of Rh immune globulin.

Other fetal conditions now able to be diagnosed which may be treated medically in utero are cited by Harrison et al.[24] These authors gave glucocorticoids to pregnant women to increase deficient fetal pulmonary surfactant in pulmonary immaturity. They treated severe hydrops by administering digitalis and diuretics in addition to intrauterine transfusion of blood. They gave massive doses of vitamin B_{12} to a pregnant woman whose fetus had vitamin B_{12} responsive methylmalonic acidemia. They also administered pharmacologic doses of biotin to a woman whose fetus had multiple decarboxylase deficiency. These authors suggested that intraamniotic thyroid hormone can be used to treat congenital hypothyroidism and goiter and to help mature the fetal lung. Furthermore, the intrauterine growth-retarded fetus might be fed orally by instilling nutrients into the amniotic fluid.

Another group of investigators gave drug therapy to suppress the fetal adrenal glands in a case of suspected congenital adrenal hyperplasia.[25] Yet other authors have successfully treated fetal congestive heart failure by giving digoxin to the mother.[26]

FETAL SURGERY

The first reported fetal surgery was performed in April 1981 in San Francisco on a 31-week-old fetal twin suffering from severe bilateral hydronephrosis.[27] The affected twin had a suprapubic cystostomy performed percutaneously. The catheter functioned well, and both babies were delivered successfully. Other early approaches included percutaneous nephrostomy in utero,[28] repeated transabdominal renocentesis,[29] and hydronephrotic decompression through hysterotomy.[30] More recently, catheter shunts between the obstructed fetal urinary tract and the amniotic fluid have been performed in many cases.

Decompression of fetal hydrocephalus was first performed in Boston[31] and Denver[32] by attempts to place an indwelling catheter to produce long term ventriculo-amniotic shunts.

The International Fetal Surgery Registry reported[33] that between 1982 and 1985, 73 placements of catheter shunts for fetal obstructive uropathy and 44 drainage procedures for obstructive hydrocephalus were performed. The attempts to decompress the obstructed fetal urinary tracts resulted in the survival of 30 fetuses (41%). Pulmonary hypoplasia was the major cause of death in both treated and untreated fetuses.

The results of shunt procedures for obstructive hydrocephalus were less encouraging. Although 34 of 44 (83%) fetuses survived, the procedure-related death rate was 10%, more than half the survivors have serious neurologic handicaps, and only one third are developing normally. The report concludes with the strong recommendation for prospective controlled trials of these still experimental fetal surgical interventions.

A third fetal abnormality potentially correctable by surgery in utero is diaphragmatic hernia; however, most of these fetuses also have lethal nonpulmonary associated anomalies at autopsy.[34]

Other fetal interventions are also possible. One fetus with bilateral pleural effusions on ultrasound examination had two intrauterine thoracenteses and a catheter left in the left pleural cavity until just before vaginal delivery.[35] It seems likely that surgical correction of spina bifida and some forms of congenital heart disease may be possible before birth in the near future.[36] Some authors even suggest that cleft lips[37] and hand deformities[38] might be surgically corrected antenatally.

Ethical Issues

In 1981, Fletcher[39] pointed out four ethical issues in fetal therapy. The first is the conflict between the perceived interests of the fetus with a correctable defect and the interests of the parents, especially the mother, who must give consent for the fetal medical or surgical intervention. The second ethical issue concerns the apparent inconsistency of encouraging fetal therapy on the one hand by considering the fetus as a "patient," and respecting parental choice about abortion on the other. The third ethical issue concerns the proper conditions for learning about the fetus as an object of therapy, ie, ethical guidelines for fetal research. The final issue is the question of the social and economic priority that should be assigned to investigations of the risks and benefits of fetal therapy.

In a later paper, Fletcher[40] raised additional ethical considerations, including prior group review of fetal experimental protocols, selection of fetal candidates for therapy, and the informed consent process. He suggested that research protocols should be approved in advance by institutional review boards according to the guidelines for fetal experimentation in federal research regulations that protect the human fetus and the pregnant woman. He proposed criteria for the selection of fetal candidates for in utero surgery which would maximize benefit and minimize harm to the fetus. The risk to the pregnant mother must also be considered.

Fletcher asserted that informed consent for fetal therapy may be more difficult to obtain from the parents than in other health care situations. The affected fetus is nearly always a wanted child. Many parents are either morally opposed to abortion, especially when fetal therapy is offered as an alternative, however risky the procedure. Thus, a careful consent process should be designed and implemented.

Fletcher also discussed two long term issues. What is society's obligation to protect the treatable, nonviable human fetus? The ethics of abortion and the ethics of fetal therapy will collide. Abortion gives the mother's interests precedence when these are in conflict with those of a nonviable fetus. Fetal therapy, however, requires society to protect the treatable viable fetus.

Further, is fetal therapy worth the costs of developing it for wide use? Will the correction of fetal disorders simply preserve more genetic disorders in the population and add to the burden of suffering? Fletcher concludes that

> the benefits to society are indeed richer than the economic savings of fetal therapy . . . .The economic investment to develop and expand fetal therapy represents society's interest in the life and well-being of the fetus with a treatable congenital malformation and the family in which this hopefully more frequent event occurs.

McCormick[41] pointed out that the 1973 Supreme Court decision in *Roe v Wade* gives the legal decision whether to perform an abortion until the time of fetal viability to the prospective mother. However, from an ethical viewpoint, the answer to the question of who has the right to choose among the alternatives for a defective fetus (eg, treatment, nontreatment, or abortion) "would come out differently depending on what ethical position is held on abortion." McCormick's view is that when effective fetal therapy or surgery is available, treatment is the only morally defensible alternative.

Elias and Annas[42] reiterated the concern about the fact that fetal surgery is still experimental and, therefore, all the appropriate federal, state, and local guidelines for human experimentation and fetal research must be followed. Thus, the research should "meet the health needs of the particular fetus placed at risk only to the minimum extent necessary to meet such needs." The consent of both the mother and father is required. These authors also pointed out that the issue of resource allocation of societal funds to fetal research is fundamentally a political one but with ethical overtones. Should society spend more money to extend the life of the elderly or provide some of these funds to help fetuses and neonates with the best chance to live a healthy life?

Elias and Annas discussed the enhanced status of the fetus in light of developments in fetal therapy and surgery.

> These innovations raise complex ethical questions about the rights of the mother and fetus as patients. If both are really patients, do both need their own physicians? Is the obstetrician to view the fetus or the mother as his patient? Usually, there is no need to make a distinction, but what if the physician believes a procedure [such as fetal surgery, if and when it becomes "accepted medical practice"] is indicated and the mother refuses to consent? At least two practices are possible: 1) try to persuade the mother, but if she will not change her mind, do what she wants; 2) take legal action to try to compel the woman to submit to the procedure.

If the procedure can be done with minimal invasiveness and risk to the mother and significant benefit to the fetus, there is an argument to be made that the woman should not be permitted to reject it. Such rejection of therapy could be considered "fetal abuse" and, at a late stage in pregnancy, "child abuse," and an appropriate court order sought to force treatment. Such forced treatments raise difficult ethical issues. The above authors point out two major problems: 1) a woman is coerced to undergo a procedure for the benefit of another; 2) one must weigh the indignity and deprival of autonomy involved in invading the woman's body against the potential benefit to life and health of the fetus. From a functional point of view the question posed by Elias and Annas is as follows: Whether we call the fetus a person, a patient, or a fetus, what rights will we accord it? When will we leave all decisions regarding its health to its mother, and when, if ever, will we as a society decide to restrict the autonomy of pregnant women for the sake of their fetuses? These authors conclude that

> respect for the pregnant woman's dignity and autonomy and the tradition of medicine as a voluntary service require the voluntary and informed consent of the pregnant woman prior to the performance of fetal research or therapy. The right to make a mistake should continue to be the pregnant women's, not the physician's or the judge's.

LEGAL ISSUES

One of the most difficult legal issues presently being faced is how to balance the rights of the mother and the medical needs of the fetus when they conflict. The basic issue is whether the fetus is a patient separate from its mother. Although many obstetricians view the mother and fetus as a single biological entity, this perception is bound to be altered in the light of advances in fetal care that clearly differentiate the fetus from its mother for treatment purposes. Moreover, most perinatologists are advocates on behalf of the fetus. Thus, advances in fetal surgery are accentuating the potential and actual conflict between maternal interests and the interests of the fetus as a patient.

Blanck[43] has discussed women's rights and responsibilities during gestation. He pointed out that the mother's right to autonomy and privacy in reproduction is fundamental, but like all other constitutional rights, it is not absolute. He quoted Robertson, who said the following:

> The mother has, if she conceives and chooses not to abort, a legal and moral duty to bring the child into the world as healthy as is reasonably possible. She has a duty to avoid actions or omissions that will damage the fetus and child, just as she has a duty to protect the child's welfare once it is born until she transfers this duty to another. In terms of fetal rights, a fetus has no right to be conceived—or, once conceived, to be carried to viability. But once the mother decides not to terminate the pregnancy, the viable fetus acquires rights to have the mother conduct her life in ways that will not injure it.

Blanck further asserted that the core of the debate over the legal parameters of maternal responsibility is the question of the legal status of the fetus with seemingly paradoxically opposing legal rulings. In *Roe v Wade*, the Supreme Court clearly stated that the fetus is not a person and may be aborted at the discretion of the mother. On the other hand, case law in most jurisdictions accepts liability for death or injury negligently caused at the preventable stage. These facts lead to "the paradox that a pregnant woman might be sued for harming but not for aborting a fetus."[43]

There is an emerging trend in tort law toward recognition of maternal responsibility for the well-being of the unborn child. Blanck[43] reviewed all the legal cases involving court-ordered interventions on behalf of the unborn fetus and pointed out that "the availability of a broad array of prenatal diagnostic techniques such as amniocentesis, ultrasound and fetoscopy, along with advances in genetic screening and collaborative conception techniques, place increased pressures on parents to use these technologies."

Robertson[44] stated that the constitutional right of procreative autonomy involves not only the decision to reproduce but also the decision not to reproduce and control of the characteristics of offspring. He traced the history of the legal regulation of in utero therapy and discussed the unusual situation of twins where one fetus is healthy and the other fetus needs intervention. Selective abortion of a Down syndrome twin at 20 weeks with live birth of the healthy twin was first performed by Kerenyi and Chitkara.[45]

Robertson concluded that neither law nor ethics present any barrier to the resolution of novel legal and ethical problems posed by in utero surgery. Doctors and parents acting in good faith may use an experimental in utero intervention on a fetus that is at great risk without the procedure, but they are not legally or morally obligated to do so. When the therapy is established, a duty to use it may arise, and a mother's refusal of the intervention may be penalized or overridden by the courts.[44]

Johnsen[46] also pointed out that our legal system has historically treated the fetus as part of the mother and has afforded it no independent rights. In recent years, however, courts and state legislatures have increasingly granted fetuses rights traditionally enjoyed by persons. Johnsen traced the development and recent expansion of fetal rights as well as the constitutional limitations on the recognition of fetal rights. She concluded that any law granting rights to the fetus should not put women at a disadvantage or in any way infringe on the autonomy of pregnant women.

In a specific case of a court-ordered cesarean section to safeguard the life of the unborn infant in spite of the objections of the mother, Bowes and Selgestad[47] cited the court decision which supported five legal points: jurisdiction of the juvenile court to act; compelling interest of the state to protect the unborn fetus; extant protectable rights for a full term viable fetus; validity of an order by a court of competent jurisdiction to order medical procedures to protect the unborn; and potential liability of physicians requiring proper court action. Leiberman et al[48] plead for a law in this country to make it a felony for a woman to refuse surgery that would save the life of her fetus. But Shriner[49] correctly pointed out that such a statute would have to exempt good faith refusals based on a woman's fear for her own life, or prejudice, ignorance, language difficulty, inadequate doctor-patient rapport, or other "occult" reason for maternal refusal of surgery.

Engelhardt[50] believes that the availability of abortion has paradoxically increased the plausibility of maternal duties to fetuses, in that a woman's continuing with a pregnancy is an expression of her free choice. These duties will surely lead to cases in which one will be able to argue successfully that "it will be morally permissible to compel a cesarean section and perhaps even fetal surgical procedure."[50]

On the other hand, Nelson and Milliken[51] have argued that the pregnant woman's ethical obligation to care for her fetus should not be legally enforced. These authors discussed the ethical basis of the physician-patient relationship during pregnancy and presented their perspective on the ethical reconciliation of maternal-fetal conflict. They also analyzed the legal aspects of physician management of pregnant women who refuse medically indicated treatment. Finally, they presented policy reasons why neither the medical profession nor society should support judicially compelled treatment of pregnant women.

MATERNAL RIGHTS—FETAL RIGHTS

With improved technology and enhanced fetal legal status, well thought out guidelines that safeguard and protect both the rights of the fetus and the mother must emerge. Although the general operating premise of most physicians is to consider primarily the maternal rights, it is important that the well-being of the fetus also be considered.[52] The emerging rights of the unborn, where they conflict with the health and personal interests of the mother, present complex questions, especially when the rights pertaining to viability conflict with the choices of the pregnant woman.[53]

The American College of Obstetricians and Gynecologists' Committee on Ethics issued a statement in 1987[54] which asserted that "the maternal-fetal relationship remains a unique one, requiring a balance of maternal health, autonomy, and fetal needs. Every reasonable effort should be made to protect the fetus, but the pregnant woman's autonomy should be respected." The statement recommended consultation with an institutional ethics committee when appropriate. "The use of the courts to resolve these conflicts is almost never warranted."

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Himself, Mrs Plucker, and the rites of spring

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He was a large man who had a quick step and a slight bounce to his walk, a warm smile, and a pleasant habit of mischievously peering out over the top of his spectacles. The wooden barracks where we worked creaked and echoed; yet his approach was almost always silent. What told us he was coming was the distinctive push he gave to the Hollywood-style saloon doors that set the infirmary off from the rest of the corridor. "Good morning," he would say, standing in the doorway, not stepping in until he was invited. He had a booming voice, an infectious smile, and quickly put us at ease. As he stepped over the threshold he always removed the brown straw hat that seemed so much a natural part of his anatomy. He asked us how everything was, inquired about the sick and injured, and amused us by offering therapeutic advice after listening to case histories. He had absolutely no medical training, but his interest in medicine was a passionate one, forged in part by personal conquest of illness. He willingly assumed the burden of concern for the sick and injured, no matter who they were, and spared no effort or expense to help them. We saw him as a man of unique admirable qualities who inspired loyalty, respect, and affection. Yet the public he had left behind in Brooklyn, New York, saw him from afar through their disappointment and anger.

Although Walter O'Malley had moved the Dodger baseball team from Brooklyn to Los Angeles in 1958, the links to Brooklyn were more than residual. One of these links was Dr Herbert C. Fett, the former team physician who was Chief of Orthopedic Surgery at the Long Island College Hospital where I was doing my residency in internal medicine. For a number of years, Dr Fett sent house staff volunteers to Dodgertown, the spring training camp in Vero Beach, Florida. They provided front line medical services for close to 400 players and staff during the two months of spring training. At first blush virtually every resident expressed interest. However, the number of volunteers rapidly dwindled when it became known that the time away would count against annual leave.

Dr Fett personally screened the few candidates there were, along with Mrs Anastasia Plucker, the chief nurse on Neurosurgery, who regularly went to Dodgertown for two months every year. Mrs Plucker, and I still refer to her as such, was a formidable combination of mother figure and field marshal who ruled rather than administered the Neurosurgery floor. The house staff and attending physicians stood in awe of her, greatly respected her knowledge and ability, and entered her domain through sufferance. Mrs Plucker had a biblical interpretation of people. They were either good or bad; there was no middle ground. She had a formidable presence and could with but a stare let people know what she thought of them.

Mrs Plucker took an instant liking to me and as with all those whom she liked, effortlessly overlooked my professional and personal shortcomings. As far as she was concerned I was perfect and could do no wrong. I think that her response to me was determined in part by the fact that she lived in a brownstone on Douglas Street in Brooklyn, two blocks from my grandparents' former home where for many years one of my uncles had his medical practice. Mrs Plucker knew my family, worshipped in the same church, and saw me as a product of the neighborhood of which she was fiercely proud. For she always spoke of "our neighborhood" and "our church." It didn't seem to matter to her that I had never lived in Brooklyn. When once I mentioned this to her she said with an Irish lilt to her voice, "Oh now don't be contradicting me."

Until I went there, Dodgertown was an abstraction set in the Florida sunshine. There was no formal orientation, just assurances from Dr Fett, Mrs Plucker, and other residents who had been there that I would do well. It never occurred to me at the time, although it has since, that in principle a resident in orthopedic surgery or one in sports medicine would have been better suited as a baseball camp physician than a resident in internal medicine. Sports medicine had barely emerged as a specialty at that time and no orthopedic residents were available. Yet I learned soon after my arrival that accidents were few and usually not serious and that the commonest problems among the several hundred healthy adult males in the camp were upper respiratory tract infections and heat exhaustion. An excellent surgeon in the town of Vero Beach, Dr Phil D. Morgan, managed all major injury problems, and there were other specialists in town to whom players could be referred if the need arose.

In March of 1964 I rode down to Vero Beach from New York with David Handler, the Director of Laboratories at Long Island College Hospital, who was relocating to Florida. I always thought that Dave had missed his vocation in life and should have been a jet pilot. We made it to Vero Beach in a day and a half, and at times it seemed to me that we rode a few inches above the roadway. Mrs Plucker, who knew how fast Dave drove, was fretful when we walked into the infirmary. "You see, I delivered him to

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you safe and sound," Dave said jokingly. She wasn't amused.

Dodgertown is a former naval air station which at that time consisted of two large wooden barracks buildings, each two stories high and set on stilts. The infirmary was in a wing at the end of the compound, adjacent to an Olympic-sized swimming pool which was rarely used. The infirmary consisted of several rooms, two of which had three beds each for inpatient care. Mrs Plucker's own room was adjacent to the infirmary while the physician's room was in the other barracks building. Everyone lived in the barracks: players, administrators, staff, the press corps, and visitors. The barracks were spartan and spare but clean and comfortable. Yet the walls were thin and even a light footed person made the floors vibrate. As lines of players rolled down the stairs and left for their daily practice sessions Mrs Plucker would remark, "They'll bring the heavens down on us yet." There were two baseball equipment items that were prohibited in the barracks, bats and cleats, the former because they could become weapons in a heated argument and the latter because they would have wreaked havoc on the floors.

After I had gotten my bags to my room, Mrs Plucker said, "Now himself will be wanting to meet you." I didn't know who "himself" was and when I asked she replied, "Why, Mr Walter O'Malley, of course." Mrs Plucker had obviously acted as a one-person advance publicity team judging from the reception Mr O'Malley gave me. He was seated behind a desk in his no-frills office in the barracks. It opened onto the central corridor and as I would later learn he regularly hailed people down as they passed by.

The only advice Mr O'Malley ever gave me was sound. I was part of management and had to avoid fraternizing with the players and had to be discreet with the members of the press corps because I would possess confidential information about the health of individual players. The camp was akin to a small village in which information of any kind quickly spread. Although women were stereotyped in those years as the purveyors of gossip, the overwhelmingly male population of the camp regularly disproved this then conventional wisdom. There was scarcely a sprained ankle or cough that escaped comment as the day's field performances were discussed in the halls and at meal times.

The camp nurse and physician were on call 24 hours a day, seven days a week. Although this was something of a monastic schedule, I found it a pleasant change from my hospital routine and not as grim as one might imagine.

The minor league players practiced from early morning until late afternoon, after which practice games were often held under the lights in the camp's Holman Stadium. There was always a steady stream of players with minor injuries and medical problems which kept us occupied most of the time.

My professional reputation in the camp was made by luck I think within two hours of my arrival. I was still unpacking in my room when Mrs Plucker phoned to say that one of the major league players was there for his monthly steroid injection into his knee. Panic seized me as I listened to her firmly announce that she had him all set up on the examining table. I had never observed even one such procedure, much less done one. Fortunately I had taken along with me a pocket sized emergency medical guide which one of my physician uncles had given me a year before. I frantically flipped through the index and breathed a sigh of relief when I found not only a description of the procedure but a few drawings as well, clearly demonstrating technique.

Armed with this hastily acquired knowledge and the blissful arrogance of youth I walked over to the dispensary where the player, a trainer, and Mrs Plucker were waiting for me with an air of expectancy. The knee was already positioned, the tray set up, and with all eyes turned towards me I felt like a neophyte conductor about to launch the first notes of a leading symphony orchestra.

Mrs Plucker appeared a bit nervous, knowing that a great deal was riding on this single injection. She left nothing to chance and promptly told the player that not all knees were quite the same, that the good Lord made them all a bit differently. That's why it was important that he tell us, if he knew, where the orthopedist in California regularly injected the knee and at what angle. She became visibly upset when he said he didn't know. With the sketches clearly in my mind it took but one firm push to place the needle into the joint. As the solution flowed in the player sensed it was in the right place and said, "You give a great injection, Doc."

After the player left, Mrs Plucker said, with obvious satisfaction, "Now himself will be hearing about this soon enough." Soon enough turned out to be less than an hour later.

In the early 1960s, the Dodgers maintained a large number of farm teams whose composite strength was several hundred. They arrived at Dodgertown a month after the major league team had begun spring training and remained there until the middle of April. Each player had to submit a medical history and receive a physical examination and this activity kept Mrs Plucker and me busy for the first ten days. Some of the major league players also came in at this time for their annual physicals, although most had had them in California.

The first few weeks of spring training were reserved for the major league players. The barracks were relatively empty then, except for staff, since most of these players lived in town. The dining room had linen tablecloths, baseball shaped daily menu cards, and waitresses. As soon as the several hundred minor league players arrived it was transformed into a mess hall with long rows of picnic tables, and even Mr and Mrs O'Malley stood on line for counter service.

The most audible Brooklyn connection in the camp was Herman, who ran the mail room, supervised the laundry service, and began the camp's day with an expression of patriotism that pierced the thin building walls at 7 AM and negated the need for alarm clocks. After Herman raised the flag and the 1959 and 1963 blue and white World Series Championship banners, he recited the pledge of allegiance with a classic Brooklynese accent that rode on megaphonic decibels across the camp grounds. He had worked for the Brooklyn Dodgers, was retired in Brooklyn, and regularly came to spring training. By the time anyone got to the central lounge area of the principal barracks where his office was he had already read the previous day's *New York Daily News* which regularly arrived at sunrise. Some people found Herman's manner gruff but that coupled with a conversational tone befitting a town crier helped to keep high spirited ball players in check.

By 8 AM the sound of cracking bats could be heard coming from the batting cages and other practice activities began, including defense and bunting, infield and outfield exercises, running, and sliding. Several practice games between minor league teams were held on most days and on some evenings. Practice games and sliding into bases seemed to me to produce the most injuries, not because players were careless but because they wanted to excel and achieve their dreams of reaching the major leagues. I vividly recall one player who severely fractured his lower left fibula while sliding into first base. Because of the severe swelling Phil Morgan couldn't apply a cast and thus we had to keep the player in the infirmary with his leg immobilized. The morning after the injury occurred, Mrs Plucker made a startling discovery when she opened the door to his room. There he was positioned upside down on his hands and head with his swollen leg pointing to the ceiling. She never told me exactly what she said but related that he refused to put himself upright, saying he was healing his leg. Within minutes the full weight of Dodger officialdom came down on his still upturned feet. The man who set him straight was L. Fresco Thompson, the Vice President and Director of Minor League Operations. He was a no-nonsense manager who had played baseball himself for 17 years. He knew what players were capable of doing even before they had thought of it themselves. However, he had never encountered the likes of this one before and urged us to get a cast on him as soon as possible so that he could be sent back to California. The player, who practiced yoga and other uncategorized meditation exercises, sincerely believed in the therapeutic value of what he was doing and was taken aback by all the fuss. Until we put a cast on his leg several days later, he continued his exercises when he thought we weren't looking and entertained Mrs Plucker and me with his guitar music.

Minor cases of heat exhaustion regularly occurred for which Mrs Plucker concocted a salt solution of her own that seemed to work. There was a practice field behind the infirmary and the minor league players using it often came to the window for a slug of this solution. Mrs Plucker would fill up paper cups, unlatch the screen, and, leaning over the window sill, hand the cups to the players. They would gulp the solution down and grimace a bit even as she was saying, "Now it will do you a world of good." Mrs Plucker also dispensed pills, salves, bandages, and motherly advice over the windowsill and occasionally players thrust up a few wildflowers they said they had found on the edge of the field. She gave these a place of honor on her desk in an old cough medicine bottle. In this open window medical care system of hers, no names were ever taken down and no records kept and at first this troubled me. But I soon came around to her way of thinking, realizing that players used the window in the middle of practice as a quick first aid station. And as Mrs Plucker said, "They don't have time to remove and put on their cleats just to come in here and himself won't be having them come in here with those cleats on." I also discovered that she never forgot a name or face and pretty much knew what and how much she had given to an individual player.

There was one young player who regularly came to the window asking for Mrs Plucker's "salt tonic" as he called it, even though he clearly didn't need it. One day I caught up to him as he made his way back to the practice field and asked him why he was drinking so much of the solution. He looked uncomfortable and in a flash of insight I told him it did the opposite of what he thought it did. "The fellas told me it really works," he said with conviction, to which I replied, "Then why don't they drink it every day themselves?" I never told Mrs Plucker that her salt solution was being touted as an aphrodisiac.

The complex administration of Dodgertown was managed by Peter O'Malley, Mr O'Malley's son. He had to oversee housekeeping, the upkeep of the grounds, repair and maintenance, the scheduling of practice facilities, the food service, and numerous other activities including the printing of the daily schedule. This schedule was printed in Dodger blue and always ended with a quote for the day. I thought that the quote appearing on the eve of the camp's closing captured the feelings of the minor league players whose hearts were set on making it to the majors. It read: "Faith is the substance of things hoped for, the evidence of things unseen."

Dodgertown ran according to fairly strict rules. Players had to be in by 9 PM to insure a good night's sleep and maximum potential performance the next day, and no children were allowed in the barracks. Epidemic childhood diseases were still common in the United States then and many players lacked natural immunity. An epidemic of chickenpox or measles would have had a devastating impact on the team's performance. The 9 PM curfew was enforced by barring entry at all but two doors where at times various team officials posted themselves behind trees with flashlights in hand. There weren't many curfew breakers. However, one who had been out drinking was discovered as he was trying to enter. With two team officials in pursuit, he climbed up a ladder on the side of the building and then ran across the flat roof until he disappeared over the edge. A search on the ground didn't turn him up, but at 1 AM I received a call from the hospital saying that "one of your players is here with a fractured ankle." The player had pulled himself into the crawl space under the building and then gotten himself to the hospital when the coast was clear.

Fresco Thompson regularly made late evening rounds through the barracks and on several occasions I accompanied him. By that time he had been in baseball over 40 years, both as a player and manager, and had just written his memoirs, *Every Diamond Doesn't Sparkle.* The minor league players stood in awe of him and word of his approach spread a wave of silence down the block-long corridors. Once we startled a raucous group of card players in a dead end corridor. There was no escape and to their relief he let them off with only a mild rebuke. As we watched them walk off to their rooms he commented that it never ceased to amaze him how within a few hours of arrival in the camp, the drinkers and card players always teamed up.

Although rigorous practice schedules, a curfew, and middle aged women secretaries helped rivet player attention only on baseball, some players managed to get to town with regularity. A few of them were reminded of their visits several days later when symptoms appeared. "Sore throat, eh?" Mrs Plucker would say to them as she gave the injections, letting us know that she was in on the secret.

Walter O'Malley exercised no privileges in the camp and freely mingled with the staff and players. A measure of his values were the Dodger staff and his friends who came to Dodgertown for short vacations. My relationship with him was strengthened, I think, by our mutual interest in Africa where we both had been and to which we both would return. His openness and candor led many to facile summations of his character. But a fuller portrait could only be synthesized from closer quarters, from a nuance here, a word there, and steady observation of his fine values in the conduct of human affairs. The quality and competence of the Dodger management was reflected in the excellence of the team which had just won the 1963 World Series and which would go on to a fourth world title in 1965. This was a team which at the time included manager Walter Alston, pitchers Sandy Koufax and Don Drysdale, outfielders Willy Davis and Wally Moon, and infielders Nate Oliver, Bart Shirley, Maury Wills, and Wes Parker.

Three of Mr O'Malley's friends became friends of mine. That our friendships were short lived was no fault of ours. For they were in the late twilight years of their lives while I was entering mid-morning. They took me with them on their outings and tutored me in the technical nuances of baseball at exhibition games. Often they had good natured disagreements over pitches and plays and in time turned to me, their student, for opinions.

Roscoe E. McGowan had been a sportswriter for the *New York Times* for 30 years during which time he primarily covered the Brooklyn Dodgers. Although he was 78 when I first met him he was still tall, dignified in speech and manner, and could effortlessly compose verse on the spur of the moment and recite lengthy passages from Shakespeare. He was modest and warm and spoke with a basso profundo voice that was a pleasure to hear. Roscoe drove down to Vero Beach with Dr Joseph Kelly, Mrs O'Malley's New York physician. Dr Kelly was an otolaryngologist and 76 years old at the time. Although soft spoken and modest, he exhibited unexpected daring and risk in his driving which Roscoe once memorialized in verse. The troika was completed by Frank Shaughnessy, who was then 81 and one of baseball's giants. Known as "Shag," he had devised the playoff system in 1933 and had served as president of the International League. Mr Shaughnessy had been an all around athlete at Notre Dame and had gone on to become a baseball and football coach after receiving a law degree. He spoke with a bellowing voice and freely expressed his opinions with a conviction no one dared challenge, except Dr Kelly.

One of my responsibilities as camp physician was to attend exhibition games in Holman Stadium while Mrs Plucker kept watch at the infirmary. Most of the spectators were senior citizens whose slow reflexes in the face of an oncoming fly ball always kept me on alert. The four of us usually sat together and in time Roscoe suggested that I sit between Dr Kelly and Mr Shaughnessy to mediate their irreconcilable opinions on the technical aspects of plays. Mr Shaughnessy used to whisper to me that Dr Kelly was all wet while Dr Kelly's standard phrase was, "Shag is mistaken." Their most heated argument erupted during a game when Dr Kelly suggested that he could fit the pitcher with a rotating prism in his eyeglasses that would enable him to throw all sorts of balls directly across the plate. Mr Shaughnessy's reaction was immediate and terse. "Now that's the most fool thing I've ever heard of." He then whispered to me that he thought that Dr Kelly was slipping in his mental faculties. Dr Kelly promptly replied to this because Mr Shaughnessy's whispers were usually audible for several rows around. Their bantering was all done in good humor and before long they were instructing me again about baseball techniques and strategy.

Roscoe was a wonderful letter writer and we kept in touch until shortly before he died of lung cancer on November 5, 1966. Our correspondence primarily dealt with literature and writing. In a May 21, 1965, letter to me from North Woodstock, Connecticut, he wrote, " . . . About the elegies, I have read all of them excepting Shelley's Adonais . . . Having read little of Arnold I'd never heard of his Thyrsis, but I'll have to get a copy of it . . . " Earlier, on May 4, he had sent me a double quatrain on his own indisposition. It was written in the same meter as Gilbert's "A Policeman's Lot Is Not a Happy One."

> When a fellow drinks a little too much bourbon,
> When he fails to get his proper meed of sleep,
> The end results may be a bit disturbin'—
> In fact, may make his guardian angel weep.
>
> For, while he may enjoy his dissipation
> And for a time drift gaily with the stream,
> There is nothing like some "hearty" fibrillation
> To put the guy right back on virtue's beam!

A month later Roscoe wrote again, unhappy about his use of the word *meed* in the second line: " . . . that was stretching the meaning of 'meed' quite a bit," he wrote, " . . . I don't like 'meed,' so perhaps the line should be rewritten to read: 'And when he fails to get sufficient sleep or and when he fails to get his needed sleep.' "

There came a day when Roscoe's letters ceased and after a while I received one from his granddaughter telling me what I already feared. Dr Kelly and I spoke over the phone about his passing but nine months later Dr Kelly, too, was gone. The lone survivor was Mr Shaughnessy, who sent me a pair of binoculars as I left for several years in Africa. He lived until he was 86 and died in May 1969.

All three of these men enriched my life in different ways, Roscoe with his love of the classics and literature, Dr Kelly with his wisdom, and Mr Shaughnessy with his wit and knowledge of baseball. Intentional social segregation from people my own age had brought me into intense contact with three unique men whose friendship opened broad vistas beyond the world of medicine. Their shared link was Walter O'Malley and they were a reflection of him as much as he was of them.

The day finally came when the camp closed down and the staff left for either California or New York. The barracks were silent, surrounded by empty fields, and Mrs Plucker locked her window screen until the following year. The minor league players gathered out in front, waiting for buses to take them into another season of hope. These rites of spring had also been a time of hope, of success for some and disappointment for a few. Yet most dreams had not gone unfulfilled, for in the limitless horizons of youth there was always next year.

Typhlitis occurring in a myelodysplastic syndrome

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Necrotizing lesions of the bowel wall are known complications of hematologic malignancies and may occur in as many as 12% of leukemia patients.[1] Various clinical terms, such as necrotizing colitis, ileocecal syndrome, typhlitis, and neutropenic colitis, have been used to describe this process, which typically produces inflammation and necrosis of the ileal and cecal areas.[2-6] The clinical presentation of these disorders includes fever and right lower quadrant abdominal pain and may suggest acute appendicitis.[6-8]

The etiology of the ileocecal syndromes remains unclear. Known clinical features of leukemia, including neutropenia, thrombocytopenia, and the use of ablative chemotherapy, are thought to contribute to development of these processes, and much emphasis has been placed on neutropenia as a causative factor.[2-5] We report a patient with myelodysplastic syndrome (MDS) in whom necrosis of the cecum developed soon after receiving attenuated doses of chemotherapy. The association of cecal necrosis in MDS has not been previously reported, and the patient was not severely neutropenic at the time this complication developed. Although thrombocytopenia was present throughout the hospital course, there was no evidence that hemorrhage into the bowel wall developed.

CASE REPORT

A 62-year-old man was admitted to the hospital with a three-month history of fatigue and progressive dyspnea on exertion. One month prior to admission, a nonproductive cough developed, and the patient's primary care physician gave him tetracycline for three days prior to admission.

Physical examination revealed a pale, afebrile man who appeared chronically ill. The chest was clear to percussion and auscultation. Cardiac examination revealed a regular rhythm with an S4 gallop and grade 2/6 systolic ejection murmur audible along the left sternal border. The spleen was not palpable, and there was no lymphadenopathy.

The patient was markedly anemic on admission (hematocrit, 19%; hemoglobin, 2.9 g/dL), and the reticulocyte count was 1%. Morphologic abnormalities in the peripheral blood leukocytes, including numerous pseudo-Pelger-Huët cells, suggested the presence of a preleukemic or myelodysplastic syndrome. A bone marrow aspirate and biopsy performed on the second day of the patient's hospital stay revealed marked erythroid hyperplasia with an increase in myeloblasts and promyelocytes. The overall hematologic picture was most compatible with the MDS classification refractory anemia with excess blasts (RAEB).[9]

The patient received a transfusion of packed red blood cells, and chemotherapy was begun with attenuated doses of daunomycin, cytarabine and 6-thioguanine (Fig 1). He tolerated the treatment extremely well, experiencing only mild nausea on the first day of treatment. The course of chemotherapy (five days overall) was completed by day 13 of his hospital stay. At this time, the patient was doing very well clinically and remained in the hospital only so that his blood counts could be closely monitored.

On day 18 he began having temperature elevations reaching 39.5°C. Results of the physical examination were unchanged. In particular, the abdominal examination was entirely normal. Numerous blood cultures of blood and urine showed no bacterial growth. In spite of repeated vigorous investigations, no source for the patient's daily temperature elevations could be found. Broad spectrum antimicrobial therapy was begun with piperacillin, clindamycin, and tobramycin, but this had no effect on his febrile course. On day 29 of his course severe right lower quadrant abdominal pain developed, with tenderness and guarding suggestive of peritonitis, and an urgent laparotomy was performed. The surgeon found a necrotic cecum and a pericecal abscess which required ileocecal resection and colostomy. Blood cultures obtained during this time grew *Clostridium septicum*, and penicillin G was added to the treatment regimen. After surgery, severe lactic acidosis and renal failure developed requiring hemodialysis. The patient died three days after surgery.

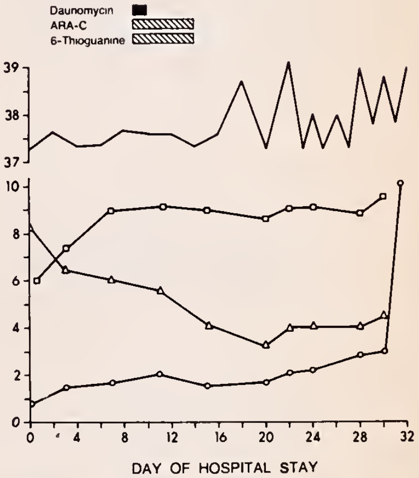

FIGURE 1. Clinical course of the patient. Open circles represent absolute neutrophil count (× 1,000). Open triangles indicate platelet count (× 10,000), and open squares indicate hemoglobin (g/dL). The continuous line at the top indicates oral temperature in degrees Centigrade. Chemotherapy administered during the first five days of the patient's hospital stay is indicated at the top of the figure.

Pathologic examination of tissues removed at surgery showed mucosal necrosis and ulceration of the cecum. The underlying wall showed marked cellulitis with infiltration of lymphocytes. The wall was markedly edematous, and several small abscesses were noted. Sections of the ascending colon showed prominent lymphocytic infiltration and one small area of hemorrhage. Hemosiderin stains of the tissue were negative.

DISCUSSION

Although inflammation and necrosis of the cecum are described in association with hematologic malignancy,[1-8] we find no other reference to such a de-

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velopment in MDS. The clinical setting in which this fatal complication developed in this patient raises some questions about the proposed pathophysiology of the ileocecal syndrome, which includes such features as neutropenia, hemorrhage, and mucositis. Neutropenia appears to be a critical predisposing factor in most cases, but this patient's absolute neutrophil count was 900 at its lowest point, and he was able to mount a brisk leukocytic response around the time of his surgery. A second common feature is intramural hemorrhage, but the negative hemosiderin stain of bowel wall sections in this case further suggests that hemorrhage did not play a substantial role here. Mucositis can certainly develop following chemotherapy, but it is much more common with higher doses of ablative chemotherapy than in the attenuated regimen used here.

This case indicates that MDS must be included among those clinical situations in which necrotizing lesions of the bowel should be considered. Although no inference of frequency with which cecal necrosis may occur in MDS can be made from this case, the clinical presentation and the fatal clinical outcome were similar to that seen in acute leukemia.

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See also pp 58–59.

Campylobacter fetus as a pulmonary pathogen in man

Case report of post-pneumonectomy empyema

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Campylobacter fetus was first isolated in 1909 by McFadyean and Stockman in Great Britian during an investigation into the etiology of abortions in sheep.[1] Its only known role as a pathogen was limited to causing abortions in domestic animals, until 1947 when Vinzent and coworkers reported a pregnant woman who aborted following an episode of *C fetus* bacteremia.[2] Despite the fact that the epidemiology of *C fetus* infections in humans is unclear and the pathogenesis of systemic infection poorly understood, this organism is becoming increasingly recognized as a significant pathogen under a variety of conditions, in addition to being a common cause of gastroenteritis in the pediatric age group. The most common manifestation of *C fetus* infection is thought to be a bacteremia without localized infection. However, despite the bacteremia, infections of the pulmonary parenchymal and pleural space are quite rare. In 1977, Targan et al[3] reported the second case in the literature of human pulmonary involvement with this organism in a patient with *C fetus* pneumonia, lung abscess, and empyema. We recently recovered *C fetus* as the causative agent in a patient in whom a post-pneumonectomy empyema developed. The slow but increasing role of this bacillus as a pathogen, recent advances in isolation techniques and bacteriologic identification, and the treatment regimen constitute the basis for this report and literature review.

CASE REPORT

A 64-year-old man was admitted to the hospital with shortness of breath, productive cough, wheezing, and occasional hemoptysis. He had been treated for the past three months by his private physician with ampicillin and bronchodilators without relief. On admission to this institution he denied fever, chills, weight loss, or night sweats. He had been treated for tuberculosis in 1952 and 1955. He also had a 40-pack/year smoking history.

Physical examination was negative except for occasional inspiratory and expiratory wheezing on the right which cleared with coughing. Chest films taken on admission showed a right hilar mass which had not been present a year before. Sputum cytology, acid-fast bacilli smears, and liver-spleen and bone scans were negative. Computed tomography of the chest showed a 7-cm hilar mass compressing the right main stem bronchus and enlarged mediastinal lymph nodes. Bronchoscopic biopsy of a mass 3 cm from the carina in the right main stem was reported as squamous metaplasia.

The patient consented to surgery and underwent a Carlen mediastinoscopy, which was negative, followed by a radical intrapericardial pneumonectomy for Stage II squamous cell carcinoma. Pathologic study revealed a single focus of tumor in the subcarinal nodes. A 12 French red Robinson catheter was left in the third intercostal space anteriorly, connected to a pneumonectomy underwater collection system, and was removed after 24 hours.

The postoperative course was uneventful, and the patient was discharged after two weeks. However, he returned 11 days later complaining of chills and a temperature of 103°F. Physical examination showed a tender, erythematous incision.

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His white blood cell count was 14,000/mm^3 with a differential count of 75 polymorphonuclear leukocytes, 15 lymphocytes, 10 monocytes, and 1 eosinophil. Thoracentesis produced serosanguinous fluid with no organisms on Gram stain. Blood and pleural cultures grew curvilinear, anaerobic gram-negative rods, which were eventually identified by the Centers for Disease Control (Atlanta, Ga) as *Campylobacter fetus*, subspecies fetus, formerly subspecies intestinalis. There was no evidence of a bronchopleural fistula.

The patient was treated initially with a cephalosporin medication but his drug regimen was changed to clindamycin after sensitivities were reported. His chest was initially drained by tube thoracostomy followed by a Clagett procedure when the former proved to be inadequate. The antibiotics were continued for four weeks. The patient remained asymptomatic for a 20-month follow-up period, with a decreasing cavity size which has not required closure.

Discussion

Campylobacter, a global zoonosis, is becoming recognized as a pathogen capable of causing a variety of diseases in adults in addition to its more traditional role as a cause of gastroenteritis in children. Reported extraintestinal, nonpulmonary sites have included thrombophlebitis, pericarditis, endocarditis, mycotic aneurysms, and septic joints, with most patients presenting with a subacute febrile bacteremia.[4–7] Although the epidemiology in humans is unclear, the organism has been identified as a normal component of mouth flora, and patients have been known to develop *C fetus* bacteremia following extraction of infected teeth.[6] Contact with infected dogs and cats has also been suggested as a source of transmission to humans, as has person-to-person spread, particularly among children.[8]

This patient informed us that on returning home following his pneumonectomy, he found all of his dogs, which he kept inside, sick with diarrhea. He did not recall whether they had been ill prior to his hospitalization. Assuming that the animals were the source of his infection, we initially postulated that postoperative contact with his animals may have resulted in the transmission of the bacillus via inhalation or ingestion, but it would be difficult to explain how the organisms colonized a clean, closed space. It is perhaps more likely that colonization occurred before surgery, and that the bacillus simply found a suitable growth environment within the pleural space after surgery. Unfortunately, none of the patient's dogs was cultured, because the diagnosis was made very late in the course of his disease.

This bacillus is a spiral or curved, motile, nonspore forming, gram-negative rod. Morphologically it has a single flagellum at one or both ends that is particularly difficult to visualize and which seldom plays a meaningful role in making the diagnosis. Figure 1 demonstrates the typical spiral or curved organism commonly seen during the early growth phase. As the organism matures, it may assume a straight rod-like shape as shown in Figure 2. It grows well on blood agar, Albimi-Brucella agar, and under reduced oxygen tension (5–6% oxygen concentration), and is considered to be microaerophilic.[9] The organism requires special isolation techniques because its small size and motility allow it to pass through standard 0.65 mm filters. This may explain why a post-pneumonectomy hemithorax might provide an environment with all of the basic requirements necessary to promote the growth of *C fetus*. In the immediate postoperative period, there is a small amount of blood in the chest and a low oxygen concentration following chest closure. The use of the red Robinson catheter probably added to the ambient oxygen concentration within the hemithorax for perhaps 48–72 hours. After pneumonectomy, the hemithorax begins to fill very quickly with serum. We suggest that this early combination of blood, serum, and low oxygen concentration perhaps provided an environment suitable to initiate and sustain a microaerophilic infection. *C fetus* has no known cytotoxicity and does not produce enterotoxins, although the structure of its cell wall is similar to other gram-negative rods.

The diagnosis of *C fetus* can be difficult to make unless selective techniques are used. However, the recovery of a motile, spiral or curved, gram-negative rod from a location likely to have experienced a low but perceptible oxygen concentration should raise the level of suspicion necessary to initiate special testing for this organism. Gentamicin is considered the antibiotic of choice. Adjuvant treatment should also include surgical drainage or excision as appropriate.

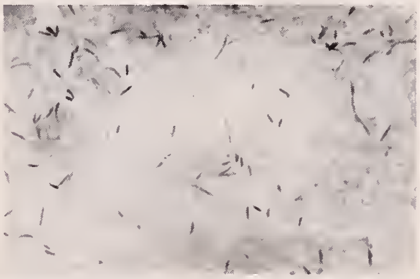
FIGURE 1. Photomicrograph of Gram stain of *C fetus* colony during early growth phase showing characteristic spiral or curved organisms (original magnification × 1,000).

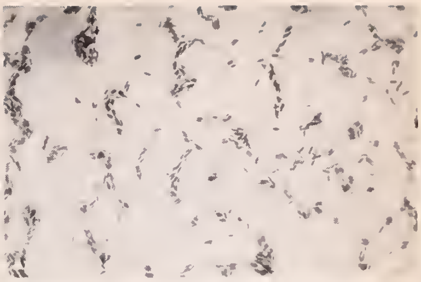
FIGURE 2. Photomicrograph of Gram stain during late growth period demonstrating a more straight, rod-like shape (original magnification × 1,000).

C fetus is becoming increasingly recognized as a pathogen in adults, causing diseases other than diarrhea. This organism may be a component of normal mouth flora in some patients, especially those with severe periodontal disease. It may also be acquired by patients exposed to dogs, cats, sheep, or chickens either as pets or as occupational hazards. Because of the indolent nature of an established *C fetus* infection, gentamicin or another antibiotic suggested by sensitivity studies should be continued for up to four weeks in conjunction with appropriate surgical debridement in order to prevent recurrences.

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Enterococcal meningitis: A subacute presentation

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Enterococcal infections involving the central nervous system are unusual.[1-3] Enterococcal meningitis usually occurs as a consequence of some other primary illness of long duration, surgery involving the central nervous system, or long courses of multiple antibiotics for other infections. The typical presentation of enterococcal meningitis is rapid onset of fever, signs of meningeal irritation, and altered sensorium. In this report, we describe a patient with acute lymphocytic leukemia in whom enterococcal meningitis developed and presented predominantly with fever, but few other symptoms or signs of central nervous system infection. Two cerebrospinal fluid specimens obtained 24 hours apart contained only one and two colonies of enterococci in the absence of effective antibiotic therapy. Enterococcal meningitis may present subacutely in patients with underlying immunosuppressive disease.

CASE REPORT

A 47-year-old man was admitted to Strong Memorial Hospital with spontaneous bruising and bleeding gums. Acute lymphocytic leukemia, diagnosed 18 months before admission, had been treated with vincristine, prednisone, daunomycin, cyclophosphamide, adriamycin, methotrexate, high-dose cytosine arabinoside, and L-asparaginase. Complete remission was documented seven months before admission.

One week before admission, spontaneous bleeding of the gums occurred, and several large ecchymoses spontaneously developed on the patient's forearms. A peripheral blood smear and bone marrow examination revealed relapsing leukemia. Treatment with aclacinomycin and etoposide was given, resulting in neutropenia on the seventh day of admission. Fever and hypotension occurred; piperacillin and tobramycin were started on the eighth day after admission. Blood cultures grew coagulase-negative staphylococci on the 12th day after admission and vancomycin was administered. On the 20th day of hospitalization, because of persistent fever, treatment was changed to cefotaxime, tobramycin, and amphotericin B. The patient's condition improved, and antibiotics were continued.

Additional chemotherapy included high dose methotrexate, vincristine, and prednisone. Repeat marrow examinations demonstrated persistent leukemia. During chemotherapy, the patient was intermittently febrile to 38.5°C.

On the 44th hospital day, the patient became febrile to 39.4°C while receiving cefotaxime, tobramycin, and amphotericin B. Blood cultures grew enterococci. Tobramycin and amphotericin B were stopped; vancomycin was given for ten days; gentamicin for 14 days. Cefotaxime was continued, and intermittent fever persisted.

On the 52nd day after admission, mild frontal heachache developed, and the patient's temperature was 38.6°C. He was receiving cefotaxime, 2 g every six hours. No other specific therapy was given, and his condition improved. Mild headache and photophobia occurred again on the 54th day. Mild neck soreness, without signs of meningeal irritation, developed on the 56th day. A lumbar puncture was performed. The level of glucose in the cerebrospinal fluid (CSF) was 16 mg/dL; total protein, 106 mg/dL; and cell count, 250 nucleated cells per mm^3 with 92% segmented cells, and less than 10 red blood cells per mm^3. No organisms were seen on Gram stain of the CSF, and cryptococcal antigen was not detected. Blood cultures were negative. Vancomycin, gentamicin, isoniazid, rifampin, and ethambutol were added to the regimen of cefotaxime. Mild confusion and headache persisted, and fluid obtained from a repeat lumbar puncture showed similar laboratory results. The patient's condition gradually improved, and he became afebrile after three days of treatment. Cerebrospinal fluid cultures grew a single colony of enterococcus from the first lumbar puncture and two colonies from the second. The patient was then given ampicillin and gentamicin intravenously. Evaluation for sources of antecedent enterococcal bacteremia and enterococcal meningitis included an echocardiogram, abdominal ultrasound, gallium scan, and a contrast study of the Hickman catheter. All were negative. He was discharged on the 69th day after admission, and five remaining days of a 14-day course of ampicillin and gentamicin were administered at home. Bone marrow transplantation was successfully performed three months later. Enterococcal infection did not recur.

DISCUSSION

Enterococci include *Streptococcus faecalis*, *S faecium*, and *S durans*, and are distinguished from nonenterococcal group D streptococci (*S bovis* and *S equinus*) by their ability to grow in broth containing 6.5% sodium chloride. *S faecalis* is the species responsible for most enterococcal human disease. Most laboratories report these organisms simply as enterococci and do not speciate them.

Enterococci are part of the normal human flora of the mouth, intestines, and colon. They are responsible for 10–20% of cases of bacterial endocarditis and produce disease on normal, abnormal, and prosthetic valves.[4] Urinary tract infections in patients with indwelling urinary catheters are frequently associated with enterococci.[5] These organisms are often present in wounds and soft tissue infections. Enterococci are the most frequently identified gram-positive organism in polymicrobial bacteremia,[6] reflecting their association with infection in the gastrointestinal tract, genitourinary tract, and soft tissues.

The treatment of enterococcal illness is difficult. The distinction between enterococci and nonenterococcal group D streptococci is important when considering antibiotic therapy. Cephalosporins are ineffective in vivo, and penicillin, ampicillin, and vancomycin are bacteriostatic against enterococci.[7,8] Addition of an aminoglycoside to a penicillin produces synergy against the organism, probably due to increased aminoglycoside uptake resulting from

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penicillin-induced damage.[9-11] Nonenterococcal group D streptococci are sensitive to penicillin, ampicillin, and many other antibiotics.

Recent reports indicate that enterococci are becoming increasingly more common as causative agents of nosocomial infections in patients who receive broad spectrum, "third generation" cephalosporins. Yu[12] noted four cases of enterococcal superinfection and five cases of colonization in 41 patients treated with moxalactam. All were compromised hosts, had multiple vascular and bladder catheterizations, and had prolonged hospital stays. Ribner et al[13] treated 93 patients with moxalactam, and in 10% of these patients, enterococcal superinfections developed. Maslow and colleagues[14] treated 18 patients with ceftriaxone; two cases of enterococcal superinfection developed. Ramphal et al[15] treated 21 febrile granulocytopenic patients with ceftazidime; in three of these, enterococcal superinfection developed. A common factor in these patients seems to be long duration of therapy and underlying illness.

Enterococcal meningitis is uncommon and accounts for less than 1% of cases in most series of bacterial meningitis. In well documented cases of enterococcal meningitis, patients have presented with fever, signs of meningeal irritation, mental status changes ranging from confusion to coma, and a brief time interval from onset of symptoms to diagnosis, usually one to two days. The patient presented here demonstrated gradual onset of symptoms that developed five days before the initial lumbar puncture was performed. In addition, he initially had no signs of meningeal irritation and a normal mental status. Lumbar puncture was done because of persistent headache and fever. An acute bacterial meningitis was not strongly suspected because other signs and symptoms of central nervous system infection were not present.

The development of enterococcal meningitis in this patient may have occurred as a result of enterococcal bacteremia, which occurred two weeks earlier. Whether an asymptomatic central nervous system infection was established at that time, or central nervous system seeding occurred closer in time to the development of mild symptoms cannot be determined. Negative blood cultures at the time when CSF specimens grew enterococci suggest that CNS seeding immediately prior to the development of meningitis was unlikely. Subsequent evaluation for a source of enterococcal bacteremia was negative. The patient had been treated with several courses of broad-spectrum antibiotics, including cefotaxime.

Small numbers of enterococci were isolated from CSF. A single colony of enterococcus grew from CSF obtained at the first lumbar puncture, and two colonies grew from a second specimen obtained 24 hours later, while the patient was receiving cefotaxime. While other reports of enterococcal meningitis have not stated numbers of colonies of enterococci isolated, more commonly, cultures of CSF during meningitis yield higher colony counts. A subset of patients with enterococcal endocarditis have a subacute presentation, and this patient may represent an analogous situation with meningitis.

Enterococcal meningitis may present as a subacute illness with low numbers of organisms isolated from cerebrospinal fluid. In patients who are immunocompromised, have severe underlying diseases, or who have been treated with long courses of broad spectrum antibiotics, particularly third generation cephalosporins, enterococcal superinfection must be suspected. Enterococci should be considered as a cause of meningitis in these patients.

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Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Temporary insanity and premenstrual syndrome

TO THE EDITOR: The recent scholarly article by Dr Allen Spiegel[1] about the temporary insanity defense invites comment.

Why did Dr Spiegel give special attention to premenstrual syndrome (PMS)? Nothing in Mary Harris' story suggested that she had PMS other than the fact that she was a woman and her symptoms were *retrospectively* reported to exacerbate at menses. *Prospective* ratings are required to diagnose PMS.[2]

In contrast to carefully researched material on Mary Harris, Dr Spiegel's references on PMS were from the popular press, not peer-reviewed journals. Furthermore, what he wrote was misleading. For example, PMS was not renamed to avoid perpetrating myths. Rather, a subgroup of women with PMS was delineated to establish a valid research category. In addition, even though the statement that "Mary Harris was also mentioned in a Canadian perspective of the legal implications of premenstrual syndrome" is technically true, Dr Spiegel neglected to state that Mary Harris was cited as an example of legal ramifications of the 19th century view that menstruation was a disease.

Late Luteal Phase Dysphoric Disorder (LLPDD) is the diagnosis in the revised *Diagnostic and Statistical Manual of the American Psychiatric Association* (DSM-III-R) Appendix A for diagnoses deserving further research. LLPDD and PMS are not synonymous. Women with both diagnoses may experience similar symptoms. However, women with LLPDD are so severely impaired that they meet criteria for a mental disorder. Premenstrually, they may be depressed and suicidal, angry and fearful of harming someone, or so cognitively impaired that they appear schizophrenic. However, unlike Mary Harris, their symptoms remit with menses. In LLPDD as with PMS, physical symptoms (such as the pain described for Mary Harris) may be present, but unlike PMS, the physical symptoms are insufficient to confirm a diagnosis of LLPDD.

Using PMS as a defense is not straightforward. Lack of consensus about definition of the condition, lack of a diagnostic test for PMS, and lack of agreement about causes or treatment contribute to the dilemma. No conclusive evidence indicates that women with PMS make more suicide attempts,[3] experience more psychiatric emergencies,[4] become more impaired cognitively,[5] or commit more violent crimes[6] during the premenstruum than women without PMS. Nor does conclusive evidence indicate that they do not.

PMS is not recognized as a substantive defense. One cannot claim that PMS is a mental disease or that a person suffering from PMS totally lacks the capacity to understand her actions. Courts seem to be growing more sympathetic to arguments that a woman with PMS may suffer from periodic diminished capacity to control her behavior.[7] PMS may then be considered a mitigating circumstance or a factor to be considered in sentencing.

In summary, Dr Spiegel's article links PMS erroneously with other psychiatric disorders that exacerbate periodically and does not delineate PMS as a distinct entity. We suggest he retitle his article "Temporary insanity and periodic catatonia" or "Temporary insanity and periodic mania" and leave PMS out.

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In reply. I am indebted to Drs Severino and Moline for supplying me with additional information relative to my article on temporary insanity and premenstrual syndrome (PMS) in an 1865 murder trial.[1] Their contribution reaffirms the fact that there is still considerable room for discussion regarding PMS and its after-effects.

Since the article was based on historical research, it is appropriate to go directly to the source. In this case, Charles Henry Nichols, MD, was the only court-recognized medical expert at the trial. In 1865, Dr Nichols was the Superintendent of the Government Hospital for the Insane and had been so since 1852. He had been in medical practice for 22 years with a specialty of the mind for the previous 18 years.

In the trial report,[2(p73)] Dr Nichols noted that he saw Mary Harris five times in her jail cell. At an early visit, Harris told Dr Nichols that she was unwell and was experiencing her monthly period. Dr Nichols noted that "she then exhibited more nervous agitation than I observed at the previous interviews."[2(p76)] Earlier, Dr Nichols commented that "It does not occur to me how I came possessed with the idea that her mental disturbance was connected with her menstrual periods."[2(p74)] He says that he corresponded with Harris' Chicago physician, Dr Calvin M. Fitch, whose diagnosis was "severe congestive dysmenorrhea." However, Dr Nichols "corroborated [his diagnosis] by what I observed myself of her condition."[2(p75)] On questioning, Dr Nichols stated that painful dysmenorrhea "is a frequent cause of mental disturbance."[2(p74)] On cross examination, Dr Nichols told the court, "but my belief is that paroxysms were more likely to appear at the monthly periods."[2(p80)] Dr Nichols, the only expert medical witness at the trial, concluded that the combination of a physical ailment—painful dysmenorrhea—and a moral cause—disappointment in love—resulted in Mary Harris' diagnosis of paroxysmal mania.[2(p74)]

In reading the trial transcript, there can be no doubt of the direct cause and effect relationship espoused by Dr Nichols. That medical expert's diagnosis resulted not only from his own prospective observations of Mary Harris in her jail cell but also from the reported observations of Louisa and Jane Devlin with whom Harris lived and worked in Chicago, from Dr Fitch's medical examinations of Harris when she was sick, from Comptroller Hugh McCullough's first-hand observations immediately after Harris shot and killed Burroughs, and from chief counsel Joseph H. Bradley's direct observations on his visits to Harris in her jail cell. Of course, Dr Nichols' diagnosis was confirmed by his prospective observations as Harris was confined to the Government Hospital for the Insane during the ensuing years while he was still the superintendent.

In 1867, Dr Ralph L. Parsons, Superintendent of the New York City Lunatic Asylum, reviewed the Mary Harris case and agreed with Dr Nichols. "Her menstrual functions became deranged so she suffered severely from dysmenorrhea."[3(p194)] "Her sufferings from dysmenorrhea were so great as to cause much nervous irritability and wildness of look."[3(p194)] "The physical and mental phenomena urged in the defense as insanity became gradually more marked and more constant until it was found necessary to confine her within the walls of an Asylum for the Insane."[3(p212)]

In 1872, Fanny Hyde shot and killed George W. Watson in Brooklyn, New York. Her defense lawyers often cited the Mary Harris case and the connection between temporary insanity and dysmenorrhea.[4] Samuel D. Morris, one of Hyde's defense attorneys, told the court that "It is trifling in nature and yet [medical] writers on this subject designate this [dysmenorrhea] as one of the fruitful sources of temporary insanity."[4(p41)] An expert medical witness, Dr John Byrne, Clinical Professor of Uterine Diseases at Long Island Medical College, testified that dysmenorrhea is a frequent cause of mental disturbance and that that opinion is sustained by medical writers.[4(p72)] Another medical expert for the defense, Dr Charles Correy, from the New York City Bloomingdale Asylum for the Insane and formerly with the New York City Lunatic Asylum, agreed as he reviewed the works of numerous physicians on the subject of menstruation and its effects.[4(p74)] Incidentally, the count stood at ten for acquittal and two for manslaughter when the judge dismissed the hung jury.[4(p161)] Fanny Hyde went free since the case was never retried.

In his article on "Psychofashion," Williams quotes from an interview with Dr Robert Spitzer, who was the head of the American Psychiatric Association's work group revising the DSM-III. "The chief advantage of the new name, according to Spitzer, was again a matter of current fashion . . . It doesn't invoke images of menstruation."[5]

Even though d'Orban's research[6] does advise looking for behavioral rather than subjective changes, her summary facts vividly may lead readers to a cause and effect conclusion. "Of 50 women charged with crimes of violence, 44 percent committed their offense during the paramenstruum [$P < 0.02$] and there was a significant lack of offenses during the ovulatory and postovulatory phases of the menstrual cycle [$P < 0.01$]."

It is true that Meehan and MacRae[7] cite five cases and comment that "PMS has been recognized as a mitigating factor in sentencing in three English decisions." But, the authors also conclude saying, "This is not to say that PMS is, strictly speaking, a substantive criminal defense in law, but the practical result is the same—acquittal."

Today's facts appear to agree with Drs Severino and Moline that "Using PMS as a defense is not straightforward." However, historical data unequivocally bolster the expert medical view in 1865 of a connection between dysmenorrhea and temporary insanity and affirm the title of my article.

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Empty lifeboat

TO THE EDITOR: I should like to congratulate you on publishing the commentary, "The empty lifeboat," by Dr Gary Crum in the October 1988 issue.[1] Many of us feel uneasy with recent pronouncements on withholding food and water from terminally ill patients and with the present subtle but increasing emphasis in the media on euthanasia. Could it be that public opinion is being primed to coincide with the predicted upswing in the number of elderly patients at the end of the century?

Dr Crum's article is intelligent, compassionate, and above all, timely.

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1. Crum G: The empty lifeboat. *NY State J Med* 1988; 88:520–521.

TO THE EDITOR: Dr Gary Crum's commentary, "The empty lifeboat,"[1] deals with a number of important concerns, including euthanasia. These moral dilemmas will become more important as our population ages.

Unfortunately, the article deals with the problems in a one-sided manner. Dr Crum would never consider any form of euthanasia, but he does not mention at all patients' wishes or desires.

It is often more compassionate to listen to the patient rather than to subject an individual to all our modern, often aggressive, modes of maintaining vital signs for a few more days or weeks.

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1. Crum G: The empty lifeboat. *NY State J Med* 1988; 88:520–521.

In reply. I agree with Dr Nayer—it is important to listen closely to the patient, and it is also important that the competent patient's legal and moral right to refuse treatment be respected.

My position is that we must have deep sympathy for the terminally ill patient who voluntarily refuses extraordinary treatment, and for the physician who in such cases ceases heroic efforts while still hoping for the unexpected healing to take place. However, I personally cannot agree with any physician who adopts death as his or her primary "treatment" goal for a patient, even if this desire is carried out only by acts of omission. Furthermore, I cannot agree with those like Dr Daniel Callahan of the Hastings Center[1] who currently seek to ration life saving acute care services as a matter of social policy.

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1. Callahan D: *Setting Limits*. New York, Simon and Schuster, 1987.

Adult day care centers for elderly patients

TO THE EDITOR: I want to underscore the sentiments expressed by Doctors Wolf-Klein, Maar, and Foley in their excellent article in the October 1988 issue of the *Journal* on the growing significance of adult day care centers.[1]

It is especially important to recognize the psychological impact of such centers on the caretakers of elderly patients. The daily relief afforded these individuals by the center personnel is essential to their maintaining their equanimity over the long term in dealing with their elderly charges. Physicians involved in geriatric care should be aware of this important therapeutic modality and should seriously consider supporting federal legislation that would authorize payment for adult day care under Medicare.

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1. Wolf-Klein GP, Maar VK, Foley CJ: Adult day care—a recent past and a growing future. *NY State J Med* 1988; 88:528-530.

ERRATUM

The article by Conetta et al in the December 1988 issue, "Cavitation as an unusual roentgenographic manifestation of *Pneumocystis carinii* pneumonia in a patient with acquired immunodeficiency syndrome," contained an illustration error. The correct illustration and caption for Figure 3 are reproduced below.

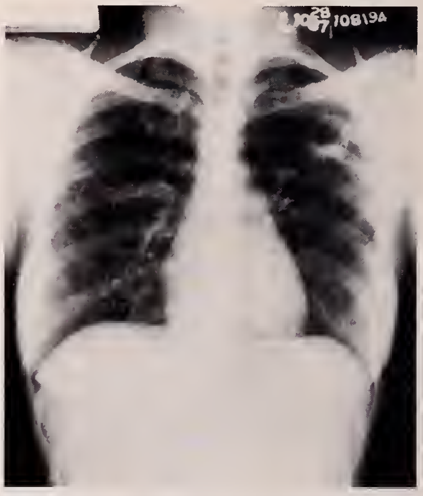

FIGURE 3. Last chest roentgenogram demonstrating resolution of the interstitial infiltrates and a reduction in size of the cavities.

Life and the law

Life-Sustaining Treatment: Making Decisions and Appointing a Health Care Agent. New York, New York State Task Force on Life and the Law, 1987. $8.50 (softcover)

Transplantation in New York State: The Procurement and Distribution of Organs. New York, New York State Task Force on Life and the Law. 1988. $8.00 (softcover)

Surrogate Parenting: Analysis and Recommendations for Public Policy. New York, New York State Task Force on Life and the Law, 1988. $7.00 (softcover)

Since the work of the Commission for the Protection of Human Subjects of Biomedical and Behavioral Research in the mid-1970s, panels of informed citizens chartered by government authorities have played a substantial role in developing social and ethical policies for health care. The President's Commission for the Study of Ethical Problems in Medicine and Biomedical and Behavioral Research produced 11 influential studies during the Carter and Reagan administrations, and the Ethics Advisory Board of the Department of Health and Human Services is about to be resurrected after eight dormant years. A special National Institutes of Health panel was recently convened to address the ethical implications of the use of fetal tissues, and a congressional bioethics board seems to be finding its feet. But below the federal level, the Empire State has taken up this model with unmatched enthusiasm.

The New York State Task Force on Life and the Law was convened by Governor Cuomo in March 1985 to develop public policy recommendations on a number of issues arising from recent medical advances. Seven reports have been published by the 25-member panel so far; its 1986 volume on do-not-resuscitate orders has shaped the subsequent law.[1] Thus it may be expected that this body will continue to have a powerful influence on New York's response to the ethical and social challenges of modern medical technology.

The task force report on life-sustaining treatment proposes legislation for the creation of a "health care proxy," a device that would make it possible to designate a health care agent empowered to make all treatment decisions if the principal becomes incompetent to do so. Several other states have such laws, and the task force notes several features of the New York environment that create a "compelling need" for such an arrangement. For example, it is unclear what authority family members have concerning decisions about treatment of noncompetent relatives. Confusion about legitimate decision making authority compromises both the patient's right to have his or her wishes respected and the physician's ability to introduce therapy in the most efficient and timely manner.

New York law has indeed set a relatively strict standard of clear and convincing evidence of an incapacitated patient's wish to refuse therapy, especially concerning decisions to withdraw life sustaining treatment. The recent Court of Appeals decision in the case of Mary O'Connor has, if anything, moved New York further away from most other jurisdictions in this regard.[2] For while several states have accepted relatives' accounts of patient wishes not to be kept alive through artificial means, the New York Court in *O'Connor* required specific reference to the kind of treatment to be forgone in a patient's historic statements, which in this case was artificial hydration and nutrition. Critics of the decision have predicted that it will result in increased resort to the courts when life sustaining therapies are in question.[3] Thus it is reasonable to suppose that the health care agent bill, which was not adopted by the legislature in its last session, will excite more attention in the aftermath of *O'Connor*.

As conceptualized by the task force, the health care agent is to use a two-level standard of decision making. First, the agent's decision should reflect the decision that the principal would have made if he or she were capable of doing so. Often called "substituted judgment," the agent's decision should be guided by any written or oral statements the principal might have made about his or her wishes, as well as his or her religious or moral beliefs. Second, if those wishes are unknown, the decision should be made according to the principal's best interests. The agent's decisions are to have priority over decisions by all others, except for those expressed by the principal either verbally or in writing, as may be done in the health care proxy document itself.

The task force proposal also contains provisions for determining lack of capacity so that the agent can be empowered. A written determination by the attending physician must be included in the medical chart that specifies the cause, nature, extent, and probable duration of the incapacity. If the lack of capacity is thought to be the result of a mental illness, then the attending physician must be, or must consult with, a health care professional who is board certified or eligible for certification in psychiatry and neurology. If the incapacity stems from a developmental disability, then an analogously qualified consultant must be involved in the determination.

What is significant about the proxy concept is the strength of the authority granted the agent. By contrast, the task force could have chosen a weaker form of representation, often called surrogacy. Surrogates are to be governed by their calculation of a patient's best interests, and may be chosen by legal authorities rather than by the principal. Thus the surrogate's preference is thought properly to be limited by the physician's evaluation of those interventions that offer a reasonable chance of medical benefits to the patient.

Evidently the task force could find no good reason not to enable competent adult patients to exercise complete control over their therapeutic options, or to do so through representatives they chose themselves. Thus, although a physician may disagree with both an agent and a surrogate about what is best for the patient, only the agent has necessarily been identified by the principal as his or her preferred decision maker. The underlying argument is straightforward: If there is a moral obligation to respect a competent adult's choices about medical treatment, then that person's selection of an alternative health care decision maker falls within that ambit.

Following the lead of the president's commission, the task force has elected to pursue a procedural solution.[4] That is, it has accepted that the answer to the question "Who decides?" is "the patient," and it has gone a bit further to clarify that the answer to the question "Who decides if the patient cannot?" is also "the patient." It has not attempted to establish a substantive canon of ethical and unethical practices, a role traditionally assigned to the professions themselves. In a pluralistic society that must reckon with persistent novelty in medical technology this is a wise course.

Put this way, the justification for the health care agent may seem arid and formal, but there is a social psychologic rationale that gives it content: the intrinsic significance of an individual's choice of an agent. That is, one can assume that a certain agent was chosen for a particular reason. The reason could have to do with some desirable feature of the agent's personal character or with the relationship that the principal has with the agent. Of course, the task force recomends that any individual contemplating the appointment of an agent carefully set out his or her preferences, and it notes that the principal will have powerful motivation to do so.

Occasional disagreements between physicians and agents about the patient's wishes or best interests would be inevitable, and it is noteworthy that the task force does not include an appeal process in its proposal. Clearly the existence of a formalized appeal mechanism would strike at the heart of the health care proxy concept, for under this system the agent's decisions must be presumed to be as definitive as those the patient himself would make. Very rarely a disagreement between a physician and an agent may come before a court of law, but not nearly as often as the *O'Connor* decision currently appears to invite. Finally, it should be mentioned that, under prevailing New York law, disagreements about non-resuscitation orders may be taken to a dispute mediation system prior to seeking legal remedy. Presumably this would constrain a health care agent's decisions about resuscitation.

The health care proxy proposal offers an important corrective to a troubling situation in New York. *O'Connor* could well encourage pervasive skepticism among physicians about the validity of nearly all prior statements about treatment decisions, except for those few that are documented and apply precisely to the current circumstances. In the vast majority of cases a health care agent can be expected both to preserve the wishes of the patient and to avoid the possibility that New York's physicians will feel the need to set up camp in the state's courts of law.

Apart from technical issues of surgery and medication, perhaps the most important challenge in organ transplantation is the achievement and maintenance of public trust. People must be confident that organs are appropriately and sensitively procured, that they will be used whenever they can be of benefit, and that they will be distributed in an equitable manner. The task force proposals on transplantation, as laid out in *Transplantation in New York State*, suggest policies and administrative structures that bear upon each of these expectations. In this report their goal is to enhance cooperation rather than competition for organs.

The major systematic reform would be the establishment of no more than three organ procurement agencies (OPAs), one for the New York City metropolitan area, one for Northeastern New York, and one for Western New York, replacing the current patchwork of a half dozen OPAs now serving the state. Each OPA would draw up a waiting list of potential kidney recipients in its region and make selections according to national policies of need and waiting time, instead of the current intra-institutional distribution. Hearts, livers, and other extrarenal organs would go to patients on a single statewide waiting list.

Another significant innovation would be a multidisciplinary transplant council appointed by the governor and composed of experts in fields relevant to transplantation as well as members of the general public and transplant recipients. The council would recommend policies concerning organ procurement and distribution, testing procedures for matching kidney donors, and a system to facilitate organ sharing.

A chapter on recipient selection criteria wrestles with the relative roles of medical and psychosocial criteria in distributing these scarce resources. While the task force concludes that medical criteria should be the "ethical foundation" for recipient selection, it allows that psychosocial criteria may also be applied when they are not used to screen out certain populations, such as the poor or less educated. Rather, the criteria must focus on patient outcomes, including the ability to cope with major surgery and accept a drug regimen. Family structure, one of the standard reference points for transplant centers, too easily coincides with unacceptable criteria like race and is at best highly suspect.

The key recommendation concerning surrogate motherhood, contained in *Surrogate Parenting*, is that legislation should declare such contracts void as against public policy. In support of this conclusion the task force arrays several concerns about the consequences of public acceptance of such arrangements. First, society has an interest in protecting the best interests of children and in shielding reproduction from commercial interests. Second, the payment of fees in return for relinquishing a newborn is not in children's best interests and could undermine the dignity of women, children, and reproduction. Third, these arrangements threaten the long recognized parental right to a relationship with one's own child.

Thus the task force proposes that no fees for surrogates or brokers for surrogates be permitted in the state. Surrogacy that did not involve fees but only payment for reasonable expenses associated with pregnancy and childbirth would not be barred. In the event of a dispute custody should be awarded to the birth mother unless a court finds clear and convincing evidence that it would be in the child's best interests to award custody to the father and/or genetic mother. Drafts of legislation in Albany have somewhat modified this latter provision.

The task force's position on surrogate parenting is rapidly becoming the predominant view. The most serious counterargument appears to stem from an argument about constitutional rights to privacy, particularly in the area of reproduction. According to this view, a society that grants married couples coital freedom should include the right to create contracts around gestational matters, and it is reasonable to expect these contracts to be enforced by the state. In response, the task force found that surrogate parenting arrangements are not constitutionally protected because they involve commercial and contractual decisions that set the rights of several individuals in conflict. The forced surrender of a child, the use of another person's body over an extended period, and the market character of surrogacy for profit are among the elements that take it beyond the domain of constitutionally protected private acts, according to the task force. The panel's analysis of a topic that has generated more heat than light is refreshingly sensible and admirably comprehensive. Indeed, similar remarks apply to all three volumes. The taxpayers of New York have been well served by this task force.

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2. *Matter of O'Connor*, 57 USLW 2241 (NY Ct App Oct 14, 1988) (No. 312).
3. Annas GJ: Precatory prediction and mindless mimicry: The case of Mary O'Connor. *Hastings Cent Rep* 1988; 18(6):31–33.
4. President's Commission for the Study of Ethical Problems in Medicine and Biomedical and Behavioral Research: *Making Health Care Decisions*. Washington, DC, 1982.

AN APPRECIATION

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Hysterectomies in New York State: 1984–1986

Hysterectomies are one of the most frequently performed surgical procedures for women in the United States. In 1985, approximately 670,000 hysterectomies were performed in short stay, nonfederal hospitals. This was the second most frequent major surgical procedure for females, surpassed only by cesarean deliveries. Hysterectomies ranked as the third most common surgical procedure performed. In 1985, hysterectomies were ranked as the sixth most common procedure performed, whereas nationally they ranked third. The overall rate for hysterectomies in New York State also varied from that observed nationally. The overall rate for hysterectomies in the state in 1984 was 4.3 per 1,000 women aged 15 and over, as compared to the national rate of 6.9 per 1,000. This follows the trend of lower hysterectomy rates observed in the northeastern United States as compared to other sections of the country.[1]

The following is an analysis of data from the Statewide Planning and Research Cooperative System (SPARCS) during 1984–1986. During this time, a total of 93,152 hysterectomies were performed in the state.

Geographic Variation

According to SPARCS data, hysterectomy rates vary considerably with geographic location. The New York City, Long Island, and the lower Hudson River valley areas had the lowest hysterectomy rates in the state. High hysterectomy rates were observed for many of the counties adjacent to the St Lawrence River, Lake Erie, and Lake Ontario. Since hysterectomy rates vary considerably among age groups, the differences in the age distribution of the female population can affect the comparison of overall rates. In order to examine whether age plays a role in this difference, hysterectomy rates were adjusted using the direct method of standardization. This method applies the age specific rate in each county to the standard population. The adjusted rate for each county is the hysterectomy rate that county would have experienced if the age structure were that of the state. Figure 1 represents the age-adjusted hysterectomy rates by county. This adjustment for age did not significantly alter the overall geographic variation observed for the crude rates. Thus, the geographic differences in hysterectomy rates were not accounted for by varying female age structures within counties.

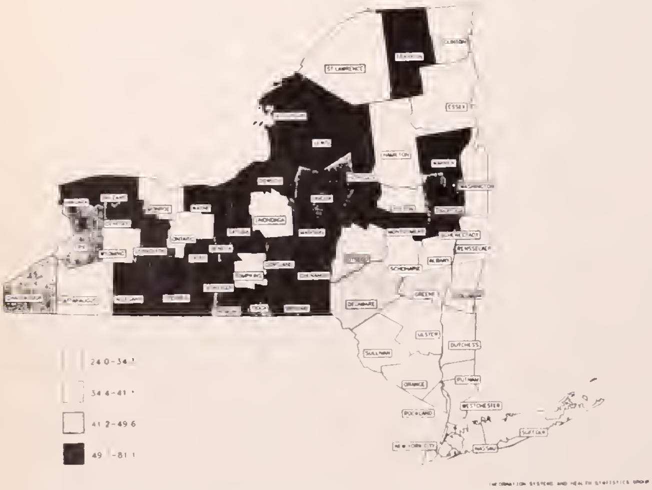

FIGURE 1. Hysterectomies in New York State 1984–1986: Age-adjusted hysterectomy rates per 10,000 female population.

Age and Race Characteristics

The average age of women who had a hysterectomy in New York State was 47.4 years. Over 48% of all hysterectomies were performed on women ages 15–44. Women 45–49 had the highest rate of hysterectomies, 119 per 10,000 females, followed by the 40–44 group at 114 per 10,000 females. The age groups also showed rate variations in the type of hysterectomy performed. Figure 2 represents rates for nonradical hysterectomy by age group. The rate for abdominal hysterectomies performed increased up to the 45–49 age group and decreased steadily thereafter. The rates for vaginal hysterectomies showed a more constant trend over the various age groups.

The vaginal nonradical hysterectomy procedure was performed much less frequently for black females as opposed to white females. The proportion of all vaginal procedures was 9% in black females compared to 20% in white females.

Associated Diagnosis and Procedures

Hysterectomies are performed for a variety of reasons and are associated with other procedures during a hospital

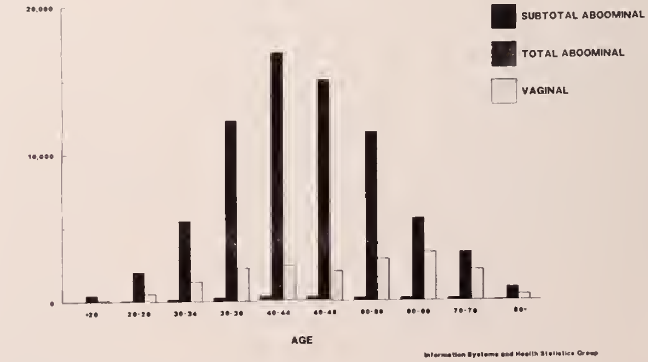

FIGURE 2. Frequency of hysterectomies in New York State, 1984–1986: type of hysterectomy by age.

stay. For instance, hysterectomies defined as either pregnancy related, cancer related or other are proposed by Wingo et al.[2] For all hysterectomies, approximately 87% were classified as other, with those related to cancer and pregnancy at 12% and 1% respectively.

The most common associated procedure for abdominal hysterectomies was bilateral salpingo-oophorectomy—the removal of both ovaries and fallopian tubes at the time of hysterectomy. In subtotal abdominal hysterectomies, this procedure was performed 59% of the time and in total abdominal procedures 61% of the time. For vaginal hysterectomies, the most common procedure reported was repair to the cystocele and rectocele (71%). Age also played an important role in differentiating the distribution of associated procedures. For example, in women 45 and older, the removal of the ovaries was more common. Among vaginal procedures, the probability of a repair of a cystocele or rectocele increased with increasing age. In women under 45 years, lysis of the peritoneal tissue was more commonly associated with hysterectomies.

The principal diagnosis entered on the hospital discharge record should indicate the reason for performing the hysterectomy. The most common diagnosis for both total and subtotal abdominal hysterectomies was uterine leiomyoma, a benign tumor of the uterine muscle. For total abdominal procedures it was diagnosed 47% of the time and for subtotal procedures 28%. The most common diagnosis in vaginal hysterectomies was genital prolapse which occurred in 67% of the cases. In radical hysterectomies, malignant neoplasms of the cervic uteri were the most common principal diagnosis.

This article was adapted from *Hysterectomies in New York State: A Statistical Profile*, prepared by the Information Systems and Health Statistics Group. A copy of the report is available from: Bureau of Information Science and Policy, New York State Department of Health, Empire State Plaza, Tower Building, Room 308, Albany, NY 12237.

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Management of head lice infestations

An infestation of the scalp and hair produced by the head louse, *Pediculus humanus capitis*, is a form of pediculosis. Individual cases and outbreaks may pose a recurring problem for many institutions such as schools and daycare centers. Although head lice infestations do not present a serious health threat, efforts to control them may be expensive, time-consuming and frustrating to parents, school personnel and health professionals. The following has been developed to assist health personnel in the surveillance and control of pediculosis in school and other institutional settings.

Biology of Head Lice

The head louse is a blood-sucking insect that lives on the scalp and hair of humans, usually behind the ears and at the nape of the neck. Adult lice may be capable of living for 14–30 days under ideal conditions on the human host. Lice require warmth and frequent blood meals for survival and will die at room temperature in two days or less if accidentally brushed off the host. The female louse will feed every four hours on the host, and will produce one egg from each blood meal. Therefore, one female can produce up to six eggs (nit) per day, and in her lifetime is capable of laying almost 180 eggs. Following fertilization, the female attaches her eggs to the base of individual hair shafts with a cement-like substance which makes them impossible to remove with normal shampooing or treatment with an insecticidal shampoo. The eggs hatch in seven to nine days and develop through three nymphal stages over five to ten days before reaching sexual maturity as an adult. As the embryo develops, the hair is also growing longer and will carry the egg along with it. Following the seven- to nine-day incubation period, the nit is expected to be about one quarter inch from the scalp when the nymph emerges. The nit's appearance changes from a whitish robust surface to a translucent eggshell which may be collapsed. Lice can crawl, but are not capable of either jumping or flying. It is possible to see nits and lice with the naked eye, but a hand lens or microscope is necessary to differentiate between nits, dandruff, hair casts or other artifacts.

The most common symptom associated with lice is itching. This is caused by an immune response to the louse saliva inoculated during feeding. Secondary infections may occur due to excoriation of the scalp.

Transmission

Head lice are normally transmitted by direct person-to-person contact. Transmission by fomites may be possible if children share storage areas, clothing, headwear or combs and brushes.

Individual Treatment

Many shampoos and lotions are available at local pharmacies. Some over-the-counter products contain synergized pyrethrins (A-200, RID, R&C). Others such as lindane (Kwell) and synthetic pyrethroids (Nix) require a physician's prescription. It is important to carefully follow the instructions on the product label. The efficacy of each product is influenced by the exposure time. Underexposure may not provide complete control, whereas overexposure may be dangerous to the patient. Most pediculicides are not 100 percent effective with a single treatment. Therefore, a second or possibly even a third treatment may be necessary in some cases. Second and third applications should be spaced seven to ten days apart.

The proper shampoo does not physically remove the nits. After the second treatment, the presence of nits on the hair shafts does not necessarily indicate that an active infestation still exists. Nits should be removed from the hair using a special comb designed for this purpose. These combs (Derbac, Innomed, Medicomb) contain very fine teeth and a tapered design which allows them to remove the small nits attached to the hair shafts. It may be useful to have the hair dampened when using these combs. Some patients have found that the addition of a conditioner may assist in nit removal.

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* Use of trade names does not imply endorsement by the New York State Department of Health.

Since lice are generally transferred between close contacts (family members, classmates and close playmates), they should be checked for lice. If lice are found on these contacts, they should be treated at the same time as the initial case to prevent reinfestation of the index case.

Control Summary

Prompt treatment of cases, screening of contacts and education of all patients and their families should be implemented. The following measures should be included for the proper control of lice:

- Confirm the case. Examine lice and/or nits under some form of magnification to eliminate improper diagnosis such as hair casts, dandruff, etc.
- Ensure proper treatment with an appropriate insecticidal shampoo followed by the removal of nits (complete removal is preferred).
- Ensure that washable items such as coats, hats, clothing and bedding are properly laundered and dried in a dryer for 30 minutes. Nonwashable items should be dry cleaned.
- Personal items such as brushes and combs should be washed thoroughly or treated with insecticidal shampoo as noted on the label or soaked in hot water (160°F) for five minutes.
- Family members and classmates should be screened and treated where necessary. All cases should be treated concurrently. If multiple cases or outbreaks appear to be occurring, contact the local or state health department.
- In a classroom or in similar settings, coats and hats should be placed in an individual bag if separate lockers and cubicles are not available for each student.

Please direct any questions about head lice to the Bureau of Communicable Disease Control at (518) 473-4436 or to the nearest regional office of the New York State Department of Health.

Cancer control intervention using available data bases

In 1987, funding was received from the National Cancer Institute (NCI) for a project entitled *Data-based Interventions for Cancer Control* (DBICC). In the project's first year, an integrated cancer prevention plan, *Cancer in New York State*, utilizing existing data bases was developed. Its focus is on cancers amenable to primary prevention such as lung or skin cancer. Emphasis is also on breast or uterine cervix cancer in which mortality can be reduced through proven cancer screening modalities. For each identified cancer site, the etiology and impact on the population is discussed and incidence and mortality rates are mapped by county and hospital market area. CAN*TROL, software developed by Dr David Eddy of Duke University, was used to model the potential benefits derived from implementation of cancer control interventions. The roles of both occupational exposure to carcinogens and diet are also discussed, as is access to oncologists and state-of-the-art cancer screening and treatment. The document also provides an overview of existing Department of Health activities using the framework of the Health Assurance Model. The model was developed to prioritize the department's goals for improving the health of New York State residents. Within the model, objectives are outlined that identify changes necessary for meeting the stated goals. These changes may be individual in nature, such as quitting smoking, or involve changes in the health system such as reducing the false-negative Pap smear rate. Objectives also include changes in the nonhealth system such as achieving a smokefree environment. Use of the Health Assurance Model allows the department to measure effectiveness of the programs and if goals are being met, as well as to identify gaps in programs or resources.

Proposed Intervention Projects

Two separate intervention projects are planned for the anticipated five-year term of the NCI award. The first aims to decrease breast cancer mortality in Nassau County. Women in Nassau County have experienced an unusually high breast cancer rate, consistently ranking among the top three New York State counties for breast cancer mortality. Computer modeling indicates that widespread use of physician examinations and mammographic screening might reduce this mortality by 20% in women over age 50. A multimedia approach will be developed to increase awareness, knowledge and actual screening participation levels. A baseline and four annual surveys will be conducted in order to measure the short-term effects of the promotional programs. These surveys will examine mammography utilization, clinical examination and breast self-examinations. The health promotion approaches developed for this project will serve as models for reducing breast cancer mortality rates in other high-risk areas of the state. The distribution of breast cancers in the clinical stage at the time of diagnosis will be examined in the fifth year to determine the effects of increased screening.

The second intervention project involves the development of cancer data profiles in manual form. These manuals will be distributed to local health units to assist them in determining local needs for cancer prevention and control efforts. Particular emphasis will be placed on small area analyses within counties. The mapping procedures developed for this grant will link local incidence and mortality patterns, sociodemographic characteristics, risk factors and service availability. Pilot versions of the manuals will first be developed for nine diverse counties. The utility of the manuals will be evaluated with potential users. Statewide small-area cancer analysis is expected to follow the pilot phase of the program.

For further information regarding this project, contact Patricia Kraichy, Bureau of Cancer Epidemiology, (518) 473-7817.

INTENSIVE CARE MEDICINE

Edited by James M. Rippe, MD, Richard S. Irwin, MD, Joseph S. Alpert, MD, and James E. Dalen, MD. 1,203 pp, illustrated. Boston, Little, Brown & Co, 1985. $80.00 (clothbound)

Intensive Care Medicine, written predominately by physicians at the University of Massachusetts Medical Center, provides an excellent overview of the practice of critical care medicine. One of the intrinsic strengths of this book lies in the precise description of all the procedures that physicians are required to master in order to properly care for patients in critical care units. The spectrum of the technical procedures ranges from the insertion of arterial and central venous catheters to the more complex procedures such as the insertion of the intra-aortic counterpulsation balloon in the treatment of patients with low cardiac output states. The indications, preparations, techniques, and related complications for each technical procedure are described in detail. In addition, comprehensive diagrams illustrate the important anatomical landmarks and the proper methodology for each procedure.

The book also describes the catastrophic illnesses that may involve the brain and the cardiovascular, pulmonary, gastrointestinal, renal, endocrine, and hematological systems. Characteristic features and basic treatment plans are provided for each major illness. As the authors attempt to discuss all the major illnesses, however, the pathophysiologic descriptions of the various disease processes and the treatment protocols are often brief. In addition, as the book was published in 1985, neither the use of recombinant-tissue plasminogen activator in the treatment of patients with acute myocardial infarction nor the use of azidothymidine (AZT) in the treatment of patients with acquired immunodeficiency syndrome is described.

The medical-legal and ethical issues of intensive care medicine are discussed in the final chapters of the book. The decision making processes regarding brain death and do-not-resuscitate orders in terminally ill patients are reviewed. Pertinent court decisions as well as the report of the President's Commission for the Study of Ethical Problems in Medicine are concisely reviewed in order to provide important guidelines for the intensive care physician who cares for terminally ill patients.

The authors are to be commended for condensing voluminous information into a relevant, concise, and highly readable book. *Intensive Care Medicine* is a worthwhile acquisition for any physician who seeks insight into the many facets of the care of critically ill patients.

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LONG TERM HEALTH CARE: PROVIDING A SPECTRUM OF SERVICES TO THE AGED

By Philip W. Brickner, MD, Anthony J. Lechich, MD, Roberta Lipsman, MSSW, and Linda K. Scharer, MUP. 383 pp, New York, Basic Books, Inc, 1987. $34.95 (hardcover)

"Between 1980 and 2020 the total number of people in the United States is expected to grow by 30 percent. At the same time, the number of elderly over 65 will grow by 100 percent." By 2020, people over age 65 will constitute more than 29 percent of the total US population. At that time people over 85 will account for more than 14% of the elderly and 2.4% of the entire population. The impact of these demographics for the US health care system is obvious since the elderly require and use more health services than the rest of the population.

Despite the undeniable "graying of America," our nation has not established a clearly defined national policy aimed at providing a spectrum of services to the aged. For whatever the reason, this lack of a national policy can lead to the inevitable crisis which may or may not lead in turn to a rational, reasonable, and equitable solution. Based on past history, the "resolution" could come from a Bandaid approach, rather than a neat professional bandage.

Combining many years of practical experience in providing services to the elderly, Brickner and his associates tackle the long term health care problem with thoughtfulness and solid documentation. Humanistic case studies provide personal warmth to their admittedly non-neutral stance as they cover all fronts on the long term health care scene.

In considering the long term care spectrum, areas identified for immediate attention include rising costs, inadequate discharge planning, and hospital overstay. Costs could be controlled via services provided by capitation payments to health maintenance organizations (HMOs or social/HMOs), via changes in entitlement programs such as enhancing consumer choice through the use of vouchers, and through block grant programs to research increasing the income of the elderly. The latter could also be applied in trying innovative fringe benefit choices while working, promoting reverse mortgage concepts, and defining long term care insurance mechanisms.

Government financing for long term care could include a voucher system to stimulate consumer incentives to control costs, an increase in Medicare revenues, and the expansion of a variety of prospective payment reimbursement options. Private funding could involve tax deductions for in-home services, conversion of employee health benefits, an Individual Retirement Account (IRA) to pay for long term care, and adjustments in Social Security payments.

A chapter devoted to clinical concerns emphasizes dementia, Alzheimer disease, and alcoholism. However, the major thrust concerns the relationship of health and disease to functional ability.

Long term home health care should be utilized whenever possible as long as individuals are capable of being cared for at home. In view of the importance of this service, the authors devote an entire chapter to that subject. Home health care relies on paraprofessional workers and informal support from family and friends. Both of these vital adjuncts to home care receive attention in this book.

Day care, foster care, hospice, and respite services are not neglected in the spectrum of services discussed. In addition, housing is also considered. Finally, the authors discuss the nursing home, which does have a place in the spectrum of services, but that only at the end of the list.

In coming to the end of this well-documented and thought-provoking book about a major and ever increasing health care problem, the authors state a "bottom line" truth. "Our nation's ability to establish a long term care spectrum for the aged will depend in large part on the attitudes of those in the position to develop and pay for it." That group includes the taxpayers, elected representatives, and the bureaucrats hired to administer the programs.

If changing attitudes is the first step, then this book by Brickner and his associates should be required reading.

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OCCUPATIONAL BACK PAIN; SPINE: STATE OF THE ART REVIEWS

By Richard A. Deyo, MD, MPH. 168 pp. Philadelphia, PA, Hanley & Belfus, Inc, 1987. $32.00 (hardcover)

The issue of *Spine: State of the Art Reviews*, dedicated to occupational back pain (Vol. 2, No. 1), consists of a series of well-written, well-referenced articles which attempt to "summarize current knowledge and opinion regarding occupational risk factors for back pain, the success of various preventive measures, the role of the Worker's Compensation system and the role of the primary care, surgical and rehabilitative professionals." The 13 articles included in the book are written by recognized authorities in the fields of occupational medicine, family medicine, physical medicine and rehabilitation, orthopedics, the neurosciences, pain therapy, and compensation insurance carriers.

Certain themes are repeatedly encountered throughout the book, although the context of presentation does vary. The reader is left in no doubt with regard to the enormity of the problem of work-related low back pain and the fact that causes are complex, approaches to treatment variable, and outcomes unpredictable. Rather than being tedious and repetitious, however, the recurring theme should convince the reader that physicians who deal with victims of occupational back pain must be aggressive in their treatment, and consistent in their approach to back pain if any impact is to be made in lowering costs and disability. Employers must also recognize the importance of their role in controlling the problem through workplace modifications, prevention programs, and a willingness to allow the physician to be aggressive in establishing an accurate diagnosis, providing definitive care, and keeping the employee active and productive even on a limited basis during treatment and rehabilitation from a back injury.

Most of the articles are broad overviews of accumulated experiences in particular areas of the back pain problem, including the areas of cost, causative factors, and compensation issues. There are several articles dealing with specific treatment approaches including spinal manipulation, invasive therapeutic procedures, aerobic exercise, and pain therapy programs. These too, however, are compilations of accumulated experiences from the authors' personal practice and from the literature, and do not offer specific treatment protocols or approaches. The article on preplacement screening radiography of the low back is an example of one article which does attempt to make specific recommendations as to when such screening radiographs are appropriate and what one can hope to find when the screening is performed. It provides the practitioner with data to support a decision whether or not to get involved in such screenings in certain lower risk industries when requested to do so.

One of the greatest strengths of the book lies in the fact that it concisely summarizes in one place an enormous accumulation of experience and data in the care of back injured patients. The extensive reference listings (ranging from 17 to 114 references) annotated in the body of each article serve to direct the reader to specific in-depth articles if desired, and a complete index directs the reader to specific information. Occasional typographical errors (which have been seen in other books in the *State of the Art Review* series) and some difficult-to-read radiographic reproductions are the only drawbacks that I found in the book.

Occupational Back Pain achieves the editor's goals as stated in his preface to the book. It will make a fine reference text for anyone interested in broad overviews of current knowledge in the many important areas of concern in dealing with work-related back pain. It should not be considered a textbook to instruct the reader in definitive management of back pain. Rather, it will be a useful addition to the library of any health care professional who needs to be kept up to date in current thinking in a clinical area full of controversy and having a large impact on the physician, the patient, the employer, and the health care dollar.

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REHABILITATION OF ATHLETIC INJURIES; AN ATLAS OF THERAPEUTIC EXERCISE

By Joseph S. Torg, MD, Joseph J. Vesgo, MD, and Elizabeth Torg, BA. 275 pp, illustrated. Chicago, Yearbook Medical Publishers, Inc, 1987. $29.95 (hardcover)

This volume is a significant addition to the literature of a field that is already inundated. It is directed to all those who treat sports trauma. The authors, particularly Joseph S. Torg, MD, are recognized authorities in the field of sports trauma and rehabilitation. Many of the activities of the University of Pennsylvania's Sports Medicine Center are directed and coordinated by the authors.

The text consists of 12 chapters. Chapter 1 is devoted to common rehabilitative modalities. Diathermy, ultrasound paraffin baths, infrared, electrogalvanic stimulation, etc, are mentioned as additional treatment possibilities but are not discussed in detail. Chapters 2 through 11 present common sports injuries by anatomical sites: foot, ankle, leg, knee, hip, low back, shoulder, neck, elbow, wrist, and hand. The format of these chapters is uniform: injury type; problem; treatment, including splinting, strapping, and casting; and rehabilitation (stretching and strengthening exercises, range of motion exercises). The illustrations are clear, explanatory, and abundant. Chapter 12 expands the rehabilitation discussion to include medications (nonopiate and opiate drugs) and cryotherapy.

There are several weak areas in the text. It would have been useful to have had a discussion in chapter 1 about the history of an athletic trauma event: both the acute and chronic phases. The diagnostic approaches and treatment modalities certainly differ for these phases. It would be nice to recognize the familiar RICE acronym (*R*est, *I*ce, *I*mmobilization, *C*ompression, *E*levation) in the material, although most of its components are discussed in the text at some point. Chapter 12 should have included material on some of the newer nonopiate drugs (ie, choline trisalicylate) and injection corticosteroids. The additional sections dealing with the asthmatic athlete, fluid and electrolytes, motion sickness, allergic disorders, leg cramps, and anabolic steroids are interesting and important topics, but seem to be incongruous with the rehabilitation theme of the book. Also, the American College of Sports Medicine has developed excellent position papers on several of these topics, none of which are referenced.

Nonetheless, this text has filled a major vacuum in the sports medicine literary field. In particular, its organized, pithy style is most appealing for primary care physicians. Busy practitioners are given clear rehabilitative and treatment programs to follow and suggested guidelines for the consultative process. The book should do much to improve the primary care physician's understanding of the unappealing rehabilitative process and facilitate relationships with consultants in physical therapy, rehabilitation, and orthopedic surgery. Lastly, it is reassuring to see an orthopedic group successfully address a neglected health care issue in a nonsurgical, nonsubspecialty manner.

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THE SELF IN THE SYSTEM: EXPANDING THE LIMITS OF FAMILY THERAPY

By Michael P. Nichols, PhD. 326 pp. New York, Brunner/Mazel Publishers, 1987. $30.00 (hardcover)

Reading Dr Nichols' book stimulated my memories of the days when Jackson, Bateson, and Haley first began to teach and advocate family therapy. At that time, it seemed to us young practitioners that the solutions to mental illness had at last been found. Later, as we learned about other therapies, the power of their teaching seemed to diminish. Family therapy as a single entity lost its seductiveness for us, finally seeming to be practiced by polarized and polemic therapists who ordered people around, forgot about the individual, and didn't seem to understand the inner workings and psyche of the individual. Dr Nichols, Professor of Psychiatry at Albany Medical College, is a family therapist who has long studied the field of family therapy, its history, and its place in the treatment armamentarium of today. He has offered a needed corrective view of family therapy that directs it back into the group of mainstream therapies.

In a well written and easily readable book, he offers a critique of the field that he knows so well and gently criticizes its isolation from the rest of psychiatric and psychological thinking. It was obvious as I read this book that he is a skilled teacher. He can present complicated ideas succinctly and understandably. Utilizing appropriate case examples from his own experience, he discusses the concepts of the family as a system and the individual as a system until he develops a smooth flowing hierarchy. He deals with the problems of change, the need for interaction with the family with empathy, how to access the family, and how to reaccess the family as treatment goes on. He touches upon object relations and psychopathology. He also deals with understanding and with resistance.

This book is welcome reading for any family therapist who has lost perspective of the self, or any individual therapist who has lost view of the family. For those not familiar with family treatment, it is a useful and easily read overview that should help one to understand this very important field. For those who are unfamiliar with psychiatry or psychology, it also helps as an introduction to the field. I am pleased to recommend it as an excellent and highly readable book offering a positive introduction to the field.

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TEXTBOOK OF DIAGNOSTIC MEDICINE

Edited by A. H. Samiy, MD. 990 pp, illustrated. Philadelphia, Lea & Febiger, 1987. $69.50 (hardcover)

This is a multiauthored text written to bridge the gap between the disease-oriented textbooks of medicine and the presenting complaint of the patient. As described in the preface, this book's goal is to be "an expert guide in the evaluation of the patient's problem, with particular emphasis on the evaluation of symptoms and signs."

The book is organized into chapters beginning first with the general principles of clinical diagnosis and laboratory testing followed by chapters which deal with specific organ systems. Each chapter is written by one or more subspecialists in that particular field. The chapters are subdivided into topics which are clinical problems or complaints that might be encountered in practice. Each topic is reviewed in terms of definition, pathophysiology, clinical presentation, differential diagnosis, diagnostic evaluation, and workup. At the end of each chapter is a list of references for further reading. More than 500 specific complaints or problems are discussed.

This text is very readable and includes one of the most comprehensible explanations of basic clinical statistics. The discussions are concise and include well integrated lists of differential diagnoses and flow diagrams. At the end of each topic the reader is left with a basic understanding of pathophysiology, differential diagnosis, and how to proceed with the further workup of the patient's presenting complaint.

Clearly, it is not the goal of this book to serve as an all-inclusive medical resource, but rather to work synergistically with most of the presently available textbooks of medicine. Since most medical textbooks are organized along the lines of specific disease entities, they can only be fully utilized if the full differential is known. This book facilitates this by expanding the patient's clinical complaint into a differential diagnosis which can be further explored in the disease-oriented textbooks of medicine.

While in general there is high consistency to the quality of each chapter's content, the references at the conclusions of the chapters are of variable usefulness. Ideally, the references in a text such as this should add direction to the reader's further exploration into the topic of discussion. While many of the chapters give a good selection of both specific and general references, other chapters have relatively short lists or contain references only available in foreign languages. While this is a relatively minor weakness, it is important since many who may be using this text will want to read further on specific topics.

Overall, this is a well-written text which nicely complements presently available medical references. It is highly suitable for medical students during their clerkships and clinical rotations, and would further expand the knowledge of most house staff. Practicing physicians may also find it useful in fully appreciating and evaluating their patient's complaints.

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Battling AIDS through behavior modification

In addition to the millions of dollars earmarked for research on AIDS, the federal government allocated nearly half a billion dollars last year for programs aimed at educating the public about AIDS and reducing the transmission of the AIDS virus among high risk populations. Unfortunately, understanding and manipulating the social responses to AIDS can prove as complex a problem as tracking or controlling the virus itself in the laboratory.

Preventing the spread of AIDS by altering human behaviors is a formidable task, both in theory and in practice. Attempting to modify behaviors linked to intense physical and emotional needs, such as drug abuse and sexual practices, poses countless difficulties, observes William Booth (*Science* 1988; 242:1237–1238), "even if the social engineers knew what they were doing, which most freely concede they do not."

Yet, behavior modification based on an approach that combines knowledge of the threatening behavior, realistic risk assessment, a healthy dose of fear, alternative behaviors, and societal pressure is possible. For example, Booth cites the "remarkable changes in behavior . . . reported from some quarters, particularly in cities where there are large, well-organized gay communities that have been ravaged by the AIDS epidemic."

Current public education efforts to prevent AIDS are not based on universal theories or proven methods, primarily because social scientists are not really sure what motivates people to engage in risky behaviors, and, therefore, there is no clear consensus as to how to alter risk-taking tendencies. The author describes the current AIDS prevention programs as being "built upon the idea that it is better to do something—anything—than nothing at all."

He outlines the general principles incorporated in behavior modification programs: recognition that one is at risk and understanding the risk; awareness of how to avoid danger; acceptance of one's ability to change and the rewards accompanying such change; and reinforcement of the new behavior via societal pressure, in which the new behavior becomes the societal norm.

One problem inherent in any behavior modification program is that people tend to deny personal risk. An important factor in AIDS prevention is convincing people that the threat of AIDS is a real one, and that everyone is potentially at risk for AIDS—it is not strictly a disease affecting high risk groups such as homosexual men and intravenous drug abusers.

Behavior modification programs often use fear to convince subjects of the need for change. "Yet the problem with fear is that it is like a drug: it does not last for long." There is a fine line between using fear as a motivating factor and creating excessive fear that promotes denial and an inability to act. Booth emphasizes the widely held belief that "if fear is going to be used to motivate people, it is essential that they be offered a solution." Therefore, AIDS prevention programs teach homosexual men about safer sex practices and drug abusers how to clean their injection paraphernalia with bleach.

The key factor in changing behaviors is to alter group norms and standards. If a segment of a population adopts the new behavior as common practice, the new behavior will inevitably become the norm, and convincing the majority of the community to follow the crowd and adopt the new behavior will be a simpler task.

Sloan-Kettering targets women in new ad campaign

The Memorial Sloan-Kettering Cancer Institute's "Early Detection: It's a Way of Life" campaign is a nationwide public education program that stresses the importance of early cancer detection and outlines the medical care available to lessen the incidence, severity, and mortality rates of various types of cancer.

The campaign's current focus is on early detection of breast cancer. Aimed at women over 35 years of age, the advertising strategy will employ both the print and broadcast media to alert women to the need for regular mammograms and a continual breast care program that includes yearly breast examinations by a physician and monthly breast self-examination. Sloan-Kettering will provide a toll-free number for individuals to request written information on cancer detection.

Consensus on postmenopausal hormone therapy

Estrogen therapy is a fairly well accepted means of treating women with severe symptoms of menopause, such as hot flushes and mood changes. Estrogen deficiency due to menopause also accelerates bone loss in women and is believed to be a major factor in the increased risk of osteoporosis in postmenopausal women. Therefore, estrogen replacement therapy is generally recommended for the prevention and treatment of postmenopausal osteoporosis in women at high risk for the disorder (*Mayo Clin Proc* 1988; 63:453–460).

According to Lufkin et al at the Mayo Clinic, risk factors include Caucasian race, advanced age, thin habitus, estrogen deficiency, immobilization, extremely strenuous exercise, alcoholism, and certain endocrine diseases. Hereditary factors, dietary calcium deficiency, smoking, and inadequate exercise may also contribute to one's risk.

Estrogen treatment itself is not without risks. Possible side effects include endometrial cancer, breast cancer, thromboembolic disease, hypertension, and migraine headaches. Furthermore, there is ongoing debate as to the optimal mode of delivery of estrogen, the dosage, the length of treatment, and the appropriate age to initiate treatment.

Despite these lingering questions, estrogen replacement therapy is becoming more common among postmenopausal women. Ongoing studies continue to probe the effectiveness, risks, and long term outcomes of the treatment.

One problem with estrogen therapy in postmenopausal women is the risk of endometrial hyperstimulation. The solution appears to be a combination therapy, in which progestagen is administered on a cyclic schedule together with the estrogen. Recommendations for appropriate pro-

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NEWS BRIEFS are compiled and written by Vicki Glaser.

gestagen use are presented in a consensus report prepared in conjunction with the first International Consensus Conference on Progestagen Use in Postmenopausal Women that took place in September 1988 (*Lancet* 1988; 1:1243-1244). Following are excerpts from the report:

- Basic science, clinical, and epidemiological studies have shown that progestagen treatment is efficacious and is indicated for opposing the effects of oestrogen on the endometrium.
- ... increasing the number of days of progestagen treatment, beyond 7-10 days each cycle/month, has a further beneficial suppressive effect on endometrial proliferation . . . .the duration of progestagen administration should be individually tailored.
- The available data indicate that the addition of progestagen to the oestrogen therapy does not influence the skeletal response provided that the oestrogen dose is adequate.
- Short-term studies have shown that some progestagens, by themselves, are capable of reducing postmenopausal bone loss. However, there are no data on the long-term effects of progestagen-only treatment on bone mass, nor on fracture frequency.
- More information is urgently needed on the pharmacodynamics of progestagens.
- There are insufficient long-term histological and epidemiological data to advocate the routine use of daily doses of both progestagen and oestrogen (continuous-combined therapy).
- Some progestagens in large doses cause adverse symptomatic and psychological changes; the lowest dose required for endometrial protection should be prescribed.
- More clinical data are urgently required.

GM-CSF demonstrates ability to lower serum cholesterol levels

A recent, unexpected finding has led to speculation about a powerful new weapon in the fight against high cholesterol and atherosclerosis. The compound being studied is human granulocytemacrophage colony-stimulating factor (GM-CSF), a glycoprotein hormone that stimulates the growth and differentiation of myeloid progenitor cells and enhances the activities of mature neutrophils, eosinophils, and mononuclear phagocytes.

Based on their knowledge of GM-CSF functioning, Nimer, Champlin, and Golde of the UCLA School of Medicine were studying the ability of a recombinant form of human GM-CSF to improve hematopoiesis in patients with aplastic anemia (*JAMA* 1988; 260:3297-3300). Eight patients participated in the GM-CSF clinical trial. As data accumulated from the weekly SMA-12 chemistry studies, which included determinations of serum triglyceride and cholesterol levels, the researchers noted dramatic reductions in the serum cholesterol levels of seven of the eight patients. Decreased serum cholesterol levels were apparent within the first ten days of GM-CSF therapy and continued throughout the course of treatment; however, discontinuation of GM-CSF therapy resulted in a return of serum cholesterol concentrations to baseline levels.

Cholesterol levels before and during GM-CSF treatment were significantly different ($p = 0.008$). In six of the seven patients who demonstrated decreased serum cholesterol levels, the percentage reduction ranged from 27% to 53%. There were no consistent changes in serum triglyceride levels.

Although the mechanism of GM-CSF's cholesterol lowering activity is not yet clear, Nimer et al observed that "the effect of GM-CSF on lowering serum cholesterol levels was not dependent on changes in the white blood cell count, suggesting that GM-CSF has primary effects on cholesterol metabolism or transport." The authors proposed that GM-CSF acts on mononuclear phagocytes, which have an active role in processing and transporting cholesterol. These are preliminary findings, however, and further clinical studies are required to define the mechanisms of action of GM-CSF and to explore the cholesterol lowering ability of other hematopoietins.

Adverse effects associated with GM-CSF therapy included headache, myalgias, and mild fatigue. Nimer and colleagues reported that the toxic effects of GM-CSF were "mild and tolerable" at doses less than or equal to 16 μg/kg/d. Higher doses were not well tolerated. Additional studies are needed to determine whether the beneficial cholesterol lowering effects of GM-CSF therapy can be maintained at doses that minimize adverse reactions.

Increasing physician participation in alternative delivery systems

An increasing number of practitioners, particularly younger physicians and female physicians, are becoming involved with alternative health care delivery systems such as health maintenance organizations (HMOs) and preferred provider organizations (PPOs). Using data from the American Medical Association's Socioeconomic Monitoring System, the Center for Health Policy Research of the AMA estimates that in 1986, more than a third of physicians received income from an HMO or an independent practice association (IPA). The data also demonstrated a substantial rise in the proportion of physicians' incomes that came from participation in such organizations. Between 1983 and 1986, the percentage of physicians involved with PPOs more than tripled.

Federal tax legislation that restricted or eliminated the advantages of incorporation is blamed for the appreciable decrease noted in the percentage of physicians in incorporated practices in 1986.

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Nathan Ambinder, MD, New York. Died August 12, 1988; age 82. Dr Ambinder was a 1934 graduate of the Universitaet Wien, Medizinische Fakultaet, Wien, Austria. He was a Fellow of the American Academy of Family Practice. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Sureyya Naci Arkun, MD, Brooklyn. Died May 1, 1988; age 71. Dr Arkun was a 1943 graduate of the Bursa Tip Fakultesi Istanbul Universitesi, Bursa, Turkey. His memberships included the International Society of Hematology, the American Geriatrics Society, the American Society of Hematology, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Addison J. Burke, MD, Garden City. Died November 4, 1988; age 60. Dr Burke was a 1952 graduate of the New York Medical College. He was a Fellow of the American College of Anesthesiologists and a Diplomate of the American Board of Anesthesiology. His memberships included the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the Nassau County Medical Society, and the Medical Society of the State of New York.

Saul Commins, MD, Rochester. Died October 22, 1988; age 73. Dr Commins was a 1940 graduate of the New York Medical College. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. He was a member of the Medical Society of the County of Monroe and the Medical Society of the State of New York.

J. Robert Del Terzo, MD, New York. Died October 2, 1988; age 75. Dr Del Terzo was a 1942 graduate of the St Louis University School of Medicine, St Louis, Missouri. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. He was a member of the New York Academy of Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Ephraim Donoso, MD, New York. Died November 11, 1988; age 71. Dr Donoso was a 1942 graduate of the Universidad Mayor de San Andres, Facultad de Medicina, La Paz, Bolivia. He was a Fellow of the American College of Cardiology and the American College of Physicians. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Irving Drabkin, MD, Massapequa. Died November 25, 1988; age 92. Dr Drabkin was a 1924 graduate of the University of Virginia School of Medicine, Charlottesville, Virginia. His memberships included the New York Academy of Medicine, the American Academy of Family Physicians, the Nassau County Medical Society, and the Medical Society of the State of New York.

Richard L. Dreifuss, MD, Forest Hills. Died November 9, 1988; age 75. Dr Dreifuss was a 1940 graduate of the University of London Faculty of Medicine, London, England. He was a Fellow of the American Academy of Pediatrics and a Diplomate of the American Board of Pediatrics. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Charles Fisher, MD, New York. Died November 16, 1988; age 80. Dr Fisher was a 1940 graduate of Northwestern University Medical School, Chicago, Illinois. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the American Psychiatric Association, the American Psychoanalytic Association, the New York County Medical Society, and the Medical Society of the State of New York.

Dorothy Fogel, MD, Brooklyn. Died October 28, 1988; age 81. Dr Fogel was a 1933 graduate of the New York University School of Medicine. She was a member of the New York Society for Physical Medicine & Rehabilitation, the American Geriatrics Society, the New York County Medical Society, and the Medical Society of the State of New York.

Charles Amos Galyon, MD, Kingston. Died July 1, 1988; age 63. Dr Galyon was a 1955 graduate of the Albany Medical College of Union University, Albany, New York. He was a member of the Medical Society of the County of Ulster and the Medical Society of the State of New York.

David Gersten, MD, Margate, Florida. Died August 14, 1988; age 81. Dr Gersten was a 1934 graduate of the Faculty of Medicine University of Glasgow, Glasgow, Scotland. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

H. Pelham Glasier, MD, Guilderland. Died October 20, 1988; age 82. Dr Glasier was a 1931 graduate of Columbia University College of Physicians and Surgeons, New York. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. His memberships included the New York Academy of Medicine, the American Geriatrics Society, the Society for Experimental Biology and Medicine, the Medical Society of the County of Albany, and the Medical Society of the State of New York.

Harold Bernard Hermann, MD, Brooklyn. Died November 22, 1988; age 88. Dr Hermann was a 1925 graduate of the New York University School of Medicine. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Urology. His memberships included the American Urological Association, the American Association of Clinical Urology, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Ludmila Krikun, MD, Forest Hills. Died October 4, 1988; age 62. Dr Krikun was a 1951 graduate of the First Moscow

Order of Lenin Medical Institute, Moscow, USSR. She was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Louis A. Lanzetta, MD, Long Beach. Died October 28, 1988; age 81. Dr Lanzetta was a 1933 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the American Occupational Medical Association, the New York County Medical Society, and the Medical Society of the State of New York.

Abraham Lew, MD, Long Beach. Died July 10, 1988; age 96. Dr Lew was a 1922 graduate of the Universitaet Wien, Medizinische Fakultaet, Wien, Austria. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Leon Samuel Loizeaux, Jr, MD, Irvington. Died November 17, 1988; age 79. Dr Loizeaux was a 1934 graduate of the Cornell University Medical College, New York. He was a Fellow of the American College of Obstetricians and Gynecologists, a Fellow of the American College of Surgeons, and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Joseph Anthony Malejka, MD, New York. Died November 7, 1988; age 63. Dr Malejka was a 1952 graduate of the National University of Ireland. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Harold C. May, Jr, MD, New York. Died July 25, 1988; age 45. Dr May was a 1972 graduate of the Wayne State University School of Medicine, Detroit, Michigan. He was a Fellow of the American College of Obstetricians and Gynecologists and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Frank Guy Moore, MD, Rockville, Maryland. Died November 14, 1988; age 92. Dr Moore was a 1922 graduate of the Johns Hopkins University School of Medicine, Baltimore, Maryland. He was a member of the Broome County Medical Society and the Medical Society of the State of New York.

Sidney N. Neichin, MD, Brooklyn. Died April 14, 1988; age 81. Dr Neichin was a 1934 graduate of the University of Tennessee Center for Health Sciences, Memphis, Tennessee. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Cornelius J. O'Connell, MD, Buffalo. Died October 28, 1988; age 57. Dr O'Connell was a 1956 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. His memberships included the New York State Society of Internal Medicine, the American Society of Internal Medicine, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Constantine J. Papastrat, MD, Poughkeepsie. Died October 29, 1988; age 66. Dr Papastrat was a 1952 graduate of the Boston University School of Medicine, Boston, Massachusetts. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. He was a member of the Dutchess County Medical Society and the Medical Society of the State of New York.

John David Persse, MD, Sarasota, Florida. Died October 21, 1988; age 72. Dr Persse was a 1942 graduate of the State University of New York at Buffalo School of Medicine. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. His memberships included the New York Academy of Medicine, the New York State Surgical Society, the American Society of Abdominal Surgeons, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Edward A. Quinlan, Jr, MD, Rye. Died November 4, 1988; age 82. Dr Quinlan was a 1938 graduate of the Medizinische Fakultaet der Julius Maximiliams Universitaet, Wurzburg, Bayern, Germany. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Eugene David Rosenfeld, MD, New York. Died October 25, 1988; age 71. Dr Rosenfeld was a 1943 graduate of the University of Colorado School of Medicine, Denver, Colorado. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Daniel Perry Schultz, MD, Port Jervis. Died October 31, 1988; age 83. Dr Schultz was a 1935 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a member of the American Society of Abdominal Surgeons, the Medical Society of the County of Orange, and the Medical Society of the State of New York.

Bertram S. Schwartz, MD, Jericho. Died October 29, 1988; age 65. Dr Schwartz was a 1951 graduate of the State University of New York Health Science Center at Brooklyn. He was a Fellow of the American Academy of Pediatrics and a Diplomate of the American Board of Pediatrics. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Richard George Taylor, MD, Hamburg. Died October 24, 1988; age 82. Dr Taylor was a 1930 graduate of the State University of New York at Buffalo School of Medicine. He was a Qualified Fellow of the International College of Surgeons. He was a member of the Medical Society of County of Erie and the Medical Society of the State of New York.

Robert E. Tschorn, MD, Sandgate, Vermont. Died October 1988; age 83. Dr Tschorn was a 1931 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

William E. Wakeley, MD, Chatham, New Jersey. Died October 8, 1988; age 72. Dr Wakeley was a 1942 graduate of the Cornell University Medical College, New York. He was a Diplomate of the American Board of Internal Medicine. His memberships included the American Occupational Medical Association, the New York County Medical Society, and the Medical Society of the State of New York.

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1988; 88:456–462). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith[2] reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al*. Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83:925–927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics*, ed 12. Philadelphia, WB Saunders Co, 1983, pp 337–338.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Pascal James Imperato, MD, Editor, *New York State Journal of Medicine*, PO Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

Schedule of Sessions 113
Officers, Councilors, and Trustees 115
Members of the House 118
Reference Committees 122
Nominating Committee 123
Report of the House Committee on Bylaws 124

HOUSE OF DELEGATES

April 6 through 9, 1989
Stouffer Plaza Hotel

SCHEDULE OF SESSIONS

Thursday, April 6	
Opening Session	1:00 p.m.
Friday, April 7	
Reference Committee Hearings	8:30 a.m.–5:00 p.m.
Saturday, April 8	
Second Session	9:00 a.m.
Third Session	1:30 p.m.
Sunday, April 9	
Closing Session	9:00 a.m.

A Guide to the 1989 Annual Meeting

Policy-making in The House of Delegates

This section of the New York State Journal of Medicine is a call to the officers, delegates, and members of the Medical Society of the State of New York to attend the 183rd Annual Meeting of the Society and the meeting of its House of Delegates; 296 delegates representing 61 county medical societies, 9 district branches, specialty societies, the hospital medical staff section, 12 medical schools, the resident and medical student membership will meet at the Stouffer Plaza Hotel in Rochester, New York on April 6–9, 1989.

The officers, members of the Council and the Board of Trustees, past presidents of the MSSNY, the Commissioner of the New York State Department of Health, the Past President of the American Medical Association from New York, Gerald D. Dorman, MD, and Past Executive Vice-President of the Medical Society of the State of New York, George J. Lawrence, Jr., MD, are also official representatives to the House.

Mandated by the Bylaws of the Society, this section lists the names of elected representatives, delegates who are serving on reference committees, and selections of the Nominating Committee for the election of Officers, Councilors, Trustees and AMA Delegates. Also included are the amendments to the Bylaws which will be considered by the House of Delegates.

Since the House of Delegates is the legislative body of the Society, charged with the general management of the State Society and its affairs, members of the Society are urged to use this information to consult with and advise their representatives. Resolutions introduced by the representatives bring members' concerns to the attention of the entire Society. Representatives not only have the responsibility to present the views of their constituents, but also to inform them of the actions taken by this governing body. The House of Delegates also formulates policy for the Society. No officer, councilor, board, commission, committee, or employee may initiate any policy unless such policy has been expressly approved by the House of Delegates, or by the Council of the Society.

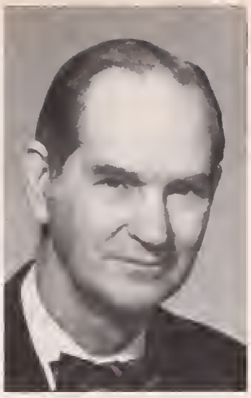
If one were to choose the most important work of the House of Delegates, it would have to be that of the reference committees. Any member of the Medical Society of the State of New York has the right to appear before any reference committee and participate in its deliberations. It is also in the reference committees that people outside of the State Society are asked to give their views. Nonmember physicians, guests or interested observers who wish to express their opinions may, with permission, come before the reference committee and speak to the business of the committee. I strongly urge members to use this forum to express their views.

Charles N. Aswad, MD, Speaker
House of Delegates

Seymour R. Stall, MD, Vice-Speaker
House of Delegates

MEDICAL SOCIETY OF THE STATE OF NEW YORK

Officers 1988–1989



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President-Elect

Samuel M. Gelfand

Nassau
Past-President

Robert A. Mayers

Westchester
Vice-President

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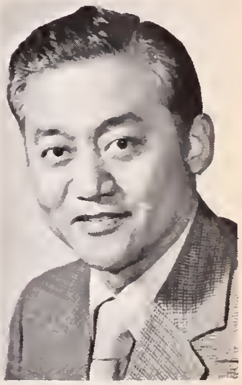

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Assistant Treasurer

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Dutchess
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Victor J. Tofany

Monroe
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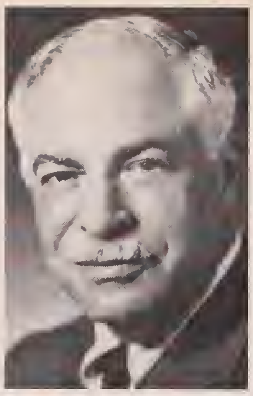
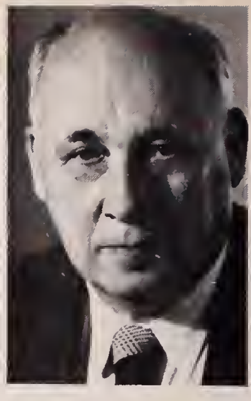


M. Theodore Tanenhaus

Kings

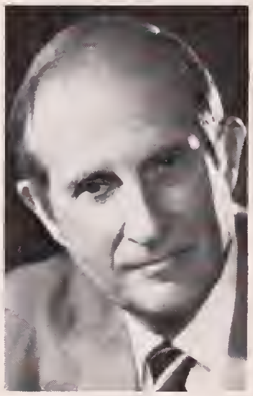


Bernard J. Pisani

New York

Daniel F. O'Keeffe

Warren



Allison B. Landolt

Westchester

John A. Finkbeiner

New York

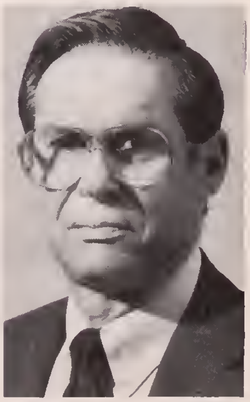
James M. Flanagan

Wayne

Councilors 1988–1989

Robert E. Fear

Suffolk

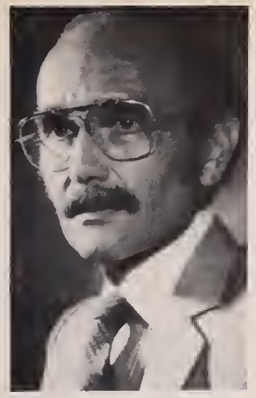


Stanley L. Grossman

Orange

Thomas D. Pemrick

Rensselaer



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Duane M. Cady

Onondaga

William A. Dolan

Monroe

Robert E. Gordon

Kings

Stuart I. Orsher

New York

Franklin A. DePeters

Warren

Sidney Mishkin

Nassau

Jeffrey A. Ribner

Broome

Anthony P. Santomauro

Erie

Robert J. Newborn

New York

Penny Stern

The Bronx

1989 HOUSE OF DELEGATES MEMBERS

The following is a list of members of the 1989 House of Delegates of the Medical Society of the State of New York.

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Term Expires 1991
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Term Expires 1989
Penny Stern, Bronx

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1971–1972—George Himler, New York
1972–1973—Edward Siegel, Clinton
1974–1975—Lynn R. Callin, Monroe
1975–1976—Ralph S. Emerson, Nassau
1976–1977—George L. Collins, Jr., Erie
1977–1978—Carl Goldmark, Jr., New York
1978–1979—George T. C. Way, Dutchess
1980–1981—Ralph M. Schwartz, Kings
1982–1983—Richard D. Eberle, Onondaga
1983–1984—Bernard J. Pisani, New York
1984–1985—Allison B. Landolt, Westchester
1985–1986—Daniel F. O'Keeffe, Warren
1986–1987—John A. Finkbeiner, New York
1987–1988—Samuel M. Gelfand, Nassau

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Past President of the American Medical Association
Gerald D. Dorman, New York

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Commissioner, New York State Department of Health
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(*Elected Delegates of District Branches*)
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Second—Bruce H. Berlin, Suffolk
Third—
Fourth—Michael J. Lynch, Washington
Fifth—Frederic A. Stone, Jefferson
Sixth—Anton A. Vreede, Broome
Seventh—Norman R. Loomis, Ontario
Eighth—Robert J. Reszel, Niagara
Ninth—John A. Ramsdell, Westchester

Specialty Society Delegates
New York State Society of Anesthesiology—Edward C. Sinnott, Nassau
New York State Society of Dermatology—Donald L. Levin, Onondaga
New York Chapter American College of Emergency Physicians—
New York State Academy of Family Physicians—James F. Wright, Warren
New York State Society of Internal Medicine—Robert M. Kohn, Erie
New York State Neurosurgical Society, Inc.—Mihai D. Dimancescu, Nassau
American College of Obstetricians and Gynecologists—District II—

New York Occupational Medical Association, Inc.—Brock Powell, Monroe
New York State Ophthalmological Society—Frank J. Piper, Onondaga
New York State Society of Orthopaedic Surgeons, Inc.—Leo Arthur Green, Queens
New York State Society of Otolaryngology—Head and Neck Surgery—Lyon M. Greenberg, Albany
New York State Society of Pathologists—Edward C. Zaino, Nassau
American Academy of Pediatrics—District II—
New York Society of Physical Medicine and Rehabilitation, Inc.—David Ernstoff, Queens
New York Regional Society of Plastic and Reconstructive Surgery—
New York State Psychiatric Association—George L. Ginsberg, New York
The New York State Radiological Society, Inc. (A Chapter of the American College of Radiology)—Victor A. Panaro, Erie
New York State Society of Surgeons, Inc.—Theodore C. Max, Oneida
New York State Urological Society, Inc.—Marcus L. Shoobe, Albany

Delegates from Component Medical Societies

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John W. Abbuhl, Albany
James H. Puleo, Albany
Nathan P. Reed, Albany
Demostene Romanucci, Latham

Allegany (*1*)
M. Keith Sale, Wellsville
Bronx (*10*)
John P. Albanese, Bronx
Sana Bloch, Bronx
Dattatraya G. Lanjewar, Pelham Manor
Charles LaVerdi, Bronx
Stanley Luftschein, Rye
Hugo M. Morales, Bronx
Venkatachala Pathy, Bronx
Narayan Pai, Bronx
Raul Pohorille, New Rochelle
Juana Toporovsky, Bronx

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Antonio F. LaSorte, Binghamton
Theodore R. Poritz, Binghamton
Richard M. Simon, Johnson City

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Zahid M. Chohan, Olean

Cayuga (*1*)
Richard J. Freeman, Auburn

Chautauqua (*2*)
Bert W. Rappole, Jamestown
Dana M. Wheelock, Dunkirk

Chemung (*2*)
Donald L. Brooks, Elmira
Gerald P. Schneider, Elmira

Chenango (*1*)
Norman C. Lyster, Norwich

Clinton (*2*)
Stephen P. Nicknish, Plattsburgh

Columbia (*1*)
Jeffrey I. Monkash, Hudson

Cortland (*1*)
S. Robert Paik, Cortland

Delaware (*1*)
Gary Preiser, Walton

Dutchess (*4*)
Eric R. Brocks, Fishkill
Stephen R. Chernay, Fishkill
Eleanor C. Kane, Poughkeepsie
Florian M. Cortese, Hyde Park

Erie (*9*)
Victorino Anllo, Kenmore
Thomas W. Bradley, Kenmore
Edmond J. Gicewicz, Buffalo
Ross L. Guarino, Buffalo
Allen L. Lesswing, Buffalo
Leo E. Manning, Amherst
M. Luther Musselman, Buffalo
Nancy H. Nielsen, Orchard Park
James F. Phillips, Buffalo

Essex (*1*)
Glen D. Chapman, Ticonderoga

Franklin (*1*)
Alfred A. Hartmann, Jr., Malone

Fulton (*1*)

Genesee (*1*)
Bruce E. Baker, LeRoy

Greene (*1*)
Karuvath Enu, Hudson

Herkimer (*1*)
Krishna K. Vadlamudi, Ilion

Jefferson (*2*)
W. Bruce Carter, Watertown
David O. VanEenenaam, Watertown

Kings (*19*)
Judithann Anderson, Brooklyn
Norman S. Blackman, Brooklyn
Randall D. Bloomfield, Brooklyn
Joseph R. Brennan, Brooklyn
George W. Calvosa, Brooklyn

Vernal G. Cave, Brooklyn
Duncan W. Clark, Brooklyn
Clifford Cohen, Brooklyn
Vashti R. Curlin, Broooklyn
Paul Finkelstein, Brooklyn
Ladislav P. Hinterbuchner, Rye
Iraj Iraj, Brooklyn
A. W. Martin Marino, Jr., Brooklyn
Bentley D. Merrim, Brooklyn
Hubert S. Pearlman, Brooklyn
Abdul Rehman, Staten Island
Veronica Santilli, Brooklyn
Leo J. Swirsky, Brooklyn

Lewis (*1*)
John C. Herrman, Lowville

Livingston (*1*)
Sheppard Arluck, Geneseo

Madison (*1*)
Leonard A. Argentine, Oneida

Monroe (*7*)
Marilyn R. Brown, Rochester
Arthur J. Florack, Rochester
William A. Kern, Jr., Rochester
Mario B. LoMonaco, Rochester
Joyce M. McChesney, Rochester
Carl C. Sansocie, Brockport
Edward C. Tanner, Rochester

Montgomery (*1*)
Feori F. Pipito, Amsterdam

Nassau (*11*)
Robert Bruce Bergmann, Massapequa
Lee S. Binder, Valley Stream
Paul B. Bedrossian, Rockville Centre
Joseph I. Cohn, Rockville Centre
Paul A. Hamlin, Great Neck
Arnold D. Lurie, Baldwin
Felix A. Monaco, Rockville Centre
John A. Ostuni, Freeport
Henry S. Partridge, Freeport
E. John Pesiri, Massapequa
Walter A. Trenkle, Mineola

New York (*23*)
Arnold L. Bachman, New York
Melvin H. Becker, New York
Robert S. Bernstein, New York
Michael B. Brodin, New York
Joseph B. Cleary, New York
Irwin J. Cohen, New York
Hugh Clark Davidson, New York
Sally Faith Dorfman, New York
Jonas M. Goldstone, New York
Wilbur J. Gould, New York
George Hyams, New York
Virginia Kanick, New York
Vance Lauderdale, Jr., New York
Samuel H. Madell, New York
Aaron G. Meislin, New York
Alfred Moldovan, New York
Donna B. O'Hare, New York
Richard N. Pierson, Jr., New York
William B. Rawls, Pleasant Valley
William B. Rosenblatt, New York
Clifford L. Spingarn, New York

Niagara (*3*)
Mary Ann Z. Bishara, Lewiston
Claus M. Fichte, Niagara Falls
James J. Kropelin, Niagara Falls

Oneida (*4*)
Gregory B. Brown, Utica
Robert M. George, Utica
Neville W. Harper, Rome
Sheldon Waldman, Rome

Onondaga (*6*)
Daniel L. Dombroski, Syracuse
John A. Hoepner, Syracuse
Richard A. Konys, Syracuse
Ronald A. Naumann, Syracuse
Frank J. Piper, Syracuse
George P. Tilley, Syracuse

Ontario (*2*)
Verne M. Marshall, Geneva
Kenneth T. Steadman, Geneva

Orange (*4*)
Jacob J. Barie, Middletown
Stanley F. Brunn, Port Jervis
Michael A. Parmer, Port Jervis
Seymour J. Weiner, Port Jervis

Orleans (*1*)
David B. L. Meza III, Albion

Oswego (*1*)

Otsego (*1*)
Rodman D. Carter, Cooperstown

Putnam (*1*)

Queens (*16*)
Marvin L. Blumberg, Jamaica
Daniel Chansky, Forest Hills
John W. V. Cordice, Jr., St. Albans
Daniel G. Deckler, Richmond Hill
Alexander M. De la Garza, Flushing
Fred S. Fensterer, Glendale
Fred N. Flatau, Flushing
Lorraine M. Giordano, Jamaica Estates
Victor Guarneri, Bayside
Nicetas H. Kuo, Great Neck
Maxwell J. Marder, Forest Hills
Frank Ratner, Flushing
Hilda Ratner, Beechurst
Nessim Romano, Forest Hills
Sheldon D. Sax, Whitestone
Ezra A. Wolff, Manhasset

Rensselaer (*2*)
William A. McCarty, Troy
William A. Whyland, Troy

Richmond (*4*)
Albert B. Accettola Jr., Staten Island
Robert Bonvino, Staten Island
Tano S. Carbonaro, Staten Island
Gerald J. Lustig, Staten Island

Rockland (*4*)
Herbert Z. Geller, New City
Roy H. Lieberman, Nanuet
Michael Kalvert, New City
Stanley H. Oransky, Bardonia

St. Lawrence (*1*)
Brian R. McMurray, Canton

Saratoga (*1*)
Allen T. Jones, Saratoga Springs

Schenecctady (*3*)
Richard H. Lange, Schenectady
John M. Spring, Schenectady
Donald Wexler, Schenectady

Schoharie (*1*)
Frederick J. Michel, Cobleskill

Schuyler (*1*)

Seneca (*1*)
Donald W. Bidwell, Seneca Falls

Steuben (*2*)
Jack D. O'Neil, Corning
C. Susan Rainwater, Hornell

Suffolk (*11*)
Christopher J. Beatty, Port Jefferson
William H. Bloom, Bay Shore
Stephen I. Braitman, Islip Terrace
Clive D. Caplan, Islip Terrace
Frank R. Collier, Port Jefferson
Robert A. Held, Islip Terrace
Elise H. Korman, Southampton
Kenneth B. Levites, West Sayville
Philip A. Mantia, Smithtown
Stanley A. Steckler, Smithtown
William H. Weir, Jr., Riverhead

Sullivan (*1*)
Paul D. Salzberg, Callicoon

Tioga (*1*)

Tompkins (*2*)
Robert A. Hesson, Ithaca
Elliot Rubinstein, Ithaca

Ulster (*2*)
John V. Ioia, Kingston
Geraldine T. Keyes, Stone Ridge

Warren (*2*)
Richard Hughes, Sr., Glens Falls
Robert A. Reid, Glens Falls

Washington (*1*)
Nadav Sharon, Cambridge

Wayne (*3*)
William J. Braell, Palmyra
David T. Hannan, Newark
Daniel J. Joretz, Ontario

Westchester (*9*)
Ann C. Cea, Rye Brook
Mark L. Fox, Bonxville
Sanford A. Kaplan, Bronxville
Thomas A. Lansen, Valhalla
Stanley M. Mendelowitz, Tarrytown
Toni G. Novick, New Rochelle
Stephen O. Schwartz, White Plains
Pellegrino J. Tozzo, New Rochelle
William J. Walsh, Jr., White Plains

Wyoming (*1*)

Yates (*1*)
Michael J. Boquard, Penn Yan

Medical School Delegates (12)
Albany Medical—Henry S. Pohl, Albany
Albert Einstein College of Medicine of Yeshiva University of New York—Melvin N. Zelefsky, Bronx
Columbia University College of Physicians and Surgeons—
Cornell University Medical College—
Mount Sinai School of Medicine of the City University of New York—
New York Medical College—Marilyn Pearl, Great Neck
New York University of Medicine—Stanford Wessler, New York
State University of New York Health Science Center at Brooklyn—
State University of New York at Buffalo School of Medicine—Robert L. Scheig, Erie
State University of New York at Stony Brook School of Medicine—Dorothy S. Lane, Suffolk
State University of New York Health Science Center at Syracuse—Patricia A. Randall, Onondaga
University of Rochester School of Medicine and Dentistry—Thomas E. Cardillo, Monroe

Medical Student Delegates (3)
Paul A. Pipia, New York
Howard Pomeranz, New York
Victor Sloan, New York

Resident Membership Delegate (1)
Malcolm Reid, Westchester

Hospital Medical Staff Delegate (1)
Richard J. Dwane, Cayuga

REFERENCE COMMITTEES

Reports of Officers and Administrative Matters

Rodman D. Carter, Otsego, *Chairman*
Ann C. Cea, Westchester
George L. Ginsberg, New York State Psychiatric Association
Norman R. Loomis, Seventh District Branch
William B. Rosenblatt, New York
Paul A. Hamlin, Nassau (Observer)

Bylaws

Robert A. Held, Suffolk, *Chairman*
John C. Hermann, Lewis
Sanford A. Kaplan, Westchester
Raul Pohorille, The Bronx
Hilda Ratner, Queens
Anton A. Vreede, Sixth District Branch (Observer)

Governmental Affairs and Legal Matters

Eleanor C. Kane, Dutchess, *Chairwoman*
Roger D. Billig, First District Branch
Fred N. Flatau, Queens
Louis W. Giordano, Broome
Nancy H. Nielsen, Erie
Robert A. Hesson, Tompkins (Observer)

Scientific and Educational Activities

Randall D. Bloomfield, Kings, *Chairman*
Bruce H. Berlin, Second District Branch
Stanley H. Oransky, Rockland
Howard Pomeranz, Medical Student Membership
Gary Preiser, Delaware
Henry S. Pohl, Albany Medical College (Observer)

Socio-Medical Economics

Irwin J. Cohen, New York, *Chairman*
Gregory B. Brown, Oneida
Stanley F. Brunn, Orange
Veronica Santilli, Kings
James F. Wright, New York State Academy of Family Physicians
Mary Ann Z. Bishara, Niagara (Observer)

CONVENTION COMMITTEES

Credentials

A. W. Martin Marino, Kings, *Chairman*
Elise H. Korman, Suffolk
Antonio F. LaSorte, Broome
E. John Pesiri, Nassau
Seymour J. Weiner, Orange
Glen D. Chapman, Essex (Observer)

Rules and Order of Business

Ezra A. Wolff, Queens, *Chairman*
Stephen R. Chernay, Dutchess
Joseph B. Cleary, New York
Richard Hughes Sr., Warren
Ronald Naumann, Onondaga
Venkatachala Pathy, Bronx (Observer)

Sergeant at Arms

Allen Lesswing, Erie, *Chairman*
William J. Braell, Wayne
Tano Carbonaro, Richmond
Arthur J. Florack, Monroe
Michael Kalvert, Rockland
Donald L. Levin, New York State Society of Dermatology (Observer)

Tellers

Victorino Anllo, Erie, *Chairman*
Albert Accetolla, Jr., Richmond
Sana Bloch, The Bronx
Jonas Goldstone, New York
S. Robert Paik, Cortland
Christopher Beatty, Suffolk (Observer)

NOMINATING COMMITTEE

To the House of Delegates, Ladies & Gentlemen:

In accordance with Article XI, Section 2 of the Bylaws of the Medical Society of the State of New York, the Nominating Committee met at the Society's headquarters, 420 Lakeville Road, Lake Success, New York, on Wednesday, November 9, 1988 at 11:00 a.m.

Members of the Committee present were:
Member-at-Large—**Samuel M. Gelfand, M.D.,** Nassau, *Chairman*
First District Branch—**John A. Finkbeiner, M.D.,** New York
Second District Branch—**Robert E. Fear, M.D.,** Suffolk
Third District Branch—**Albert M. Ellman, M.D.,** Albany
Fourth District Branch—**Kirk R. Panneton, M.D.,** Schenectady
Fifth District Branch—**Theodore C. Max, M.D.,** Madison
Sixth District Branch—**Antonio F. LaSorte, M.D.,** Broome
Seventh District Branch—**Michael J. Graney, M.D.,** Monroe
Eighth District Branch—**James H. Cosgriff, Jr., M.D.,** Erie
Ninth District Branch—**Seymour R. Stall, M.D.,** Dutchess
Member-at-Large—**James M. Flanagan, M.D.,** Wayne

After careful consideration of the recommendations submitted by the district branches, the county medical societies, and the resident and student membership in response to a memorandum, dated August 19, 1988, your Committee respectfully nominated the following candidates for election on April 9, 1989:

President—**David M. Benford, M.D.,** New York
President-Elect—**Robert A. Mayers, M.D.,** Westchester
Vice-President—**Charles N. Aswad, M.D.,** Broome
Secretary—**George Lim, M.D.,** Oneida
Assistant Secretary—**Joyce M. McChesney, M.D.,** Monroe
Treasurer—**Morton Kurtz, M.D.,** Queens
Assistant Treasurer—**James H. Cosgriff, Jr., M.D.,** Erie
Speaker—**Seymour R. Stall, M.D.,** Dutchess
Vice-Speaker—**John H. Carter, M.D.,** Albany
Councilors: (*four for three years*)—**Robert E. Fear, M.D.,** Suffolk
—**Stanley L. Grossman, M.D.,** Orange
—**Thomas D. Pemrick, M.D.,** Rensselaer
—**Ralph E. Schlossman, M.D.,** Queens
Resident Councilor: (*one for one year*)—**John F. Piper, M.D.,** Onondaga
Student Councilor: (*one for one year*)—* **Tanya Lumpkins,** New York
Trustees: (*two for five years*)—**Samuel M. Gelfand, M.D.,** Nassau
—**M. Theodore Tanenhaus, M.D.,** Kings

Delegates to the American Medical Association for two years commencing January 1, 1990:
David M. Benford, M.D., New York
John H. Carter, M.D., Albany
Ann Cea, M.D., Westchester
Duncan W. Clark, M.D., Kings
James H. Cosgriff, Jr., M.D., Erie
John A. Finkbeiner, M.D., New York
James M. Flanagan, M.D., Wayne
Samuel M. Gelfand, M.D., Nassau
Robert M. George, M.D., Oneida
Morton M. Kurtz, M.D., Queens
Allison B. Landolt, M.D., Westchester
Antonio F. LaSorte, M.D., Broome
George Lim, M.D., Oneida
* **Tanya Lumpkins,** New York
David B. L. Meza, III, M.D., Orleans
Daniel F. O'Keeffe, M.D., Warren
Stuart I. Orsher, M.D., New York
Bernard J. Pisani, M.D., New York
Patricia A. Randall, M.D., Onondaga
Charles D. Sherman, Jr., M.D., Monroe
Seymour R. Stall, M.D., Dutchess
* **Penny Stern,** The Bronx
M. Theodore Tanenhaus, M.D., Kings
Victor J. Tofany, M.D., Monroe

The twelve nominees receiving the largest number of votes will be delegates; the second twelve will be alternates.

Respectfully submitted:
Samuel M. Gelfand, M.D., *Chairman*

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* Medical Student

HOUSE COMMITTEE ON BYLAWS

To the House of Delegates, Ladies and Gentlemen:

The members of the House Committee on Bylaws are as follows:

George Lim, M.D., Chairman	Oneida
Marilyn R. Brown, M.D.	Monroe
Stanley F. Brunn, M.D.	Orange
Rodman D. Carter, M.D.	Otsego
Robert M. Kohn, M.D.	Erie
Sidney Mishkin, M.D.	Nassau
Donna B. O'Hare, M.D.	New York
Hilda Ratner, M.D.	Queens
Stanley A. Steckler, M.D.	Suffolk
Charles D. Sherman, M.D., President, ex officio	Monroe
John H. Carter, M.D., Secretary, ex officio	Albany
Charles N. Aswad, M.D., Speaker, ex officio	Broome
Donald F. Foy	Executive Vice President, ex officio
Robert J. O'Connor, M.D.	Deputy Executive Vice President, ex officio
Donald R. Moy, J.D.	General Counsel, ex officio

A meeting of the Reference Committee on Bylaws was held at the Hilton Hotel in New York City on April 22, 1988. The Reference Committee prepared a transcript of testimony and a summary of its recommendations to the House Committee on Bylaws. The input received from the meeting of the Reference Committee on Bylaws proved to be of great assistance to the House Committee on Bylaws when it met on December 22, 1988.

In all, eight resolutions asking for amendments to the Bylaws were referred to the Committee for its consideration.

Resolution 88-2, Expenditure for the Office of President

Introduced by the Board of Trustees

Whereas, An ad hoc committee of the Board of Trustees developed administrative guidelines and recommendations relative to the per diem allowance and reimbursement of expenses for the president and president-elect of the Society; and

Whereas, The Committee also considered alternatives to the present per diem allowance; and

Whereas, After reviewing the compensation arrangements of several large State Medical Associations and the officer honorarium and expense survey of the American Association of Medical Society Executives, the Board concluded that a per diem allowance, plus expenses, is the most equitable arrangement because of the variations in time devoted by different presidents to the duties of the Office; and

Whereas, In order to meet annual budgetary objectives, the Board recommends the establishment of a limit on the Society's total expenditures for the Office of President; therefore be it

Resolved, That Article XVI, paragraph 2 of the Bylaws of the Medical Society of the State of New York be amended to read as follows: (additions italicized)

"The president and president-elect shall be allowed a per diem and expenses when engaged on official business. The amount of the per diem shall be set by the House of Delegates. *The Board of Trustees shall each year place a limit on the Society's total annual expenditure for the Office of President.*

After considerable discussion, the House Committee on Bylaws agreed to amend the Resolution by adding the words "and President-Elect" after the phrase "Office of President." The Resolved would then read: (additions to the current Bylaws italicized)

"The president and president-elect shall be allowed a per diem and expenses when engaged on official business. The amount of the per diem shall be set by the House of Delegates. *The Board of Trustees shall each year place a limit on the Society's total annual expenditure for the Office of President and President-Elect."*

The House Committee on Bylaws agreed that the amendment will give the Board of Trustees added flexibility in managing the fiscal affairs of the Medical Society in a prudent manner.

Therefore, the Committee unanimously recommends that Resolution 88-2 be **ADOPTED AS AMENDED.**

Resolution 88-8, Dues for Members In Part-Time Practice

Introduced by Ladislav P. Hinterbuchner, M.D., as an Individual

Whereas, There are active members of the Society who are in part-time private practice; and

Whereas, There is currently no provision in the Bylaws of the Medical Society of the State of New York to permit reduction of dues for members who have attained a certain age and are not in full-time private practice; and

Whereas, Such reduction of dues is just but should be accomplished without creating a new category of members; therefore be it

Resolved, That Article XV, paragraph 3 of the Bylaws of the Medical Society of the State of New York be amended to read as follows: (additions italicized)

The dues and assessments of those active members who have been graduated from medical or osteopathic college not more than five calendar years, not counting active service in the armed forces of the United States or in the United States Public Health Service, shall be one third the amount levied on all other active members. The dues and assessments of those active members who have not completed their continuous residency training shall be one tenth the amount levied on all other active members. In no case may an active physician continue to pay such reduced dues more than seven years after the date of graduation from medical or osteopathic school. The dues and assessments of all active members entering practice for the first time, who are not otherwise eligible for reduced dues as set forth in prior provisions in this paragraph, shall be one half the amount levied on all other active members for the first two years of practice; full dues shall be paid after the first two years of practice; once an active member pays full dues, he shall no longer qualify for half-dues status. *The dues and assessments of an active member in good standing who has been a member for ten consecutive years, has attained the age of sixty seven and who is working exclusively in private practice no more than 30 hours per week may apply to the component county medical society of which he is a member for a reduction of one half of the dues levied on all other active members. His application shall be signed by the president and secretary of the component county medical society of which he is a member and sent to the secretary of the Medical Society of the State of New York for presentation to the Council for approval.*

The Reference Committee on Bylaws heard substantial testimony that Resolution 88-8 would have a serious adverse fiscal impact on the State and county medical societies. A review by staff supports this conclusion. Testimony was heard that the Resolution is unnecessary since the Bylaws of the Medical Society of the State of New York currently include provisions for dues remissions for hardship.

Accordingly, the House Committee on Bylaws unanimously recommends that Resolution 88-8 **NOT BE ADOPTED.**

Resolution 88-9, Mission Statement of the Medical Society of the State of New York

Introduced by the Council

Whereas, The Medical Society of the State of New York has developed a Strategic Plan which reflects the Society's realization that the organization must focus its activities and resources on those areas which are of most importance to its membership; and

Whereas, The Strategic Plan recognized the necessity of updating the mission statement of the Society to be more responsive to the current environment in which medical practice is being conducted, and the commitment of the Society to serve as a strong advocate for physician interests; therefore be it

Resolved, That Article I of the Bylaws of the Medical Society of the State of New York be amended to read as follows: (deletions in brackets, additions italicized)

The name and title of the State Society shall be the Medical Society of the State of New York. The purposes of the State Society shall be: [to federate into one organization the medical profession of the State of New York; to extend medical knowledge and advance the science and art of medicine; to establish the Principles of Professional Conduct which shall be binding on the members of the Medical Society of the State of New York, the members of the component county medical societies and the members of the component district branches, in all matters in their relation to each other and to the public; to enhance the delivery of medical care of high quality to all people in the most economical manner; to promote the betterment of community health; and to enlighten and direct public opinion in regard to the problems of medicine and health for the best interests of the people of the State.]

1. to contribute to the professional and personal development of member physicians by representing the profession as a whole and advocating health-related rights, responsibilities and issues. These actions are designed to promote a favorable environment for the practice of medicine and improvement of the health of the residents of New York State.

2. to serve as a resource for its members and assist them in addressing the many issues and needs which they face in providing health care to their patients.

3. to seek to educate the public regarding important health care issues, including identification and discussion of their implications for patients and physicians in the current medical practice environment.

4. to act to promote and maintain high standards in medical education and in the practice of medicine in an effort to ensure that quality medical care is available to the public.

5. to seek to attain these goals by acting, to the fullest extent possible, in concert with organizations and agencies which have similar aims.

After a thorough discussion, the House Committee on Bylaws agreed that Resolution 88-9 should be referred back to the Council for further evaluation. In re-evaluating Resolution 88-9, the Committee asks that the Council consider retaining:

1. The statement "to enhance the delivery of medical care of high quality to all people in the most economical manner," which is currently included in Article 1, in the revision. This statement should be consolidated with the statement "to act to promote and maintain high standards in medical education and in the practice of medicine in an effort to ensure that quality medical care is available to the public," that appears in paragraph 4 of the proposed revision. There was substantial agreement that the statement should appear as paragraph 1 and not as paragraph 4.

2. The statement "to federate into one organization the medical profession of the State of New York," in the revision.

Accordingly, the Committee unanimously recommends that Resolution 88-9 by **REFERRED** to the Council for further evaluation.

Resolution 88-11, Past Deputy Executive Vice-President of the State Society as a Member of the House of Delegates for Life

Introduced by the Council

Whereas, Any member of the Medical Society of the State of New York who has served as deputy executive vice-president of the State Society has gained valuable experience in dealing with the policies and issues concerning the medical profession; and

Whereas, This experience can be of great value to the House of Delegates of the Medical Society of the State of New York in its deliberations; and

Whereas, Article III, Section 1, paragraph 1 of the Bylaws of the Medical Society of the State of New York currently includes any past executive vice-president of the Medical Society of the State of New York as a member of the

House of Delegates for life; therefore be it

Resolved, That Article III, Section 1, paragraph 1 be amended by adding a new clause (i) to read:

"(i) any past deputy executive vice president of the State Society who has served a minimum of three years as deputy executive vice president, who shall be a member for life, provided that the individual is a member of the State Society, resides in the State of New York, is not otherwise a member of the House of Delegates for life in accordance with this section, and is elected as a member for life by a majority of the members of the House of Delegates present and voting."

Article III, Section 1, paragraph 1 shall then read: (additions italicized, deletions in brackets)

"The House of Delegates shall be composed of: (a) duly designated delegates from the component county medical societies; (b) officers of the Medical Society of the State of New York, councilors, and trustees; (c) a duly designated delegate from each district branch; (d) a duly designated delegate from each recognized specialty society; (e) duly designated delegates from the student membership; (f) the Commissioner of Health of the State of New York or a deputy designated by him, provided that any representative shall be a member of the State Society; (g) past-presidents of the State Society and any past president of the American Medical Association, provided that individual is a member of the Medical Society of the State of New York who shall be members for life; (h) any past executive vice-president of the State Society, who shall be a member for life, provided that the individual is a member of the Medical Society of the State of New York, resides in the State of New York and is not otherwise a member of the House of Delegates for life in accordance with this section; (i) *any past deputy executive vice-president of the State Society who has served a minimum of three years as deputy executive vice-president, who shall be a member for life, provided that the individual is a member of the State Society, resides in the State of New York, is not otherwise a member of the House of Delegates for life in accordance with this section, and is elected as a member for life by a majority of the members of the House of Delegates present and voting.* [(i)] (*j*) a representative from each of the medical schools in New York State, provided said representative is a member of the Medical Society of the State of New York; [(j)] (*k*) a delegate representing the resident membership; [(k)] (*l*) a delegate representing the hospital medical staff section and; [(l)] (*m*) elected officers, trustees, and speakers of the American Medical Association, provided those individuals are members of the Medical Society of the State of New York."

Article III, Section 1, paragraph 1 lists the composition of the House of Delegates. Subparagraphs (g) and (h) provide that certain individuals shall be members of the House of Delegates for life. Resolution 88-11 would expand the list of "life members" of the House of Delegates by adding a new subparagraph (i).

The House Committee on Bylaws does not feel that the list of life members of the House of Delegates should be expanded any further.

Accordingly, the Committee, by a majority vote, recommends that Resolution 88-11 **NOT BE ADOPTED.**

Resolution 88-12, Unified County-MSSNY-AMA Membership

Introduced by Medical Society of the County of Genesee, Inc., Medical Society of the County of Niagara, Inc., Medical Society of the County of Orleans, Inc.

Whereas, Medical organizations exist to serve physicians and their patients to ensure the continued quality of medical care; and

Whereas, In today's environment, the efficiency and effectiveness of these organizations is more important than ever; and

Whereas, The profession's efficiency and effectiveness is diminished by the fact that not all of the members of our Society are also members of the American Medical Association, the *only* organization which speaks for *all* physicians at the national level; and

Whereas, There are innumerable good and substantial reasons to unify medical societies both spiritually and financially; unification with the AMA provides benefits to all physicians; and

Whereas, The Medical Society of the State of New York has unified county-state medical society membership as a result from both an historical and statutory development; and

Whereas, There should be a recognized and effective mechanism to provide individual county medical societies with the option of unifying with the American Medical Association without affecting other component county medical societies; therefore be it

Resolved, That Article XVIII, Section 2, paragraph 1 of the Bylaws of the Medical Society of the State of New York be amended to read as follows: (additions italicized)

Eligibility for membership in a component county medical society shall be determined by that society. Except by the approval of the Council no physician shall be an active member in a component county medical society other than that of the county in which he practices or maintains legal residence. *Notwithstanding any provision in these Bylaws to the contrary, each component county medical society may establish membership in the American Medical Association as a requirement for membership in the county medical society.*

Article II, Section 1 of the Bylaws of the MSSNY provides that active members of the MSSNY "shall be all active members in good standing of the component county medical societies." In the past, the House of Delegates has voted **not** to amend Article II, Section 1 to require all active members of the MSSNY to be active members of the component county medical societies **and the American Medical Association.***

The federation of county medical societies and the State Medical Society is established by Section 2 of Article II and Article XV of the MSSNY Bylaws. Pursuant to these provisions, an active member of MSSNY must be an active member of a component county medical society in good standing.

If Resolution 88-12 is adopted, a physician who is not an AMA member may or may not qualify for active membership in MSSNY depending upon the county medical society he/she seeks to join.

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* *Resolution 79-2*

Accordingly, the Committee, by a majority vote, recommends that Resolution 88-12 **NOT BE ADOPTED.**

Resolution 88-52, Dues Waiver for Physicians Participating in Programs to Improve the Health Care of the Poor

Introduced by New York County Medical Society

Whereas, The goal of the New York City Homesteading Program, the Physician Loan Forgiveness Program of the State Education Department, and the National Health Service Corps of the United States Public Health Service is to increase the access of the poor and medically underserved to quality primary and preventive health care; and

Whereas, These three programs provide either some type of financial assistance or loan forgiveness for physicians who open a private practice in medically underserved areas; and

Whereas, The Medical Society of the State of New York encourages participation in the activities of organized medicine by these physicians who both provide necessary health care to the needy and promote the image of all physicans; therefore be it

Resolved, That Article XV, paragraph 3 of the Bylaws of the Medical Society of the State of New York be amended to read as follows: (additions italicized)

The dues and assessments of those active members who have been graduated from medical or osteopathic college not more than five calendar years, not counting active service in the armed forces of the United States or in the United States Public Health Service, shall be one third the amount levied on all other active members. The dues and assessments of those active members who have not completed their continuous residency training shall be one tenth the amount levied on all other active members. In no case may an active physician continue to pay such reduced dues more than seven years after the date of graduation from medical or osteopathic school. The dues and assessments of all active members entering practice for the first time, who are not otherwise eligible for reduced dues as set forth in prior provisions in this paragraph shall be one half the amount levied on all other active members for the first two years of practice; full dues shall be paid after the first two years of practice; once an active member pays full dues, he shall no longer qualify for half-dues status. *The dues and assessments of physicians who are participants in the New York City Homesteading Program, the Regent's Physician Loan Forgiveness Program of the State Education Department, and the National Health Service Corps of the United States Public Health Service shall be waived while the physicians are participants in the programs.*

and be it further

Resolved, That Article XV, paragraph 2 of the Bylaws of the Medical Society of the State of New York be amended to read as follows: (additions italicized)

Assessments can be levied by the House of Delegates or the Council and shall be payable within a period of time specified by them. *The Council, with the approval of the Board of Trustees, is authorized to waive the requirements regarding the payment of dues as provided in this article in the performance of any pilot membership recruitment projects.*

Fiscal note to implement the first resolved portion: None. It is anticipated that none of the participants in these programs are presently members.

The Committee believes that Resolution 88-52 should be amended by deleting the first Resolved, and by amending the second Resolved as follows: (additions to the current Bylaws are italicized)

Assessments can be levied by the House of Delegates or the Council and shall be payable within a period of time specified by them. *The Council, with the approval of the Board of Trustees and the affected county medical society, is authorized to waive, for a period not to exceed three years, the requirements regarding the payment of dues as provided in this article in the performance of any pilot membership projects.*

The first Resolved should be deleted because it would favor physicians participating in certain programs to the exclusion of others.

The Committee believes that the second Resolved, as amended, will strengthen the Council's authority to develop innovative membership programs.

Accordingly, the Committee recommends that Resolution 88-52 be **ADOPTED AS AMENDED.**

Resolution 88-64, Professional Courtesy

Introduced by The Council

Resolved, That Chapter 1 of the Principles of Professional Conduct of the Medical Society of the State of New York be amended by the addition of a new section 7 to read:

SECTION 7

"As a general rule, a physician should be discouraged from treating himself or members of his immediate family. Furthermore, a physician should be discouraged from prescribing or dispensing medication, particularly controlled substances, for his own use or for the use of members of his immediate family. Consequently, a physician is encouraged to cheerfully and without recompense give professional services to physicians or their dependents, if they are in his vicinity."

After a thorough discussion, the House Committee on Bylaws with the input of the Ethics Committee decided to present a substitute resolution to read as follows: (additions to the current Bylaws are italicized)

Section 7

A physician should consider it a privilege to offer treatment to a fellow physician and his immediate family. Therefore, he is encouraged to do so without consideration of compensation. This statement should not be construed as precluding third party payment.

The Committee believes the substitute resolution more adequately addresses the issue of "professional courtesy."

Accordingly, the Committee unanimously recommends that the Substitute Resolution be **ADOPTED.**

Resolution 88-83, Submission of Late Resolutions

Introduced by Hospital Medical Staff Section

Whereas, Under current MSSNY Bylaws there is no special provision for late submission of resolutions from the Hospital Medical Staff Section (HMSS) to the House of Delegates; and

Whereas, It has been found that the business of the hospital medical staffs and of the MSSNY House of Delegates is best served by holding the annual meeting of the HMSS in direct conjunction with the annual meeting of the MSSNY House of Delegates; therefore be it

Resolved, That Article III, Section 3, paragraph 4 of the Bylaws of the Medical Society of the State of New York be amended to read as follows: (additions italicized)

"Resolutions to be presented to the House of Delegates shall be forwarded to the speaker at least thirty days in advance of the annual meeting of the House of Delegates, *with the exception of resolutions submitted from the Hospital Medical Staff Section when its annual meeting is held in conjunction with that of the House of Delegates.* Resolutions may be presented by a voting delegate any time prior to the final day of the annual meeting of the House of Delegates but will be accepted for consideration by the House of Delegates only upon two-thirds vote of delegates present and voting."

and be it further

Resolved, That Article V, Section 2, paragraph 1 under the heading "Speaker" be amended to read as follows: (additions italicized)

"The speaker shall preside at all meetings of the House of Delegates. He shall serve on the Executive Committee with voice but without vote. He shall appoint all committees to serve during the meeting of the House of Delegates at least thirty days in advance of the meeting. All resolutions submitted by members of the House of Delegates (pursuant to Article III, Section 3, paragraph 3) to be presented to the House of Delegates shall be forwarded to the speaker at least thirty days in advance of the annual meeting of the House of Delegates and referred by him to the appropriate reference committees, *with the exception of resolutions submitted from the Hospital Medical Staff Section when its annual meeting is held in conjunction with that of the House of Delegates.*

Because of the scheduling of the annual meeting of the Hospital Medical Staff Section, the Committee agrees that Resolution 88-83 is necessary.

Accordingly, the Committee unanimously recommends that Resolution 88-83 be **ADOPTED**.

The medical student and resident physician organizations have requested that wherever appropriate in the MSSNY Bylaws, the phrases "medical student membership" and "resident membership" be deleted and replaced by the phrases "student section" and "resident physician section," respectively.

The House Committee on Bylaws believes that the changes are strictly editorial, and should be adopted without the need for a Bylaws resolution. At the time the medical student and resident physician components of the MSSNY were established, the Medical Society gave representation in the House of Delegates to Medical Specialty Sections. The terms "medical student membership" and "resident membership" were used in order to differentiate these groups from the specialty sections. Informally, medical students and resident physicians, as representative groups in the Medical Society, have always been referred to as sections.

The Committee recommends that the editorial changes be **ADOPTED**.

Your Chairman would like to thank, in addition to the members of the Committee and the physicians who testified before it, those members of staff who helped this Committee; Donald R. Moy, J.D., General Counsel, Jay Silverman, J.D., Legal Assistant, and Kathleen Wellborn for her secretarial assistance.

Respectfully submitted,

George Lim, M.D., *Chairman*

FOR MY PATIENTS

NUMBER 7/1989

BEWARE OF LEAD IN YOUR WATER

Dear Patient:

Since ancient Roman times, it has been known that lead is a poisonous chemical. If swallowed or inhaled, lead can cause serious damage to the brain, nervous system, and kidneys. This is especially true for infants and children under 6 years of age and to pregnant women. In the last few years, many thousands of children in New York State were found to have high levels of lead in their blood.

LEAD IS ALMOST EVERYWHERE

Most cases of lead poisoning occur in young children who live in old, run-down houses built before 1950. Some of them have the habit of eating old paint chips or plaster from the walls, window sills, radiators or ceilings. Others may inhale lead-containing dust, soil, or air — inside or outside their house.

Over the last ten years or so, strict federal controls on leaded gasoline and on the amount of lead permitted in indoor paint have greatly lowered the amounts of lead in air, dust, and soil around the home.

However, in the last few years 10-20% of young children who have too much lead in their blood have obtained it from drinking lead-containing water in their own homes.

HOW LEAD GETS INTO DRINKING WATER

The usual way that lead contaminates the water in the home is by "soft" acidic water corroding the lead pipes or lead solder which some buildings contain.

AGE OF THE HOUSE

Lead in drinking water is most often a problem in houses that are either very old or very new. Before 1930, most plumbing pipes contained lead. Copper pipes were used in most buildings after that time, but lead solder was still used by the plumbers. As a result, corrosion still caused lead contamination in many areas of the country. Water in houses **less than 5 years old** usually has high lead levels.

CHECKING YOUR WATER FOR LEAD

You can have your house water tested for lead — which you cannot see, taste, or smell. This can be done through a private, qualified testing laboratory or through your local health department.

Be suspicious of lead in your water if:

- you notice plumbing pipes which are dull-gray and scratch easily with a housekey.
- you see signs of corrosion such as rust-colored water or staining of dishes or laundry.
- your house was built after 1983.

Your local health department has a list of approved water-testing laboratories if you need it.

IMMEDIATE STEPS TO REDUCE LEAD

- Avoid drinking or cooking with water that has been in your plumbing system for more than 6 hours (such as overnight or during an entire work day). Before using it, let the ***cold*** water run for at least 3 minutes. Do this at all faucets in the house which are used for cooking or drinking water.
- Never cook with or drink water from the hot water tap because hot water dissolves lead more quickly than cold water. Never use the hot water tap for preparing baby formulas.
- If you have any plumbing work done, make sure only lead-free materials (pipes, solder, and flux) are used. This is in the laws of the federal and New York State governments as of 1986-1987.
- If you live in a high-rise apartment, flushing of the cold-water faucets may not remove all the lead that may be in the pipes. In this case, you may have to buy bottled water if tests of your tap water show a high lead level.
- Do **not** use carbon filters, sand filters, or cartridge filters because they don't remove lead from the water. And do **not** use water softeners because they can lead to lead problems.

MEDICAL SOCIETY OF THE STATE OF NEW YORK
ORGANIZED 1807

Office Handouts for Your Patients

This issue of the *New York State Journal of Medicine* contains another "For My Patients" letter, which appears on the preceding page.

Use this letter series to keep your patients informed about timely issues concerning diseases, treatments and health maintenance. Simply make photocopies of the page and distribute them to your patients with your newsletter or other correspondence, or place them in your reception area for your patients to read and take home.

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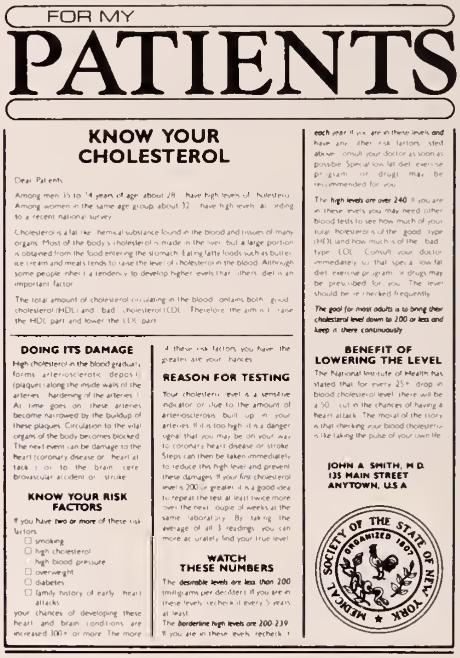

FOR MY

PATIENTS

KNOW YOUR CHOLESTEROL

Dear Patients

Among men 35 to 44 years of age about 28 have high levels of cholesterol. Among women in the same age group about 32 have high levels according to a recent national survey.

Cholesterol is a fat like chemical substance found in the blood and tissues of many organs. Most of the body's cholesterol is made in the liver but a large portion is obtained from the food entering the stomach. Eating fatty foods such as butter, ice cream and meats tends to raise the level of cholesterol in the blood. Although some people inherit a tendency to develop higher levels than others, diet is an important factor.

The total amount of cholesterol circulating in the blood contains both "good" cholesterol (HDL) and "bad" cholesterol (LDL). Therefore the aim is to raise the HDL part and lower the LDL part.

DOING ITS DAMAGE

High cholesterol in the blood gradually forms arteriosclerotic deposits (plaques) along the inside walls of the arteries (hardening of the arteries). As time goes on, these arteries become narrowed by the buildup of these plaques. Circulation to the vital organs of the body becomes blocked. The next event can be damage to the heart (coronary disease or "heart attack") or to the brain (cerebrovascular accident or "stroke").

KNOW YOUR RISK FACTORS

If you have **two or more** of these risk factors

☐ smoking
☐ high cholesterol
☐ high blood pressure
☐ overweight
☐ diabetes
☐ family history of early heart attacks

your chances of developing these heart and brain conditions are increased 300% or more. The more of these risk factors you have, the greater are your chances.

REASON FOR TESTING

Your cholesterol level is a sensitive indicator or clue to the amount of arteriosclerosis built up in your arteries. If it is too high it is a danger signal that you may be on your way to coronary heart disease or stroke. Steps can then be taken immediately to reduce this high level and prevent these damages. If your first cholesterol level is 200 or greater it is a good idea to repeat the test at least twice more over the next couple of weeks at the same laboratory. By taking the average of all 3 readings you can more accurately find your true level.

WATCH THESE NUMBERS

The **desirable levels are less than 200** (milligrams per deciliter). If you are in these levels, recheck it every 5 years at least.

The **borderline high levels are 200-239**. If you are in these levels, recheck it **each year**. If you are in these levels **and** have any other risk factors listed above, consult your doctor as soon as possible. Special low fat diet, exercise program, or drugs may be recommended for you.

The **high levels are over 240**. If you are in these levels you may need other blood tests to see how much of your total cholesterol is of the "good" type (HDL) and how much is of the "bad" type (LDL). Consult your doctor immediately so that special low fat diet, exercise program or drugs may be prescribed for you. The level should be rechecked frequently.

The goal for most adults is to bring their cholesterol level down to 200 or less and keep it there continuously.

BENEFIT OF LOWERING THE LEVEL

The National Institute of Health has stated that for every 25% drop in blood cholesterol level there will be a 50 cut in the chances of having a heart attack. The moral of the story is that checking your blood cholesterol is like taking the pulse of your own life.

JOHN A. SMITH, M.D.
135 MAIN STREET
ANYTOWN, U.S.A.

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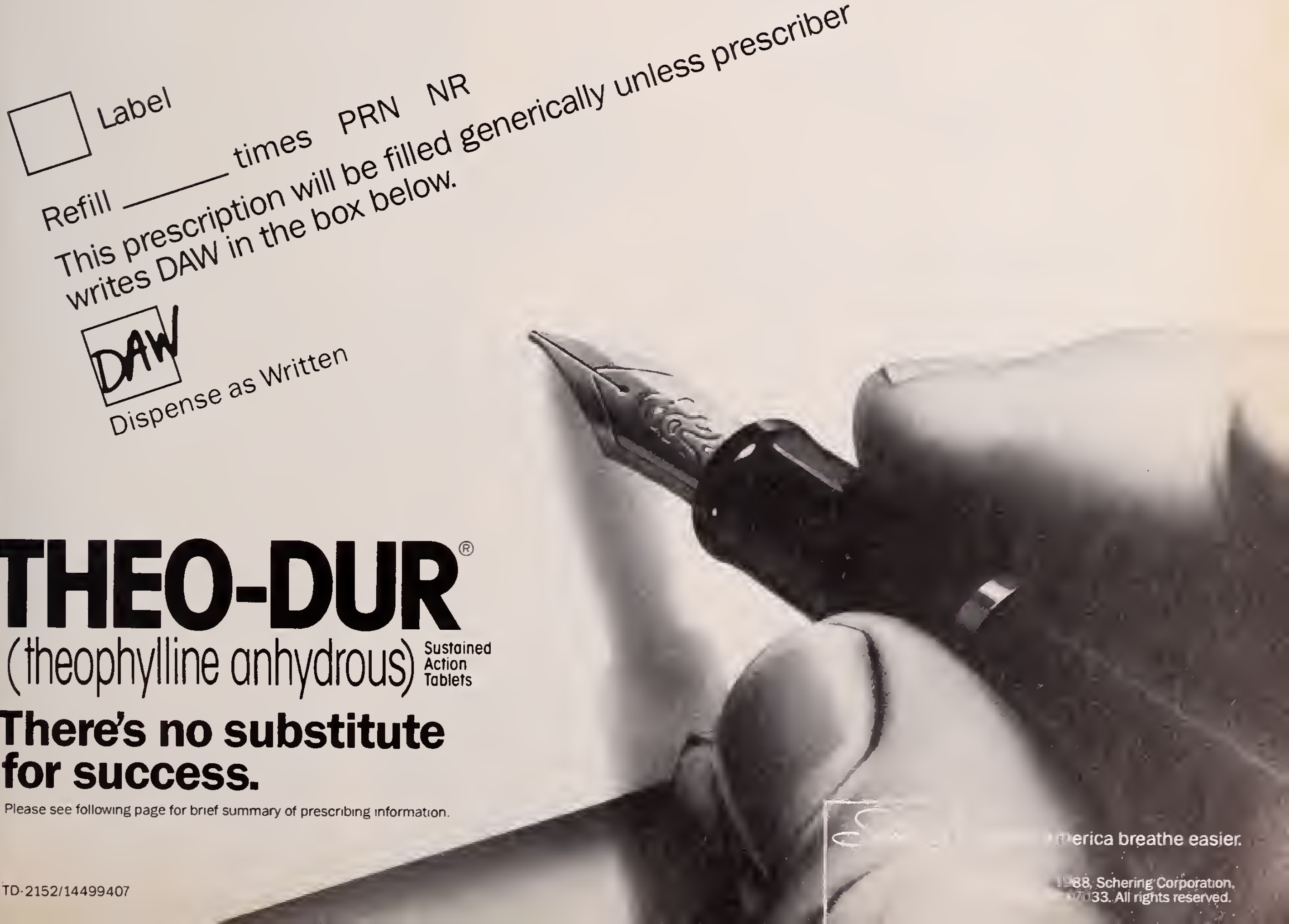

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INDICATIONS: THEO-DUR is indicated for relief and/or prevention of symptoms of asthma and for reversible bronchospasm associated with chronic bronchitis and emphysema
CONTRAINDICATIONS: THEO-DUR is contraindicated in individuals who have shown hypersensitivity to theophylline or any of the tablet components
WARNINGS: Status asthmaticus should be considered a medical emergency and is defined as that degree of bronchospasm which is not rapidly responsive to usual doses of conventional bronchodilators Optimal therapy for such patients frequently requires both *additional medication*, parenterally administered, and *close monitoring*, preferably in an intensive care setting

Although increasing the dose of theophylline may bring about relief, such treatment may be associated with toxicity The likelihood of such toxicity developing increases significantly when the serum theophylline concentration exceeds 20 mcg/ml Therefore determination of serum theophylline levels is recommended to assure maximal benefit without excessive risk

Serum levels above 20 mcg/ml are rarely found after appropriate administration of recommended doses However, in individuals in whom theophylline plasma clearance is reduced *for any reason*, even conventional doses may result in increased serum levels and potential toxicity Reduced theophylline clearance has been documented in the following readily identifiable groups: 1) patients with impaired renal or liver function, 2) patients over 55 years of age, particularly males and those with chronic lung disease, 3) those with cardiac failure from any cause, 4) neonates, and 5) those patients taking certain drugs (macrolide antibiotics and cimetidine) Decreased clearance of theophylline may be associated with either influenza immunization or active infection with influenza

Reduction of dosage and laboratory monitoring is especially appropriate in the above individuals Less serious signs of theophylline toxicity (i e nausea and restlessness) may occur frequently when initiating therapy, but are usually transient, when such signs are persistent during maintenance therapy, they are often associated with serum concentrations above 20 mcg/ml Unfortunately, however, serious side effects such as ventricular arrhythmias, convulsions or even death may appear as the first sign of toxicity without any previous warning Stated differently *serious toxicity is not reliably preceded by less severe side effects*

Many patients who require theophylline may exhibit tachycardia due to their underlying disease process so that the cause/effect relationship to elevated serum theophylline concentrations may not be appreciated

Theophylline products may cause dysrhythmia and/or worsen pre-existing arrhythmias and any significant change in rate and/or rhythm warrants monitoring and further investigation

The occurrence of arrhythmias and sudden death (with histological evidence of necrosis of the myocardium) has been recorded in laboratory animals (minipigs, rodents and dogs) when theophylline and beta agonists were administered concomitantly, although not when either was administered alone The significance of these findings when applied to human usage is currently unknown
PRECAUTIONS: THEO-DUR TABLETS SHOULD NOT BE CHEWED OR CRUSHED
General: Theophylline half-life is shorter in smokers than in non-smokers Therefore, smokers may require larger or more frequent doses Morphine and curare should be used with caution in patients with airway obstruction as they may suppress respiration and stimulate histamine release Alternative drugs should be used when possible Theophylline should not be administered concurrently with other xanthine medications Use with caution in patients with severe cardiac disease, severe hypoxemia, hypertension, hyperthyroidism, acute myocardial injury, cor pulmonale, congestive heart failure, liver disease, in the elderly (especially males) and in neonates In particular, great caution should be used in giving theophylline to patients with congestive heart failure Frequently, such patients have markedly prolonged theophylline serum levels with theophylline persisting in serum for long periods following discontinuation of the drug Individuals who are rapid metabolizers of theophylline, such as the young, smokers, and some non-smoking adults, may not be suitable candidates for once-daily dosing These individuals will generally need to be dosed at 12 hour or sometimes 8 hour intervals Such patients may exhibit symptoms of bronchospasm near the end of a dosing interval, or may have wider peak-to-trough differences than desired

Use theophylline cautiously in patients with history of peptic ulcer Theophylline may occasionally act as a local irritant to the G.I tract although gastrointestinal symptoms are more commonly centrally mediated and associated with serum drug concentrations over 20 mcg/ml
Information for Patients: The physician should reinforce the importance of taking only the prescribed dose and time interval between doses THEO-DUR tablets should not be chewed or crushed When dosing THEO-DUR on a once daily (q24h) basis, tablets should be taken whole and not split As with any controlled-release theophylline product, the patient should alert the physician if symptoms occur repeatedly, especially near the end of the dosing interval
DRUG INTERACTIONS: **Drug-Drug**: Toxic synergism with ephedrine has been documented and may occur with some other sympathomimetic bronchodilators In addition the following drug interactions have been demonstrated

Drug	Effect
Theophylline with lithium carbonate	Increased excretion of lithium carbonate
Theophylline with propranolol	Antagonism of propranolol effect
Theophylline with cimetidine	Increased theophylline blood levels
Theophylline with troleandomycin, erythromycin	Increased theophylline blood levels

Drug-Food: *THEO-DUR 100 mg Sustained Action Tablets* have not been adequately studied to determine whether their bioavailability is altered when given with food Available data suggest that drug administration at the time of food ingestion may influence the absorption characteristics of theophylline controlled-release products resulting in serum values different from those found after administration in the fasting state

A drug-food effect, if any, would likely have its greatest clinical significance when high theophylline serum levels are being maintained and/or when large single doses (greater than 13 mg/kg or 900 mg) of a controlled-release theophylline product are given

THEO-DUR (200, 300, and 450 mg) Sustained Action Tablets The rate and extent of absorption of theophylline from THEO-DUR 200 mg, 300 mg, and 450 mg tablets when administered fasting or immediately after a moderately high fat content breakfast is similar
Drug-Laboratory Test Interactions: When plasma levels of theophylline are measured by spectrophotometric methods, coffee, tea, cola beverages, chocolate, and acetaminophen contribute falsely high values
Carcinogenesis, Mutagenesis, and Impairment of Fertility. Long-term animal studies have not been performed to evaluate the carcinogenic potential, mutagenic potential, or the effect on fertility of xanthine compounds
Pregnancy: Category C—Animal reproduction studies have not been conducted with theophylline It is not known whether theophylline can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity Xanthines should be given to a pregnant woman only if clearly needed
Nursing Mothers: It has been reported that theophylline distributes readily into breast milk and may cause adverse effects in the infant. Caution must be used if prescribing xanthine to a mother who is nursing, taking into account the risk-benefit of this therapy
Pediatric Use. Safety and effectiveness of THEO-DUR administered
1 Every 24 hours in children under 12 years of age, have not been established
2 Every 12 hours in children under 6 years of age, have not been established
ADVERSE REACTIONS: The most consistent adverse reactions are usually due to overdose and are
1 *Gastrointestinal* nausea vomiting, epigastric pain, hematemesis, diarrhea
2 *Central nervous system* headaches, irritability, restlessness, insomnia, reflex hyperexcitability muscle twitching, clonic and tonic generalized convulsions
3 *Cardiovascular* palpitation, tachycardia extrasystoles, flushing hypotension, circulatory failure, ventricular arrhythmias
4 *Respiratory* tachypnea
5 *Renal* albuminuria, increased excretion of renal tubular and red blood cells, potentiation of diuresis
6 *Other* rash hyperglycemia and inappropriate ADH syndrome
OVERDOSAGE: **Management**. If potential oral overdose is established and seizure has not occurred
A Induce vomiting
B Administer a cathartic (this is particularly important if sustained-release preparations have been taken)
C Administer activated charcoal
If patient is having a seizure
A Establish an airway
B Administer oxygen
C Treat the seizure with intravenous diazepam, 0 1 to 0 3 mg/kg up to 10 mg
D Monitor vital signs, maintain blood pressure and provide adequate hydration
Post Seizure Coma
A Maintain airway and oxygenation
B If a result of oral medication follow above recommendations to prevent absorption of the drug but intubation and lavage will have to be performed instead of inducing emesis, and the cathartic and charcoal will need to be introduced via a large bore gastric lavage tube
C Continue to provide full supportive care and adequate hydration while waiting for drug to be metabolized In general, the drug is metabolized sufficiently rapid so as not to warrant consideration of dialysis however if serum levels exceed 50 mcg/ml charcoal hemoperfusion may be indicated
CAUTION: Federal law prohibits dispensing without prescription For full prescribing information, see package insert
Revised 6/87

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(*continued from p 13A*)

Distinguished Scholar Program. Living with Radiation: Risk/Benefit Equation. 14 Cat I Credits. Waldorf Astoria. *Mar 28–30.* **The Impact of Cardiac Surgery on the Quality of Life: Neurological and Psychological Aspects.** 17 Cat I Credits. New York Hilton. Contact: Ann J. Boehme, CMP, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

Mar 31. **Current Concepts in Asthma, Allergy and Pulmonary Disease.** 5 Cat I Credits. Beth Israel Medical Center, New York. Contact: Beverley R. Baptiste, Medical Education, Beth Israel Medical Center, First Ave at 16th St, New York, NY 10003. Tel: (212) 420-2849.

Mar 31. **Clinical Toxicology.** NYC Poison Control Center. Contact: Sue Callaghan, RN, MA, Bellevue Hospital Center, First Ave and 27th St, New York, NY 10016. Tel: (212) 561-6561.

AROUND THE NATION

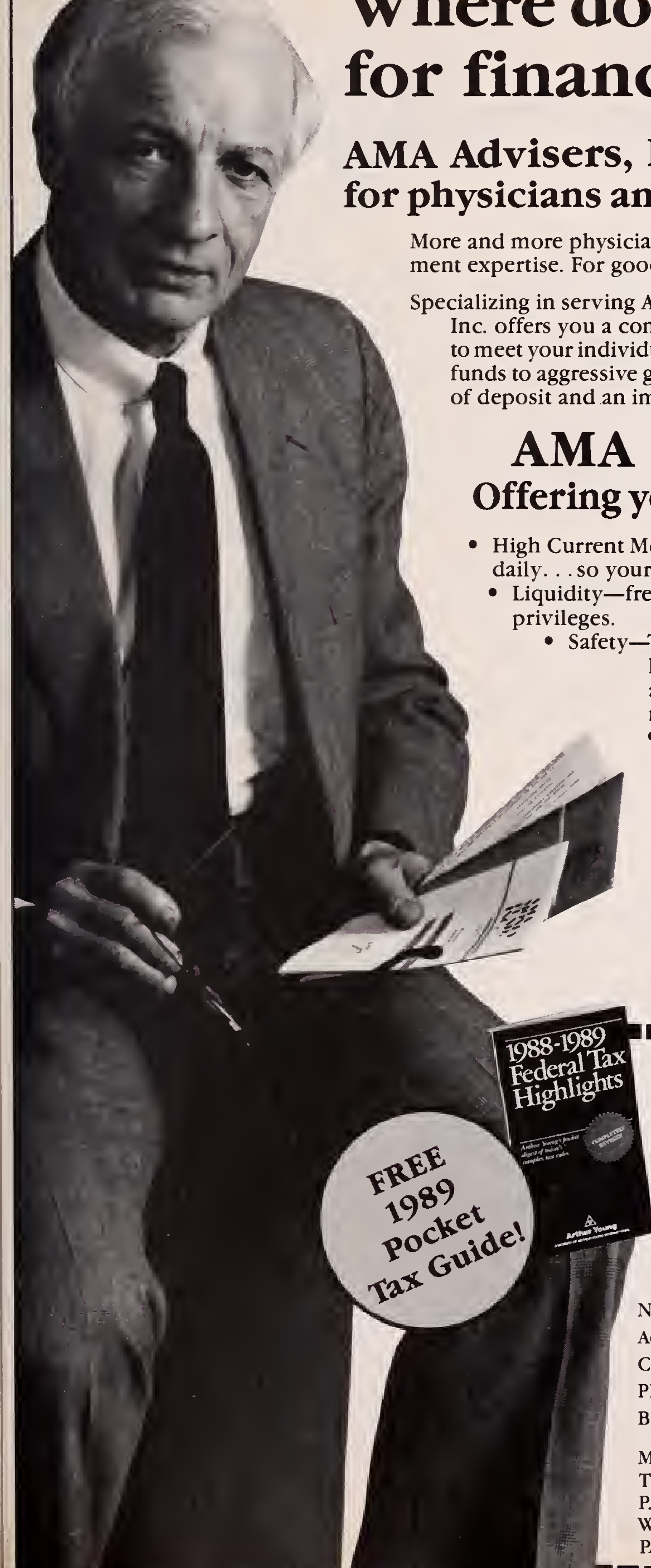
CALIFORNIA

Mar 5–9. **Thoracic Imaging '89.** 32 Cat I Credits. Hotel Del Coronado, Coronado (San Diego). *Mar 10–12.* **Musculoskeletal Imaging—The State of the Art.** 16 Cat I Credits. Four Seasons Hotel, Newport Beach. Contact: R. Dawne Ryals, Ryals and Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (404) 641-9773.

Mar 5–10. **Tenth Annual Mammoth Mountain Emergency Medicine Ski Conference.** 20 Cat I Credits. Mammoth Lakes. Contact: Daniel L. Abbott, MD, Medical Conferences, Inc, PO Box 52-B, Newport Beach, CA 92662. Tel: (714) 650-4156.

Mar 9–11. **46th Annual Meeting of the American Psychosomatic Society.** Ramada Renaissance, San Francisco. Contact: American Psychosomatic Society, 6728 Old McLean Village Dr, MacLean, VA 22101. Tel: (703) 556-9222.

(*continued on p 20A*)



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AIR FORCE

(*continued from p 18A*)

FLORIDA

Mar 2–4. **Infertility and Reproductive Endocrinology.** Sonesta Beach Hotel, Key Biscayne. Contact: American College of Obstetricians and Gynecologists, 409 12th St, SW, Washington, DC 20024-2188. Tel: (800) 533-8811.

March 13–15. **12th Annual Gold Coast Medicine Conference: Strategies for Healthy Aging . . . Must Our Aged Be Ill?** Good Samaritan Hospital, West Palm Beach. Contact: Laura J. Lyons, CME Coordinator, Good Samaritan Hospital, PO Box 3166, West Palm Beach, FL 33402. Tel: (407) 650-6236.

Mar 17–19. **The Clearwater Beach National Conference on Pediatrics.** Holiday Inn Surf Side, Clearwater. Contact: Dr Elliot F. Ellis, PO Box 5720, Jacksonville, FL 33247. Tel: (904) 390-3676.

HAWAII

Mar 30–Apr 1. **Pediatric Trends.** 16 Cat I Credits. Stouffer Waiohai Beach Resort, Kauai. Contact: CME Registration, American Academy of Pediatrics, PO Box 927, Elk Grove Village, IL 60009-0927. Tel: (312) 981-7884.

MARYLAND

Mar 20–21. **13th Annual Meeting of the American Society of Preventive Oncology.** Hyatt Regency, Bethesda. Contact: Richard R. Love, MD, American Society of Preventive Oncology, 1300 University Ave-7 C, Madison, WI 53706. Tel: (608) 263-6919.

UTAH

Mar 5–10. **Magnetic Resonance Imaging Update 1989—Winter Conference.** The Cliff Lodge, Snowbird. Contact: Radiology Postgraduate Education, Registration Office, University of California, San Francisco, CA 94143-0628. Tel: (415) 476-5731.

AROUND THE WORLD

FRANCE

Mar 4–11. **Seventh Winter Congress. Diagnostic Imaging.** Courchevelle, France. Contact: Winter Congress, 21915 Roscoe Blvd, Suite 222, Canoga Park, CA 91304. Tel: (818) 700-9821.

GREECE

Mar 20–Apr 1. **Oncogenes and Onco-Suppressor Genes.** Athens. Contact: American Health Foundation, 320 E 43rd St, New York, NY 10017. Tel: (212) 953-1900.

ITALY

Mar 15–18. **Symposium on Steroid Formation, Degradation and Action in Peripheral Tissues.** Taormina. Contact: Conference Director, The New York Academy of Sciences, 2 East 63rd St, New York, NY 10021. □

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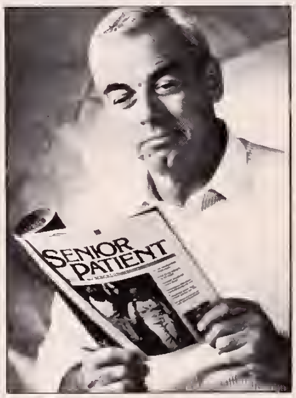

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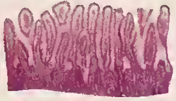
Aspirin and other nonsteroidal anti-inflammatory drugs weaken mucosal defenses, which may lead NSAID users to become prone to duodenal ulcers.[1] For those NSAID users who do develop duodenal ulcers, CARAFATE® (sucralfate/Marion) is ideal first-line therapy. Carafate rebuilds mucosal  defenses through a unique, nonsystemic mode of action. Carafate enhances the body's natural healing ability while it protects damaged mucosa from further injury. So the next time you see an arthritis patient with a duodenal ulcer, prescribe nonsystemic Carafate: therapy for the ulcer-prone patient.

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BRIEF SUMMARY

CONTRAINDICATIONS
There are no known contraindications to the use of sucralfate

PRECAUTIONS
Duodenal ulcer is a chronic, recurrent disease While short-term treatment with sucralfate can result in complete healing of the ulcer, a successful course of treatment with sucralfate should not be expected to alter the post-healing frequency or severity of duodenal ulceration.

Drug Interactions: Animal studies have shown that simultaneous administration of CARAFATE (sucralfate) with tetracycline, phenytoin, digoxin, or cimetidine will result in a statistically significant reduction in the bioavailability of these agents. The bioavailability of these agents may be restored simply by separating the administration of these agents from that of CARAFATE by two hours. This interaction appears to be nonsystemic in origin, presumably resulting from these agents being bound by CARAFATE in the gastrointestinal tract. The clinical significance of these animal studies is yet to be defined However, because of the potential of CARAFATE to alter the absorption of some drugs from the gastrointestinal tract, the separate administration of CARAFATE from that of other agents should be considered when alterations in bioavailability are felt to be critical for concomitantly administered drugs.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Chronic oral toxicity studies of 24 months' duration were conducted in mice and rats at doses up to 1 gm/kg (12 times the human dose). There was no evidence of drug-related tumorigenicity. A reproduction study in rats at doses up to 38 times the human dose did not reveal any indication of fertility impairment. Mutagenicity studies were not conducted

Pregnancy: Teratogenic effects Pregnancy Category B Teratogenicity studies have been performed in mice, rats, and rabbits at doses up to 50 times the human dose and have revealed no evidence of harm to the fetus due to sucralfate There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when sucralfate is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in children have not been established

ADVERSE REACTIONS
Adverse reactions to sucralfate in clinical trials were minor and only rarely led to discontinuation of the drug In studies involving over 2,500 patients treated with sucralfate, adverse effects were reported in 121 (4 7%).

Constipation was the most frequent complaint (2.2%). Other adverse effects, reported in no more than one of every 350 patients, were diarrhea, nausea, gastric discomfort, indigestion, dry mouth, rash, pruritus, back pain, dizziness, sleepiness, and vertigo.

OVERDOSAGE
There is no experience in humans with overdosage Acute oral toxicity studies in animals, however, using doses up to 12 gm/kg body weight, could not find a lethal dose. Risks associated with overdosage should, therefore, be minimal

DOSAGE AND ADMINISTRATION
The recommended adult oral dosage for duodenal ulcer is 1 gm four times a day on an empty stomach

Antacids may be prescribed as needed for relief of pain but should not be taken within one-half hour before or after sucralfate.

While healing with sucralfate may occur during the first week or two, treatment should be continued for 4 to 8 weeks unless healing has been demonstrated by x-ray or endoscopic examination.

HOW SUPPLIED
CARAFATE (sucralfate) 1-gm tablets are supplied in bottles of 100 (NDC 0088-1712-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1712-49). Light pink scored oblong tablets are embossed with CARAFATE on one side and 1712 bracketed by C's on the other
Issued 1/87

Reference:
1. Eliakim R, Ophir M, Rachmilewitz D: *J Clin Gastroenterol* 1987, 9(4):395-399

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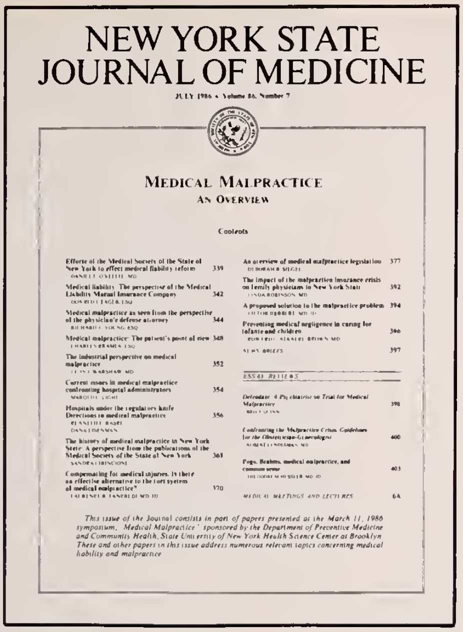

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JOURNAL OF MEDICINE

JULY 1986 • Volume 86, Number 7

MEDICAL MALPRACTICE
AN OVERVIEW

NEW YORK STATE
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SEPTEMBER 1986 • Volume 86, Number 8

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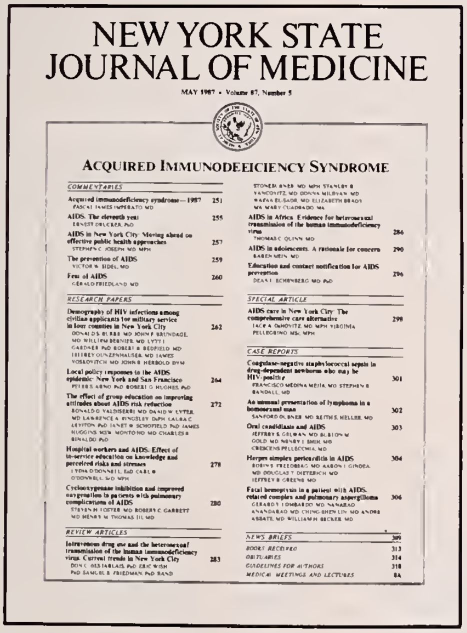

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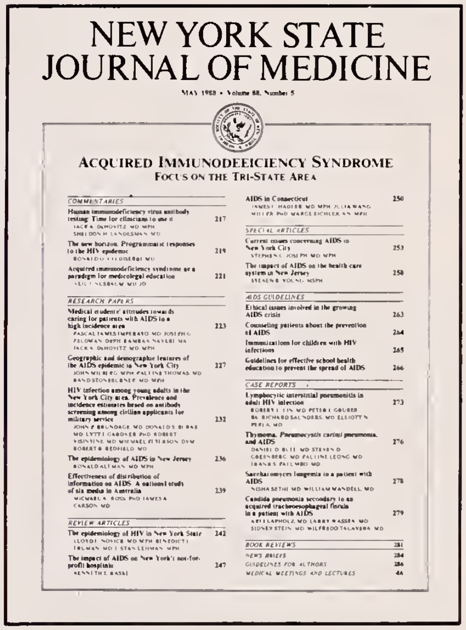

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Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:
1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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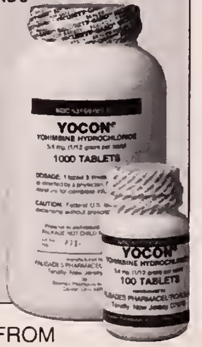

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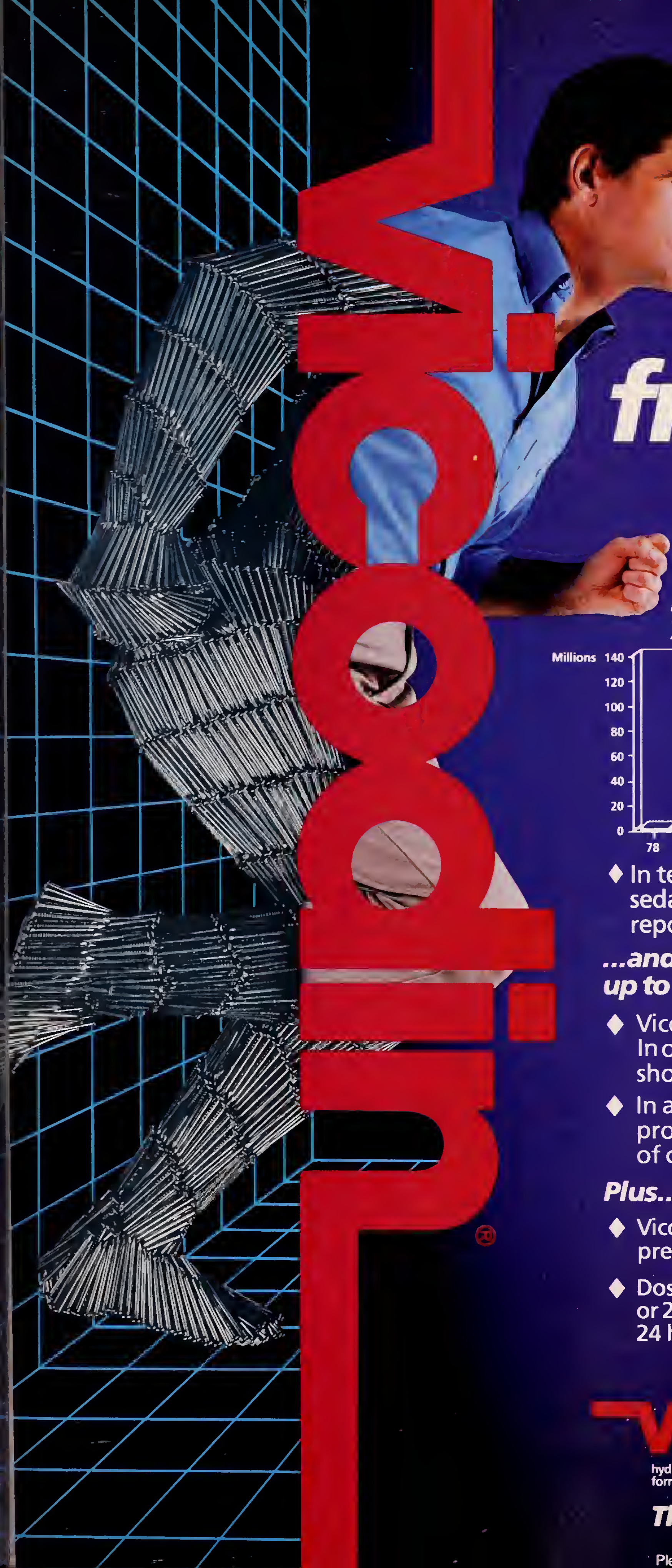

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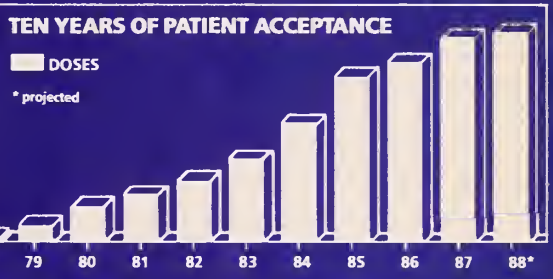

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1. Data on file, Knoll Pharmaceuticals.
2. Hopkinson JH III. *Curr Ther Res* 24:503-516, 1978
3. Beaver WT. *Arch Intern Med*, 141:293-300, 1981.

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INDICATIONS AND USAGE: For the relief of moderate to moderately severe pain.
CONTRAINDICATIONS: Hypersensitivity to acetaminophen or hydrocodone.
WARNINGS:
Allergic-Type Reaction: VICODIN contains sodium metabisulfite, a sulfite that may cause allergic-type reactions including anaphylactic symptoms and life-threatening or less severe asthmatic episodes in certain susceptible people. The overall prevalence of sulfite sensitivity in the general population is unknown and probably low Sulfite sensitivity is seen more frequently in asthmatic than non-asthmatic people.
Respiratory Depression: At high doses or in sensitive patients, hydrocodone may produce dose-related respiratory depression by acting directly on brain stem respiratory centers. Hydrocodone also affects centers that control respiratory rhythm, and may produce irregular and periodic breathing If significant respiratory depression occurs, it may be antagonized by the use of naloxone hydrochloride. (see ADVERSE REACTIONS: Respiratory Depression).
Head Injury and Increased Intracranial Pressure: The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a preexisting increase in intracranial pressure. Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries.
Acute Abdominal Conditions: The administration of narcotics may obscure the diagnosis or clinical course of patients with acute abdominal conditions.
PRECAUTIONS:
Special Risk Patients: As with any narcotic analgesic agent, VICODIN should be used with caution in elderly or debilitated patients and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture. The usual precautions should be observed and the possibility of respiratory depression should be kept in mind.
Information for Patients: VICODIN, like all narcotics, may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery; patients should be cautioned accordingly.
Cough Reflex: Hydrocodone suppresses the cough reflex; as with all narcotics, caution should be exercised when VICODIN is used postoperatively and in patients with pulmonary disease.
Drug Interactions: Patients receiving other narcotic analgesics, antipsychotics, antianxiety agents, or other CNS depressants (including alcohol) concomitantly with VICODIN may exhibit an additive CNS depression. When combined therapy is contemplated, the dose of one or both agents should be reduced.
The use of MAO inhibitors or tricyclic antidepressants with hydrocodone preparations may increase the effect of either the antidepressant or hydrocodone.
The concurrent use of anticholinergics with hydrocodone may produce paralytic ileus.
Usage in Pregnancy: Pregnancy Category C. Hydrocodone has been shown to be teratogenic in hamsters when given in doses 700 times the human dose. There are no adequate and well-controlled studies in pregnant women. VICODIN should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.
Nonteratogenic effects: Babies born to mothers who have been taking opioids regularly prior to delivery will be physically dependent. The withdrawal signs include irritability and excessive crying, tremors, hyperactive reflexes, increased respiratory rate, increased stools, sneezing, yawning, vomiting, and fever. The intensity of the syndrome does not always correlate with the duration of material opioid use or dose. There is no consensus on the best method of managing withdrawal. Chlorpromazine 0.7 to 1.0 mg/kg q6h, and paregoric 2 to 4 drops/kg q4h, have been used to treat withdrawal symptoms in infants. The duration of therapy is 4 to 28 days, with the dosage decreased as tolerated.
Labor and Delivery: As with all narcotics, administration of VICODIN to the mother shortly before delivery may result in some degree of respiratory depression in the newborn, especially if higher doses are used.
Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk and because of the potential for serious adverse reactions in nursing infants from VICODIN, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.
Pediatric Use: Safety and effectiveness in children have not been established
ADVERSE REACTIONS:
Central Nervous System: Sedation, drowsiness, mental clouding, lethargy, impairment of mental and physical performance, anxiety, fear, dysphoria, dizziness, psychic dependence, mood changes.
Gastrointestinal System: Nausea and vomiting may occur, they are more frequent in ambulatory than in recumbent patients. The antiemetic phenothiazines are useful in suppressing these effects; however, some phenothiazine derivatives seem to be antianalgesic and to increase the amount of narcotic required to produce pain relief, while other phenothiazines reduce the amount of narcotic required to produce a given level of analgesia Prolonged administration of VICODIN may produce constipation.
Genitourinary System: Ureteral spasm, spasm of vesical sphincters and urinary retention have been reported.
Respiratory Depression: (See WARNINGS.)
DOSAGE AND ADMINISTRATION: Dosage should be adjusted according to the severity of the pain and the response of the patient. However, tolerance to hydrocodone can develop with continued use and the incidence of untoward effects is dose related.
The usual adult dosage is one or two tablets every four to six hours as needed for pain. The total 24 hour dose should not exceed 8 tablets.

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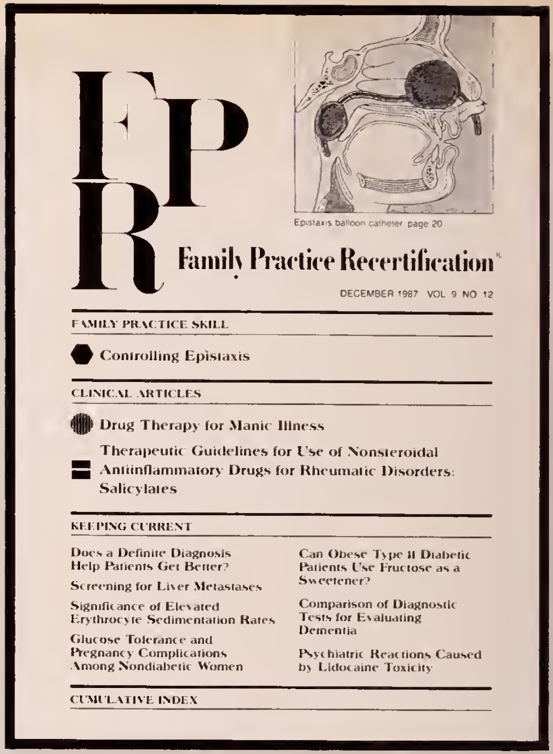

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Index to Advertisers

AMA Advisers 19A
Classified Advertising 6A, 8A, 10A, 22A, 24A, 26A, 28A
Double "E" Enterprises 13A
Family Practice Recertification Magazine 30A
Knoll Pharmaceuticals 27A, 28A
Eli Lilly and Company 14A
Marine Midland Bank 5A
Marion Laboratories 23A, 24A
McGraw Hill Publications 22A
McNeil Pharmaceuticals 7A, 8A
Medical Liability Mutual Insurance Company 21A
Merck, Sharp and Dohme 3A, 4A
New York Beef Industry Council 9A
Palisades Pharmaceuticals 26A
Physicians Reciprocal Insurers 6A
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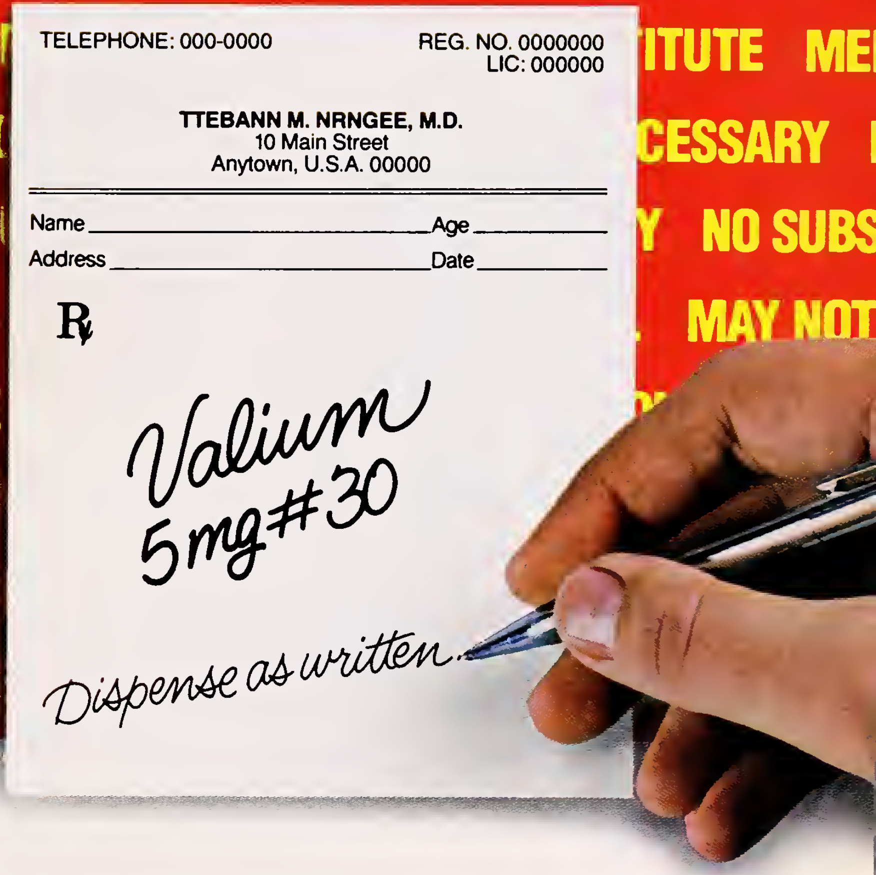

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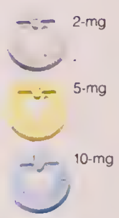

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MARCH 1989 • Volume 89, Number 3

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Contents

COMMENTARIES

Human abuse through the ages 129
FRED ROSNER, MD

The need for physicians to recognize sexual abuse in children 131
MILTON GORDON, MD

The sexual abuse of boys 132
ROBERT L. JOHNSON, MD

Child abuse: Where are we going? 133
RICHARD B. BIRRER, MD

Perestroika, new mental health legislation, and Soviet psychiatry 135
ABRAHAM L. HALPERN, MD

RESEARCH PAPER

Abuse of the child and anxiety in the adult 138
MARJORIE RASKIN, MD; H. GEORGE NURNBERG, MD; ROBERT PRINCE, PHD; JEFFREY FINE, MD; PHILIP E. LEVINE, MD; OZZIE SIEGEL, PHD

REVIEW ARTICLES

Spouse abuse 141
LAURENCE LOEB, MD

Torture and health care professionals 143
LESLIE R. PLACHTA, MD

Fetal neglect and abuse 148
HERMAN M. RISEMBERG, MD

Child abuse: The physician's responsibility 152
VINCENT J. FONTANA, MD

SPECIAL ARTICLES

Abuse of the adolescent 156
REVEREND BRUCE RITTER, OFM CONV

Abuse of the elderly: Misuses of power 159
ERIC J. CASSELL, MD

Confronting child abuse and neglect in New York State 163
BARRY R. SHERMAN, PHD

Anorexia nervosa: The destructive pursuit of thinness 166
STEPHEN NORDLICHT, MD

COMMITTEE REPORT

The physician's management of child maltreatment 168
MARVIN L. BLUMBERG, MD; DONALD LYNN, MD; ELIZA H. CALDWELL, MD; DOMINICK J. DIMAIO, MD; VINCENT J. FONTANA, MD; JOAN T. GOLD, MD; ROY H. HOROWITZ, MD; PETER S. LIEBERT, MD; LOUIS P. RODRIGUES, MD; LESTER SCHIFF, MD; HOWARD S. SCHWARTZ, MD; STANLEY SCHWARTZBERG, MD; LUCIA BOSELLI; DAPHNE GLEIT; JULE FONTANETTA; LILA WOLFF; BARRY R. SHERMAN, PHD

HISTORY

The role of physicians in America's sterilization movement, 1894–1925 169
JOHN S. HALLER, JR, PHD

CASE REPORT

Secondary syphilis in a prepubertal child: Differentiating condylomata lata from condylomata acuminata 180
JOHN M. GOLDENRING, MD, MPH

ESSAY REVIEWS

"While we have time, let us do good" 182
BRIAN QUINN

Child sexual abuse 184
I. RONALD SHENKER, MD

LEADS FROM EPIDEMIOLOGY NOTES 186

NEWS BRIEFS 190

OBITUARIES 192

GUIDELINES FOR AUTHORS 194

MEDICAL MEETINGS AND LECTURES 4A

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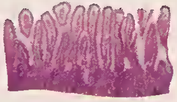
Aspirin and other nonsteroidal anti-inflammatory drugs weaken mucosal defenses, which may lead NSAID users to become prone to duodenal ulcers.[1] For those NSAID users who do develop duodenal ulcers, CARAFATE® (sucralfate/Marion) is ideal first-line therapy. Carafate rebuilds mucosal  defenses through a unique, nonsystemic mode of action. Carafate enhances the body's natural healing ability while it protects damaged mucosa from further injury. So the next time you see an arthritis patient with a duodenal ulcer, prescribe nonsystemic Carafate: therapy for the ulcer-prone patient.

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Pregnancy: Teratogenic effects Pregnancy Category B Teratogenicity studies have been performed in mice, rats, and rabbits at doses up to 50 times the human dose and have revealed no evidence of harm to the fetus due to sucralfate There are, however, no adequate and well-controlled studies in pregnant women Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when sucralfate is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in children have not been established

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OVERDOSAGE
There is no experience in humans with overdosage. Acute oral toxicity studies in animals, however, using doses up to 12 gm/kg body weight, could not find a lethal dose Risks associated with overdosage should, therefore, be minimal.

DOSAGE AND ADMINISTRATION
The recommended adult oral dosage for duodenal ulcer is 1 gm four times a day on an empty stomach.

Antacids may be prescribed as needed for relief of pain but should not be taken within one-half hour before or after sucralfate

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1. Eliakim R, Ophir M, Rachmilewitz D: *J Clin Gastroenterol* 1987; 9(4):395-399.

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BRONX

Apr 6–7. **Pediatric Advanced Life Support.** Montefiore Medical Center. Contact: Danny Flood, Director, Public Relations, Albert Einstein College of Medicine/Montefiore Medical Center, 3301 Bainbridge Ave, Bronx, NY 10467. Tel: (212) 920-6674.

BUFFALO

Apr 14–16. **The Ninth National Conference on Pediatric Lung Diseases.** The Tradewinds, St Petersburg Beach. Contact: Dr Ian Nathanson, 219 Bryant St, Buffalo, NY 14222. Tel: (716) 878-7561.

EAST MEADOW

Apr 5–7. **Advanced Conference on Neuromuscular Electrodiagnosis.** 19 Cat I Credits. Nassau County Medical Center. Contact: Dr A. Fast, PM&R, Nassau County Medical Center, East Meadow, NY 11554. Tel: (516) 542-2835.

MANHATTAN

Apr 6–8. **Antepartum Fetal Assessment.** Grand Hyatt Hotel. Contact: American College of Obstetricians and Gynecologists, 409 12th St, SW, Washington, DC 20024-2188. Tel: (800) 533-8811.

Apr 8. **61st Annual Scientific Session: Update on Arrhythmias.** Pierre Hotel. Contact: New York Cardiological Society, Inc, 84 Grove St, Suite 2, New York, NY 10014. Tel: (212) 924-5346.

Apr 13. **Professional Responsibility Update—Regulations Problems and Solutions.** University Club. Contact: Jemi Goodman, Medical Risk Management Advisory Corporation, 100 Garden City Plaza, Garden City, NY 11530. Tel: (516) 742-6000.

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Apr 10. **Current Assessments in the Diagnosis & Treatment of Anxiety.** 1 Cat I Credit. Southampton. Contact: Charlotte Henry-Sullivan, Medical Staff Office, Southampton Hospital, 240 Meeting House Lane, Southampton, NY 11968. (516) 283-2600.

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Apr 1–2. **New Drugs and New Indications.** 15 Cat I Credits. San Francisco. Contact: Continuing Education S/P, University of California, 3333 California St, Room 218, San Francisco, CA 94118. Tel: (415) 476-8469.

Apr 6–8. **Postgraduate Course in General Surgery.** San Francisco Hilton, San Francisco. *Apr 6–8.* **22nd Annual Advances and Controversies in Clinical Pediatrics.** San Francisco. Contact: Extended Programs in Medicine, University of California, Room 569-U, San Francisco, CA 94143. Tel: (415) 476-4251.

Apr 16–21. **Ninth Annual San Diego Residents' Radiology Review Course.** 39 Cat I Credits. Town and County Hotel, San Diego. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077-1025. Tel: (404) 641-9773.

FLORIDA

Apr 6–9. **Flexible Fiberoptic Sigmoidoscopy and Naso-pharyngo-laryngoscopy in the Primary Care Physician's Office.** 19½ Cat I Credits. Grosvenor Resort, Lake Buena Vista. Contact: Seminars & Symposia, Inc, 132 E 70th St, New York, NY 10021. Tel: (212) 517-7520.

(*continued on p 10A*)

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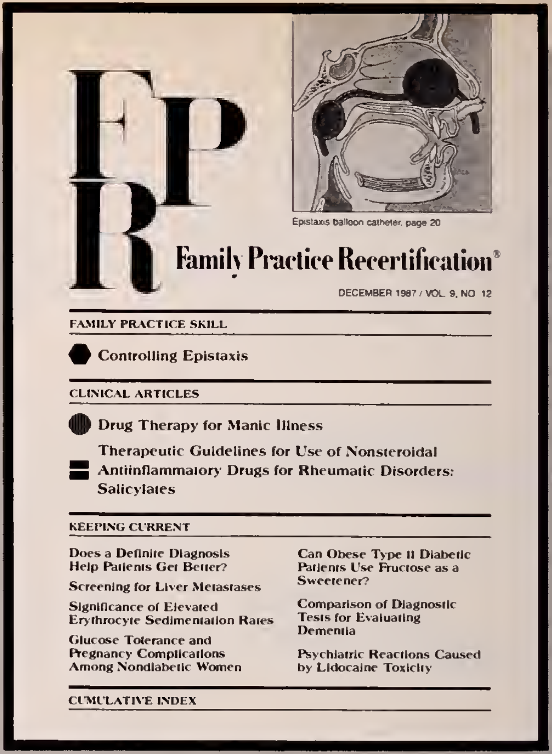

FPR

Epistaxis balloon catheter, page 20

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DECEMBER 1987 / VOL. 9, NO. 12

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'Dyazide' may be used alone or as an adjunct to other antihypertensive drugs; dosage adjustments may be necessary

Contraindications *Concomitant use with other potassium-sparing agents such as spironolactone or amiloride, potassium supplements (except in presence of severe hypokalemia), anuria, acute and chronic renal insufficiencies or significant renal impairment; hypersensitivity to drug or other sulfonamide-derived drugs, preexisting elevated serum potassium concentration*

Warnings *Abnormal elevation of serum potassium levels (greater than or equal to 5.5 mEq/liter) can occur with all potassium-conserving diuretic combinations, including 'Dyazide' Hyperkalemia is more likely to occur in patients with renal impairment, diabetes (even without evidence of renal impairment), elderly or severely ill patients. Since uncorrected hyperkalemia may be fatal, serum potassium levels must be monitored at frequent intervals especially in patients first receiving 'Dyazide', when dosages are changed or with any illness that may influence renal function.*

If hyperkalemia is suspected, obtain an ECG and monitor serum potassium If hyperkalemia develops, discontinue 'Dyazide' and initiate thiazide therapy if needed Persistent hyperkalemia may require dialysis Monitor serum electrolytes frequently in patients with mild renal dysfunction and in diabetic patients. In patients who may develop respiratory or metabolic acidosis, monitor serum electrolytes and acid/base balance frequently

Precautions *The bioavailability of the hydrochlorothiazide and triamterene components of 'Dyazide' is about 50% of the maximum obtainable with oral therapy Theoretically, a patient transferred from therapy with hydrochlorothiazide with or without triamterene might show an increase in blood pressure, fluid retention, or change in serum potassium Extensive clinical experience with 'Dyazide', however, suggests that these conditions have not been commonly observed in clinical practice (See CLINICAL PHARMACOLOGY) Use thiazides cautiously in patients with impaired hepatic function They can precipitate coma in patients with severe liver disease, potassium depletion induced by the thiazide may be important in this connection, administer 'Dyazide' cautiously and be alert for such early signs of impending coma as confusion, drowsiness and tremor, if mental confusion increases, discontinue 'Dyazide' for a few days, attention must be given to other factors that may precipitate hepatic coma, such as blood in the gastrointestinal tract or preexisting potassium depletion If patients develop hypokalemia, which is uncommon with 'Dyazide', increase potassium intake (i e , with supplements or potassium-rich foods) If repeat determinations show serum potassium concentrations below 3.0 mEq/L, discontinue 'Dyazide' and initiate potassium chloride supplementation Institute corrective measures cautiously and monitor serum potassium concentrations frequently, especially in patients receiving digitalis or those with a history of cardiac arrhythmias. Diuretics may aggravate existing electrolyte imbalances, especially at high dosages or in patients on salt-restricted diets Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Chloride replacement may be required in the treatment of metabolic acidosis If dilutional hyponatremia develops, restrict water intake In actual salt depletion, initiate sodium chloride replacement Use 'Dyazide' cautiously in patients with a history of renal stone formation*

If hyperkalemia develops when treating for hypokalemia, take corrective measures Also discontinue 'Dyazide' and, if appropriate, substitute a thiazide diuretic until potassium levels return to normal Do periodic BUN and serum creatinine determinations, especially in the elderly and in patients with suspected or confirmed renal insufficiency Serum PBI levels may decrease without signs of thyroid disturbance Discontinue thiazides before conducting parathyroid function tests.

Angiotensin-converting enzyme (ACE) inhibitors can elevate serum potassium, use with caution with 'Dyazide' Concurrent use with chlorpropamide may increase the risk of severe hyponatremia A few occurrences of acute renal failure have been reported in patients on 'Dyazide' when treated with indomethacin Therefore, caution is advised in administering nonsteroidal anti-inflammatory agents with 'Dyazide' Diuretics reduce renal clearance of lithium and increase the risk of lithium toxicity Clinically insignificant reductions in arterial responsiveness to norepinephrine have been reported Thiazides have also been shown to increase the paralyzing effect of nondepolarizing muscle relaxants such as tubocurarine, therefore use cautiously in patients undergoing surgery Monitor electrolytes in patients taking amphotericin B, corticosteroids or corticotropin concomitantly Thiazides may potentiate the action of other antihypertensive drugs The effects of oral anticoagulants may be decreased when used concurrently with hydrochlorothiazide, dosage adjustments may be needed 'Dyazide' may raise the level of blood uric acid, dosage adjustments of antigout medication may be needed to control hyperuricemia and gout The following agents given with triamterene may promote serum potassium accumulation and possibly result in hyperkalemia, especially in patients with renal insufficiency blood from blood bank (may contain up to 30 mEq of potassium per liter of plasma or up to 65 mEq of potassium per liter of whole blood when stored for more than 10 days), low-salt milk (may contain up to 60 mEq of potassium per liter), potassium-containing medications (such as parenteral penicillin G potassium), and salt substitutes (most contain substantial amounts of potassium). Exchange resins, such as sodium polystyrene sulfonate, whether administered orally or rectally, reduce serum potassium concentrations by sodium replacement of the potassium; fluid retention may occur in some patients because of the increased sodium intake Chronic or overuse of laxatives may reduce serum potassium concentrations by promoting excessive potassium loss from the intestinal tract, laxatives may interfere with the potassium-retaining effects of triamterene The effectiveness of methenamine may be decreased when used concurrently with hydrochlorothiazide because of alkalinization of the urine 'Dyazide' will interfere with the fluorescent measurement of quinidine

There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy only if clearly needed Thiazides and triamterene cross the placental barrier and appear in cord blood The use of thiazides in pregnancy requires weighing the anticipated benefit against possible hazards, including fetal or neonatal jaundice, pancreatitis, thrombocytopenia, and possibly other adverse reactions which have occurred in the adult Thiazides appear, and triamterene may appear, in breast milk If use of the drug is essential, the patient should stop nursing Safety and effectiveness in children have not been established

Adverse Reactions *The serious adverse effects associated with 'Dyazide' have commonly occurred in less than 0.1% of patients treated with this product Anaphylaxis, rash, urticaria, photosensitivity, cardiac arrhythmias, postural hypotension, diabetes mellitus, hyperkalemia, hyperglycemia, glycosuria, hyperuricemia, hypokalemia, hyponatremia, acidosis, hypochloremia, jaundice and/or liver enzyme abnormalities, pancreatitis, nausea and vomiting, diarrhea, constipation, abdominal pain, acute renal failure, interstitial nephritis, renal stones composed primarily of triamterene, elevated BUN and serum creatinine, abnormal urinary sediment, leukopenia, thrombocytopenia and purpura, megaloblastic anemia, muscle cramps, weakness, fatigue, dizziness, headache, dry mouth, impotence, sialadenitis Thiazides alone have been shown to cause the following additional adverse reactions paresthesias, vertigo, xanthopsia, transient blurred vision, allergic pneumonitis, pulmonary edema, respiratory distress, necrotizing vasculitis, exacerbation of lupus, aplastic anemia, agranulocytosis, hemolytic anemia In neonates and infants thrombocytopenia and pancreatitis—rarely, in newborns whose mothers have received thiazides during pregnancy*

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WESTERN NEW YORK—Seeking full-time physicians residency trained in emergency medicine or primary specialty for 32,000 volume emergency department. Sign-up bonus offered. Benefit package available and full malpractice insurance coverage provided. Contact: Emergency Consultants, Inc., 2240 S. Airport Rd., Room 42, Traverse City, MI 49694; 1-800-253-1795 or 1-800-632-3496 in Michigan.

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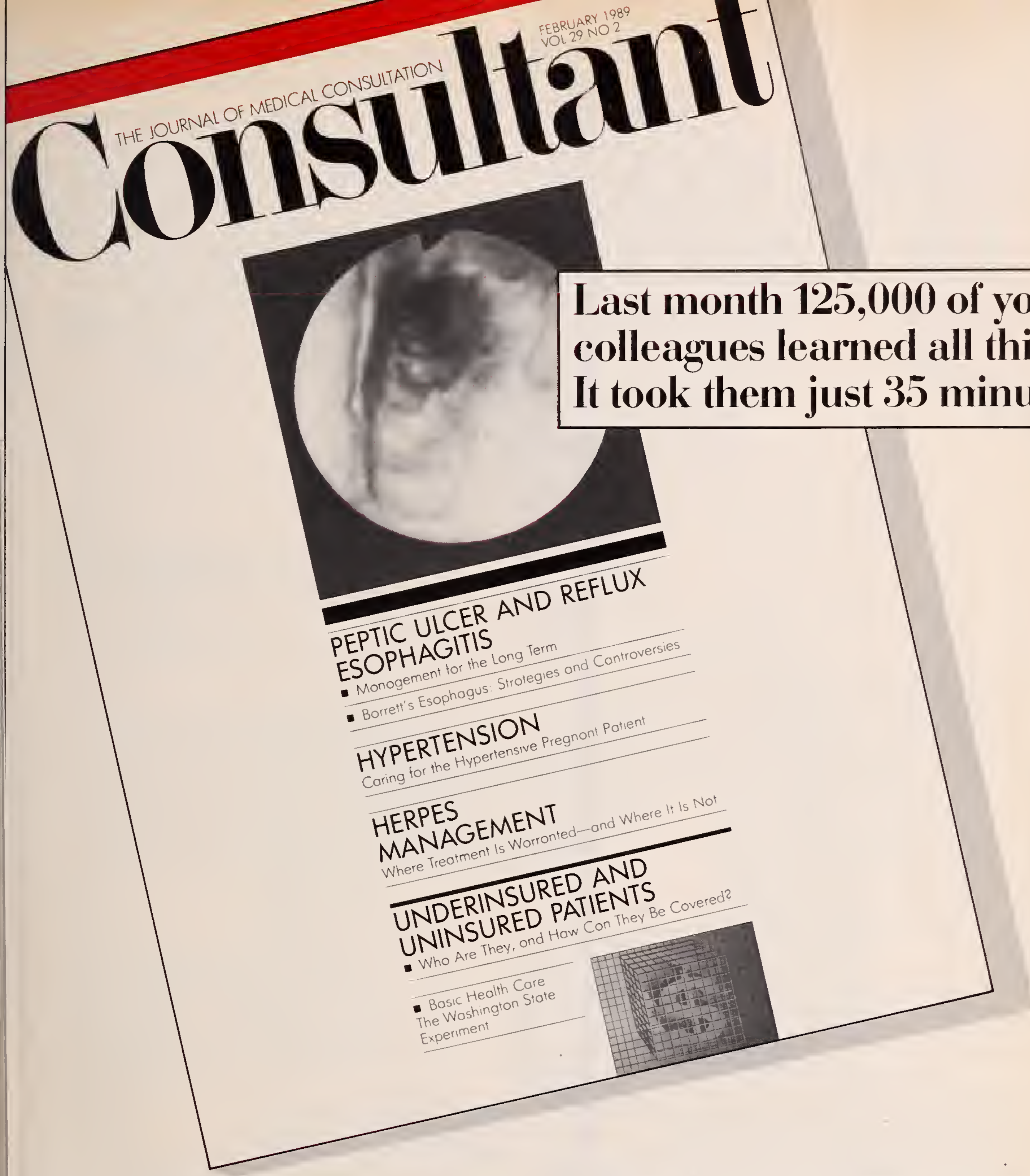

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VOL 29 NO 2
Consultant

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(*continued from p 4A*)

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Apr 10–14. **Postgraduate Course in Gynecologic and Obstetric Pathology with Clinical Correlation.** Boston. Contact: Department of Continuing Education, Harvard Medical School, 25 Shattuck St, Boston, MA 02115. Tel: (617) 732-1525.

Apr 17–20. **16th Annual Harvard Medical School Course on Intensive Care Medicine.** Cambridge Marriott Hotel, Cambridge. Contact: Bart Chernow, MD, Dept of Anaesthesia, Harvard Medical School/Massachusetts General Hospital, 32 Fruit St, Boston, MA 02114. Tel: (617) 726-2858.

NEVADA

April 14–16. **Pediatrics in Progress.** 18 Cat I Credits. Tropicana Hotel & Country Club, Las Vegas. Contact: CME Registration, American Academy of Pediatrics, PO Box 927, Elk Grove Village, IL 60009. Tel: (800) 433-9016.

NEW JERSEY

Apr 7–8. **Meeting the Public's Challenge: Preventive Medicine for the '90's.** 20 Cat I Credits. Claridge Hotel, Atlantic City. Contact: Ryna Alexander, Office of CME, Cooper Hospital/University Medical Center, 1 Cooper Plaza, Camden, NJ 08103. Tel: (609) 342-3074.

SOUTH CAROLINA

Apr 13–15. **Ultrasound for the Practic-**

(*continued on p 22A*)

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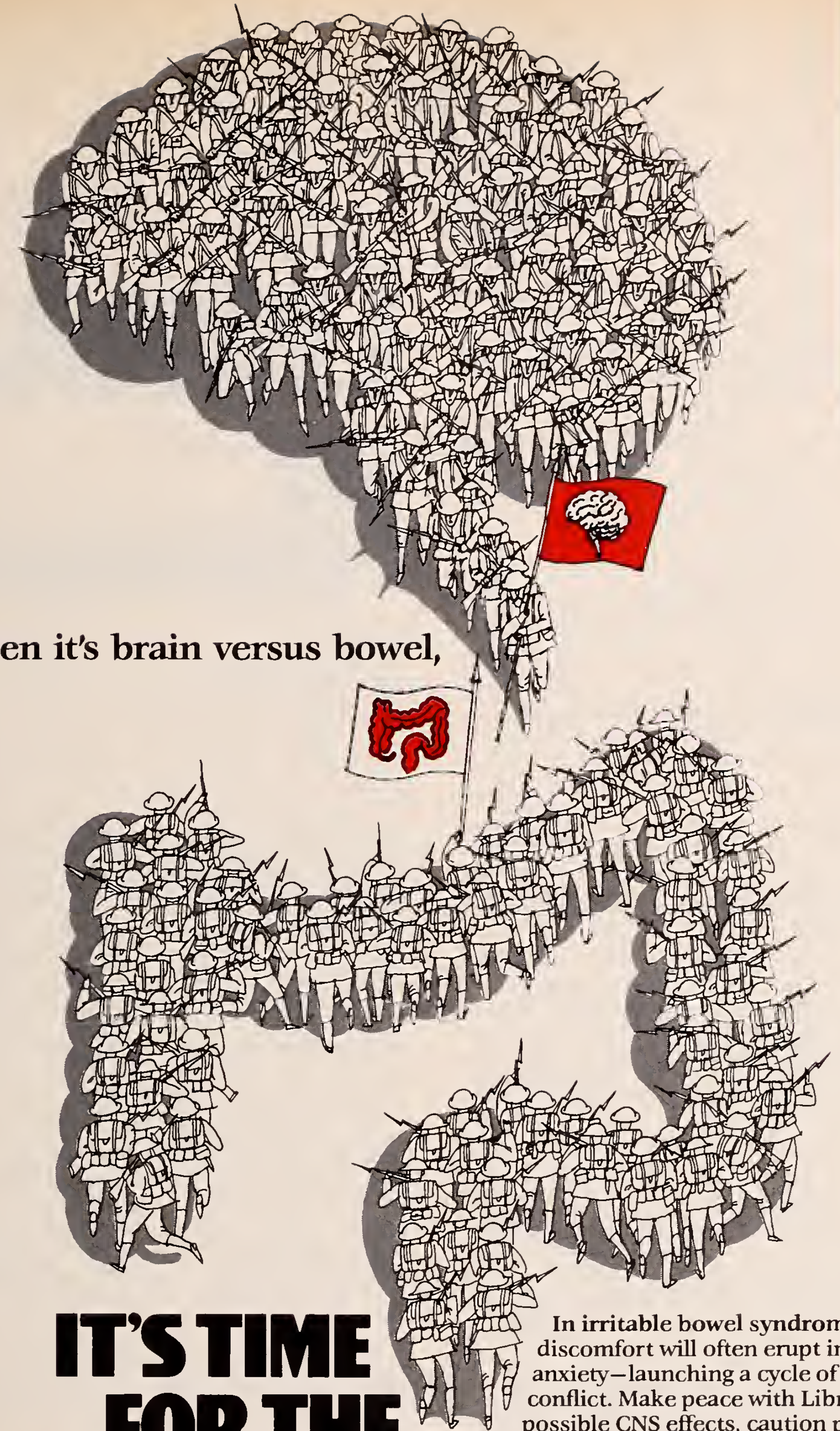

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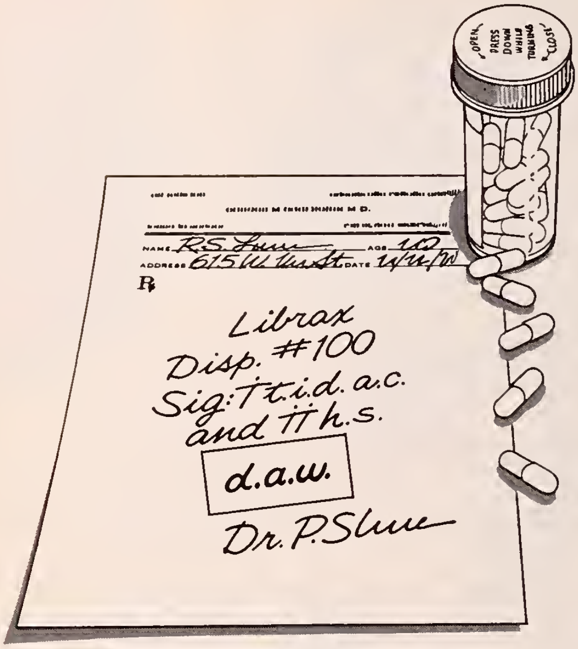

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* **Indications:** Based on a review of this drug by the National Academy of Sciences–National Research Council and/or other information, FDA has classified the indications as follows: "Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.
Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.

As with all anticholinergics, inhibition of lactation may occur.

Withdrawal symptoms of the barbiturate type have occurred after discontinuation of benzodiazepines (see Drug Abuse and Dependence).

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established. Inform patients to consult physician before increasing dose or abruptly discontinuing this drug.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

Drug Abuse and Dependence: Withdrawal symptoms similar to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of chlordiazepoxide; more severe seen after excessive doses over extended periods; milder after taking continuously at therapeutic levels for several months. After extended therapy, avoid abrupt discontinuation and taper dosage. Carefully supervise addiction-prone individuals because of predisposition to habituation and dependence.

PI 0288

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COMMENTARIES

Human abuse through the ages

The word "abuse" is defined in Webster's dictionary[1] in several ways: as a "corrupt practice or custom," as "improper use or treatment," and as "physical maltreatment." "Abuse" is also defined as follows: "to put to a wrong or improper use" and "to use so as to injure or damage." Among the myriad problems facing our society today are many forms of substance abuse, primarily involving alcohol and other psychoactive drugs such as opiates (eg, heroin), sedatives (eg, barbiturates), stimulants (eg, cocaine), inhalants (eg, alkyl nitrates), and hallucinogens (eg, phencyclidine or PCP). The problems of alcohol and drug abuse are enormous and multifaceted, encompassing medical, psychological, economic, social, and public policy aspects. Millions of Americans suffer from these problems. These people abuse themselves, their physical bodies, and their lives, as well as their interactions with their families, friends, and society at large.

At the Annual CME Assembly of the Medical Society of the State of New York on April 22, 1988, the Committee on Bioethical Issues presented a symposium entitled "Human Abuse Through the Ages: From the Womb to the Tomb." The two objectives of this symposium on the moral and ethical issues of abuse of people by people were to discuss the moral and ethical issues relating to abuse of the fetus, the child, the adolescent, the spouse, and the elderly; and to enable physicians to recognize, gain an understanding of, and treat or refer for treatment patients with these conditions or members of their families.

The five presentations made at that symposium, along with several additional articles, appear in this thematic issue of the *Journal* devoted to human abuse.

The topic of pregnant women who abuse their unborn fetuses by smoking or abusing alcohol or narcotics, all of which may injure and even cause death of their unborn babies, is discussed by Herman Risemberg.[2] He also includes physical assault on a pregnant woman as a form of fetal abuse. From extensive experience as Medical Director of New York's Foundling Hospital, Vincent Fontana[3] presents a vivid portrayal of child abuse, the battered child syndrome, maltreatment and neglect of children, emotional and verbal child abuse, and sexual abuse of children by parents and others. Dr Fontana points out that adult abusers were often themselves subjected to abuse when they were children, a point also made by Marjorie Raskin, et al.[4]

Violence against children is manifested in many ways: physically, emotionally, through neglect, by sexual exploitation, and by child labor. According to a standard text entitled *The Battered Child*,[5] considerable experience has been gained in understanding family assessment, treatment, and the physical and developmental effects of abuse and neglect. A literal explosion of information and interest has occurred in the area of sexual abuse, which has emerged as one of the major forms of child abuse.[6]

Damage to children can occur even when no physical harm is done. Psychological maltreatment can occur when a child is rejected, isolated, terrorized, ignored, or corrupted. The field of child abuse and neglect has matured to the point at which the challenges of such maltreatment can be addressed and intervention with corrective measures implemented through protection, treatment, and preventive services, the latter being the most difficult.[7] The United States Congress enacted the first federal child abuse and neglect treatment and prevention act in the mid-1970s.

Father Bruce Ritter,[8] founder of New York's Covenant House, writes about the important topic of runaway and throwaway adolescents, many of whom are abused by pimps and drug dealers who use them for profit. Ritter emphasizes the magnitude of the problem and estimates that there are about half a million such youngsters in the United States.

Laurence Loeb[9] discusses the issue of husbands who beat their wives and, occasionally, the reverse. Spouse abuse is also known as domestic or family or conjugal violence, and is a more widespread problem than many people are willing to admit. Wife abuse can be defined as a man's use of physical force (slapping, hitting, kicking, punching, or using a knife or gun) against his wife or girlfriend on a regular basis ranging from several times weekly to a few times a year.[10] There is a growing recognition of the important role that health care and human service providers can play in domestic violence intervention. Statistics collected by the police, courts, and researchers indicate that family violence is a social problem of major proportions.[10] A national prevalence study showed that each year 16 of every 100 couples in the United States are involved in marital violence, and nearly two million American wives are beaten by their husbands each year.[11]

There is a growing awareness about the victimization of the elderly, which includes crime on the street and in the home by strangers, maltreatment within institutional settings, fraud and deception, and abuse by care providers.[12] The first evidence that elderly persons are abused by their caregivers appeared in the social science research literature of the later 1960s, after public attention was focused on the tragic prevalence of child abuse and domestic violence.[13] Eric Cassell[14] discusses abuse of the elderly by members of their own families, health care personnel, and others involved in the provision of services to the often partially or totally dependent elderly members of our society.

The involvement of health care professionals, primarily physicians, in torture is discussed by Leslie Plachta,[15] who puts the aspects of medical, legal, and historical torture into perspective. In 1975, the World Medical Association[16] adopted the Declaration of Tokyo, which states that a physician "shall not countenance, condone or participate in the practice of torture or other forms of cruel, inhuman or degrading procedures," nor shall he provide any "premises, instruments, substances or knowledge to facilitate the practice of torture." Yet these restrictions have been violated in several countries such as Chile,[17] Uruguay,[18] South Africa,[19] and the Soviet Union,[19] where doctors have actively participated in torture and have falsified death certificates in order to conceal torture. Whether the new mental health legislation in the Soviet Union will reduce or eliminate the practice of involuntary hospitalization of dissidents in mental institutions, as discussed by Abraham Halpern,[20] remains to be seen.

The heinous acts of medical experimentation, torture, and mass murder chronicled at the Nuremberg trials included the subjecting of Jews, Gypsies, and others to sterilization by injection, radiation, or surgery; the injection of vaccines made from dental infections; massive bleedings for blood-group experiments; the use of human flesh for culture media and the making of lampshades and tobacco pouches; brainwashing with chemicals; deliberate infection with typhus; the application of toxic substances to various parts of the body; vivisections and mass killings by phenol injections, gassing, shooting, and other cruel and inhumane methods. These experiments and killings were only a small part of the role Nazi doctors played in Hitler's Final Solution. In fact, doctors supervised the entire killing process at Auschwitz from beginning to end. How were physicians, sworn by oath and conviction to ease suffering, transformed from healers to systematic killers? The involvement of physicians in torture and medical killing is morally repugnant and socially, legally, and religiously unacceptable and unjustifiable.

The abuse of one human being by another human being is a ubiquitous phenomenon as illustrated in some detail in this thematic issue of the *Journal* dealing with human abuse. Greater awareness among health professionals of the magnitude of the problem may lead to greater case finding and diagnosis and the development and implementation of strategies to manage, reduce the frequency of, and eventually prevent human abuse. We hope that this issue of the *Journal* contributes to the goal of increasing such awareness as a necessary first step to combating the problem of human abuse.

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The need for physicians to recognize sexual abuse in children

The results of a nationwide poll conducted by the *Los Angeles Times* in 1985 are said to have verified the massive extent of sexual abuse. Twenty-two percent of those adults surveyed reported having been sexually abused before the age of 18 years.[1] Other studies suggest that the actual incidence of abuse is probably closer to 40%. Ladson et al[2] provide conservative estimates that suggest that 25% of girls and 9% of boys are sexually abused prior to the age of 18 years. Despite these numbers, sexual abuse is said to be largely underdiagnosed and under-reported by the medical community, and as a result it often goes untreated.

A survey conducted by the Child Protective Services Bureau, Department of Social Services, Suffolk County, New York, revealed that less than 2% of all reports of cases of child sexual abuse are generated by physicians.[3] The study suggested that many more cases are either misdiagnosed or overlooked because of inadequate training or the lack of diagnostic tools to help assess a prepubescent child subject to this abuse.

Studies conducted by the Department of Pediatrics and Family Medicine at Ohio State University, College of Medicine, and the Child Abuse Program of Children's Hospital, Columbus, Ohio, revealed that physicians do not routinely examine the genitalia of pubescent girls.[4,5] Physicians do not recognize normal prepubescent female genital anatomy; therefore, they may not recognize anatomic deviations that suggest child abuse. Physicians rarely consider sexual abuse as a possible cause of venereal disease in children. Finally, the physician's age, sex, number of years of practice after residency training, and the number of pediatric patients seen per week affect his or her diagnostic skills and propensity to report suspected sexual abuse.

The Department of Pediatric and Family Medicine at Ohio State University investigated these hypotheses through a physician survey utilizing a 32-item questionnaire. The physician subjects were asked to identify behaviors they considered to be inappropriate or indicative of sexual abuse and to demonstrate knowledge of normal prepubescent genital anatomy, and were assessed with regard to their ability to recognize the association between venereal disease and sexual abuse in prepubescent girls.

The Ohio protocol was used in Suffolk County last year, and the 32-item questionnaire developed by Ohio State University was administered to the 150 pediatricians practicing in Suffolk County, as well as family practitioners. In addition, the same questionnaire was filled out by 200 physicians attending the Annual Meeting of the Family Physicians of the United States at the State University of New York at Stony Brook. Surprisingly, the findings were comparable in all three groups.[2,6]

Physicians in all three groups tended to indicate that inappropriate behavior between older children and their parents was of more concern. In addition, behavior of father/daughter diads was seen as more likely to increase the risk of abuse than the same behavior of mother/son diads beginning at age two years and beyond.

Consistent with the results of the Ohio Study, approximately three-quarters of the physicians in Suffolk County routinely checked genitalia only 50% of the time. In addition, only 50% of all physicians checked the size of the vaginal opening. More than 38% checked the vaginal opening and size of the vaginal opening less than 10% of the time.

Each group studied was asked to identify from a photograph the anatomical parts of the genitalia of a six-year-old girl. Physicians were told that the girl had been referred for genital pain and, again, in all groups only 59% of the physicians correctly identified the hymen. It is possible that some of those physicians who did not respond to the question failed to do so because of uncertainty.

It is of interest to note that those physicians who did check their patients' genitalia saw more than 25 pediatric patients per week, whereas those that did not check genitalia usually saw less than that number. In addition, those that checked the hymenal orifice saw more than 25 patients per week in their practice, whereas the other groups did not.

More than 54% of the physicians responding did not consider a vaginal orifice of 15 mm in a six-year-old girl indicative of sexual abuse. Only 45% of physicians correctly identified the normal horizontal length of the vaginal orifice as 3 mm to 6 mm.

The findings in all groups indicate that practical experience and textbooks are physicians' major sources of knowledge as to normal prepubescent genital anatomy, followed by information obtained during residency, training in medical school, and scientific journal articles.

The most disturbing finding in all groups was a major discrepancy between the proportion of physicians who saw the presence of a venereal disease in a prepubescent girl as raising suspicion of sexual abuse, and the proportion of physicians who would have reported it as possible sexual abuse. Except in cases of gonorrhea and syphilis, fewer than two-thirds of all responding physicians would subsequently report their suspicion to the proper legal authority as suspected sexual abuse. This is an alarming and disturbing finding because venereal disease in children is most likely due to sexual abuse.

There are serious repercussions if sexual abuse of a child is not recognized or reported. The sexually abused child should enter into a protective network. In addition, therapy for the sexually abused child should be instituted early as well as therapy to and/or legal action against the perpetrator. An abused child must be protected from further abuse. Untrained or misinformed physicians need to improve their knowledge of normal prepubescent genital anatomy and the dynamics as well as the consequences of sexual abuse. The medical community must not only become skilled in its recognition of sexual abuse in children, but also must support research involving both appropriate intervention and primary prevention.

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The sexual abuse of boys

The report in this issue of the *Journal* of a clinical finding which suggested that a male child had been sexually abused is an excellent example of the problematic nature of sexual abuse involving male children.[1] The under-reporting of these events has contributed to the popular notion that the sexual abuse of males is either rare or of little consequence to the victim. In 1985, we published our findings from a study of 40 male adolescents who had been sexually abused during their childhood.[2] These young men had attended a general adolescent medicine clinic for routine medical care. Their histories of childhood sexual abuse and molestation were elicited in their responses to the questions in the sexuality section of the medical history and psychosocial inventory that is obtained from all of our patients.

Only six of the 40 young men had reported these episodes of abuse to anyone before the interview. The others reported that they had been either afraid or confused about what had happened and what they could do about it. Although many of the events were single occurrences and had taken place many years ago, most of the victims were still significantly affected by them. These earlier findings are supported in our most recent studies of a population of adolescent and young adult males who had been sexually abused during their childhood by adolescent and adult females.[3] Similar findings have also been reported by Finkelhor,[4] Kempe, [5] and DeYoung.[6]

Male victims list a variety of reasons for not reporting episodes of molestation. If they have been abused by men, they are afraid that they will be considered homosexual because of the implications of sexual contact with another male. Many view sexual abuse as an assault to their virility that exposes them to embarrassment and ridicule. Others are so confused about the event that they are not sure what happened. When the assailant is a family member the victims are often fearful of the reactions of the other family members. They are afraid that no one will believe them or that they will be punished. In other instances the victims are silent in response to implied or explicit threats that have been made by the assailant.

Our findings of high rates of sexual dysfunction in this group of male adolescents belies the popular assumptions about the triviality of male sexual abuse. In addition, the existence of these patients in significant numbers in a population of adolescents who were attending a general adolescent clinic for routine health care attest to its prevalence in a normative population.

Questions about sexual abuse should be included as part of the general medical and psychosocial history of all children and adolescents. The interview should be conducted in a confidential and supportive setting that provides the patient with opportunities to explore these intimate areas of their medical histories without fear or guilt or ridicule. The inquiries should be clear and direct and the patient should be given time to fully question and explain his responses. Most importantly, the health care provider must be able to develop a therapeutic response when it is indicated.

A heightened awareness of the existence of childhood sexual abuse has been one of the beneficial consequences of society's new sexual openness. In a similar manner, the medical community has expanded the limitations of the clinical observations which suggest the possibility of some type of molestation. In addition, as more cases of male sexual abuse are reported, we are gaining a broader respect for the acute and chronic effects of a phenomenon that in the past was thought to be a rare or harmless occurrence.

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Child abuse: Where are we going?

> Some day, maybe, there will exist a well-informed, well-considered, and yet fervent public conviction that the most deadly of all possible sins is the mutilation of a child's spirit; for such mutilation undercuts the life principle of trust, without which every human act, may it feel ever so good and seem ever so right, is prone to perversion by destructive forms of conscientiousness.[1]

Violence in the form of physical and emotional abuse, sexual exploitation, and neglect is part of the mainstream of life for large numbers of families throughout the world. Violence is a cancer that erodes the family foundation and corrodes subsequent generations and their families. Violence against children, in particular, is not a new phenomenon, having existed in every conceivable form for centuries.

Thousands of years ago, newborns had no rights until the "right to live" was ritually bestowed, usually by the father.[2] If the child did not accept a drop of milk, honey, or water during the trial period or died during the ordeal (eg, a plunge into icy water, exposure to the elements, no food for the first day of life), it was thrown away. Infanticide and exposure were universal forms of child abuse that usually resulted in death. The Greeks and Romans regularly used these techniques with sick, retarded, or handicapped children. Many cultures, particularly the Chinese, selectively killed female newborns. In English society undesirable infants were disposed of, usually by nurses, by overlaying (lying upon), or starvation. Thirty to forty percent of illegitimate children, a particularly loathsome Victorian curse, disappeared quietly under foster care.[3] Before 1815 in England and 1858 in America those that did not were sold as indentured slaves to the highest industrial bidder.[4]

> Cosette . . . a creature bruised by a millstone . . . lacerated with pincers . . . beaten unmercifully . . . barefoot in the winter . . . was thin and pale . . . her eyes sunk in a sort of shadow, were almost put out by continual weeping . . . . The corners of her mouth had that curve of habitual anguish which is seen in the condemned and in the hopelessly sick. Her whole dress was nothing but a rag . . . always shivering, her skin black and blue, her naked legs red and rough . . . . The whole person of this child . . . expressed and uttered a single idea: fear.[5]

Other forms of child abuse included castration, child labor, pilferage, and beggary.[4] Children were purposefully undernourished, punished, maimed, or deformed by switches, thongs, pokers, straps, ropes, fire, or boiling water in order to maximize pity and alms and minimize the care giver's expenditure of resources. An emaciated wastrel could sweep more chimney flues than a plump one. Under the Poor Laws, children as young as four were overworked ("workhouse test") and mercilessly beaten in cotton mills and the textile industry.

> Oliver was the victim of a systematic course of treachery and deception . . . he was desperate with hunger, and reckless with misery . . . he slept in a coffin . . . so very lonely . . . and was in a fair way of being reduced, for life, to a state of brutal stupidity and sullenness by the ill-usage he had received.[6]

Prostitution ("breaking in"), pederasty (knights of the cuff), incest, and slavery were additional conduits for the exploitation of children.

Despite biblical injunctions (Genesis 42:22) and the early laws of the Greeks (Solon), Romans, Christians, and Hindus (Rigveda), effective reform awaited the development of child protective laws in the 18th and 19th centuries—a movement which paralleled the establishment of almshouses, orphanages, and foundling hospitals.[7,8] In 1871, the New York State Medical Society recognized the right of every newborn to be protected and supported, and under the direction of Abraham Jacobi, MD, president, improved child labor laws and championed the cause of the feeble, abandoned, and neglected. The Children's Aid Society, Children's Bureau, American Humane Society, Society for the Prevention of Cruelty to Children, and the Child Welfare League advocated for the rights of children.

Unfortunately, the medical profession was oblivious to child abuse until well into the 20th century. John Caffey, MD,[9] a pediatrician and radiologist, was the first physician to suggest that parents might be the instruments of childhood trauma. Another decade passed before Frederic Silverman, MD, extended Caffey's observations, emphasizing the purposeful nature of these injuries. C. Henry Kempe, MD, and his colleagues documented the various pediatric aspects of child abuse from 1951 to 1958 and in 1961, during a multidisciplinary conference, presented their findings under the emotive title: "Battered Child Syndrome." [10] A snowball effect quickly followed that included surveys, publications, symposia, and generous grants concerning child abuse and neglect. The subsequent two decades witnessed a shift in therapeutic focus from a punitive cure to a preventive, rehabilitative treatment approach. Indeed, these efforts have produced a favorable impact—the incidence of child abuse has declined since 1975.

There is much work to be done. Children of all ages are potential victims of all forms of maltreatment. Child abuse and neglect transcend all natural and cultural boundaries. Yet, an understanding and sensitivity to cross-cultural variability in child rearing beliefs and behaviors (eg, initiation rites, child care practices) is essential to achieving a cultural perspective or context that is neither ethnocentric nor relativistic. The annual incidence of reported child abuse in the United States is still 125 to 150 children per 1,000.[11] Thus, each year 900,000 to 1,000,000 children and adolescents under the age of 18 are added to the abuse rolls of various social agencies in the United States. The number of cases is probably two to three times higher. Twenty-five percent of cases involve physical abuse, 45–55% neglect, 10–20% sexual exploitation, and 10% unknown reasons. Prevalence data for the United States indicate that 20–60% of adult females and 10–25% of males are abused during childhood but only a small percentage notify authorities. At least 30% of cases are repeats (50–70% in cases of sexual exploitation); 70% are new cases. There is a minimum 10% chance of death.[12,13] The average age of abused/neglected children is 7.2 years, whereas abused children who die are an average of 3.3 years. The ethnic background of the abused child is 68% white, 27% black, and 8% Hispanic.[14] Ninety-five percent of the time a biologic parent is involved; the remainder involve teachers, grandparents, paramours, step-parents, adoptive/foster parents, siblings, babysitters, or relatives.

Abuse and neglect are maladaptive family interactions or lack of interactions that result in nonaccidental harm to a child's physical and/or developmental states. The special person (ie, the child), the stress or crisis(es) (eg, divorce, illness, loss of job), and the potential to abuse within the parent or guardian form the etiologic triad whose components in the proper sequence and amount interact to produce the protean outcome of abuse and/or neglect.[15] The rate of child abuse increases as the number and severity of parental stressors increases. Social isolation and poverty also increase the risk of child abuse. Lack of parental education per se does not increase the risk of child abuse. The etiology must be viewed as a complex series of events beginning with early childhood experiences and the manner in which these circumstances influence a parent's learned ability to develop and maintain close personal relationships. The abused/neglected child experiences unmet needs; delayed gratification; role reversals; an inability to solve problems, make decisions, or act responsibly; and is quickly conditioned to equate feelings with actions. Childhood is missed, self-esteem poor, identity fragmented, and the opportunity for love and trust irreparably lost. The world of abnormal rearing, thus, sows the seeds of abuse for the next generation.

Despite the renaissance of the movement for children's rights, society still does not have a complete grasp of the nature and extent of child abuse. There are, in fact, some serious misconceptions about certain facets of the problem. While the media have done much to focus our attention on sexual abuse in day care centers and the issue of child abductions by strangers, the effort has often missed the mark. The facts are that children of all ages are sexually abused, and the majority are sexually exploited in their own homes. Furthermore, statistics suggest that the majority of missing children are abducted by family members, not strangers. Lastly, it is reasonable to assume that most parents, while appreciating the fact that child abuse could happen to their children, perceive the problem as "something that does not happen in my home." Many of these individuals still feel tremendous anger and hostility toward abusers.

Given the prevailing interest in this major social problem, how can our nation maximize the momentum and turn that interest into activities that would significantly reduce or end child abuse?

The response should include the following bold but realistic objectives:

- The presence of a multidisciplinary child protection team in every hospital.
- The establishment of a multiprofessional "commission" in every community in order to assess the potential harm or benefit to a child when criminal prosecution is recommended for the perpetrator of the abuse.
- The identification and development of alternative points of entry into the system of health and social services for abused children, such as visiting nurses, physicians, and other providers of family and child services.
- The promotion of primary preventive services in the form of pregnancy planning, preparation for parenting, home care training, childhood, and interpersonal skills programs.
- The provision of readily available quality child care and linguistically and culturally appropriate family services that promote the health and well-being of vulnerable children (eg, runaways, disabled children, and children of homeless parents, teenage mothers, prison inmates, or substance abusers).
- The development of a federal agency responsible for the funding and implementation of prevention programs.
- The production of regular media campaigns dealing with the issue.
- The creation and utilization of a national research agenda and database network.
- The continued education of our professions and the public through improved funding and time allocations.
- The promotion of the Children's Trust Fund and similar funding mechanisms in the public and private sector.
- The requirement of training experience in child abuse for every primary care medical specialty.
- A revitalization of our judicial system in order to enhance its effectiveness in dealing with the problem.

Society lost a champion and inspiration with the death of C. Henry Kempe in 1984. His clarion call must continue through us. The world's children need us. Let us take the time and do some good.

Dum tempus habemus, operemur bonum![16]

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Perestroika, new mental health legislation, and Soviet psychiatry

In the wake of a comprehensive analysis and reappraisal of the state of the economy of the Soviet Union, a systematized program and concrete strategy for the country's further development was inaugurated in 1985. *Perestroika*, a profound restructuring of the economy affecting all segments of Soviet society, was launched, to be accompanied by *glasnost*—openness—and democratization.[1] This revolutionary step received overwhelming, but not unanimous, approval at the 19th All-Union Communist Party Conference in July 1988.

Drastic changes can be expected as well in the practice of medicine and in the management of hospitals. Thus far, the pressures of perestroika are nowhere more in evidence than in the field of psychiatry.

The practice of hospitalizing dissidents in mental hospitals aroused worldwide protest for many years and, in 1983, on the eve of a threatened expulsion, resulted in the withdrawal of the All-Union Scientific Society of Neuropathologists and Psychiatrists from the World Psychiatric Association. During the past year, public criticism of psychiatry has occurred and, under glasnost, articles and letters denouncing the hospitalization of healthy individuals have appeared in the Russian press, including *Pravda* and *Izvestia*.[2(pp1-14)] Amnesty International has recently noted that practically no cases of dissidents charged with crime have been diverted from the criminal justice system to the mental health system in the past two years.[3]

It is, of course, too early to evaluate the effects of administrative and legislative changes that have occurred. Cautious optimism may be justified, but I am reminded of the philosopher Percy Bridgman's caveat: "The true meaning of a term is to be found by observing what a man *does* with it, not by what he *says* about it."[4(p1)] However, a review of some of these changes should be of interest.

As of March 1, 1988, special psychiatric hospitals (maximum security forensic psychiatric institutions) will be the responsibility of the Ministry of Health. Prior to that time, they had been under the control of the Ministry of Internal Affairs, which houses the KGB. Whether this will result in better staffing, improved treatment programs, and lessening of the prison-like conditions remains to be seen. Also, it will be interesting to see whether these hospitals will be restricted to patients who have committed serious acts of violence, and whether the practice of hospitalizing mentally ill persons, let alone non-mentally ill persons, who have been declared to be "socially dangerous" (such as political dissidents and "refuseniks") has ceased.

A new mental health law ("The Conditions and Procedures for Providing Psychiatric Care") was ratified by the Presidium of the Supreme Soviet on January 5, 1988, and went into effect on March 1, 1988.[5(p30)] Although its provisions may fall short of the sweeping reforms that have taken place throughout the United States over the past 25 years, they call for very significant protection of the rights of citizens alleged to be mentally ill and of psychiatric patients as well. For example, a decision to hospitalize a person involuntarily can now be appealed to the chief psychiatrist of a region or directly to a court. The new law, however, makes no mention of restrictions on judges who may order the hospitalization of persons accused of crime apparently to determine their competence to stand trial or their criminal "nonaccountability" on grounds of mental illness. This is of particular concern in the case of persons charged with crimes such as "anti-Soviet agitation and propaganda," "violation of the separation of church and state," "slander of the Soviet state," and "infringement of rights under guise of religious ceremonies." We are told that these types of crimes will be eliminated in the criminal code, or at least rewritten with greater specificity, so that they cannot be interpreted to mean anything a judge says they mean.

The law, likewise, does not limit the power of the prosecutor and the "investigator." The fact is that the present legal system in the Soviet Union mandates the cooperation of all citizens (including psychiatrists) with the investigator when a person is accused of crime, and the withholding of the participation of the defense attorney (unless the investigator otherwise permits) until after the "investigation" is completed (often a period of many months). The law also mandates the imposition of an insanity ("psychiatric nonaccountability") verdict on a defendant, ostensibly for his own good, but with the result that the individual is deprived of an opportunity to face his accusers and to call witnesses on his behalf in a public trial.

The new statute proclaims that "In fulfilling his duty . . . a psychiatrist must act independently and be guided solely by medical criteria and the law." However, there is a dilemma when medical criteria collide with the law. How independently will a psychiatrist act under such circumstances? The new law states that the USSR Procurator-General and his subordinate procurators are responsible for insuring the observance of the law by psychiatric institutions. But what safeguards exist to prevent the misuse of psychiatry by the procurators?

The law states the following: "The initial psychiatric examination must be carried out by a psychiatrist, who is obliged to introduce himself formally to the person being examined." Does this formal introduction include a full description of the nature, purpose, and lack of confidentiality of the examination to the examinee at the beginning of the examination, as is required of American psychiatrists in accordance with the Principles of Medical Ethics with Annotations Especially Applicable to Psychiatry? In addition, one would have liked to see a strong affirmation that all persons hospitalized on the grounds of mental illness who have not been declared incompetent by a court shall have the right to exercise all their civil, political, and cultural rights, including the right to manage their own affairs and to vote in elections. Mental health laws in American jurisdictions specifically prohibit any discrimination and guarantee mentally ill patients protection against discrimination on the grounds of race, color, sex, language, religion, political or other opinion, national or social origin, property, birth, or other status. I find this wanting in the new Soviet legislation.

The USSR maintains a psychiatric register consisting of the names of Soviet citizens who have been diagnosed at some time as psychiatrically disturbed. Such persons are deprived of their full rights as citizens (for example, they are prohibited from holding a driving license), and they can be summoned for involuntary treatment at any time. There are some five and a half million persons on the register who are not in hospitals. Since January 1988, a massive experiment has been in progress whereby thousands of persons in various parts of the USSR, including Moscow and Leningrad, will be removed from the register. The intent is to include only the more seriously ill on the psychiatric register; they will be subject to compulsory treatment if indicated by a psychiatrist, presumably retaining the right of appeal to the courts. Dr A. Moskvichev, USSR Deputy Minister of Health, is reported to have said of this experiment, whose "social and medical" results will be reviewed in two years' time,

> Neurotic disorders, neuroses, even an illness such as schizophrenia in its benign forms present no danger to people surrounding the patient. If the patient's condition has not given rise to a drastic loss of his critical attitude towards himself and the world, he can visit the doctor as he wishes—when he needs to or wants to.

The plan is to extend the program to the whole country if the experiment proves promising. "Already psychiatrists everywhere are setting about reviewing the people under their care, with a view to removing them from the register . . . ," states Dr Moskvichev:

> At meetings with doctors now we do not tire of saying that the duration of registration or previous repeated stays in the hospital are still not grounds for continuing observation in a clinic when there is no longer any practical need. Henceforth we will put people on the register with far greater circumspection.[6(p35)]

The law requires that the rules governing psychiatric examinations and treatment, including emergency treatment of those deemed to represent a danger to society, and the "rights and duties" of patients, as laid down by the USSR Ministry of Health, be subject to publication and be available for inspection. This clearly is intended to prohibit the secret promulgation of rules and regulations concerning the practice of psychiatry in Soviet clinics and hospitals, as has frequently been the case in the past.

The issue of dangerousness continues to be a confusing one. Parts of the new law strongly imply that dangerousness, as in most American jurisdictions, refers to "likelihood to result in serious harm"; that is, a substantial risk of physical harm to the patient himself as manifested by threats of or attempts at suicide or serious bodily harm or other conduct demonstrating that he is dangerous to himself; or substantial risk of physical harm to other persons as manifested by homicidal or other violent behavior by which others are placed in reasonable fear of serious physical harm. On the other hand, reference is made in the new Soviet legislation to "socially dangerous acts," which come under the jurisdiction of the criminal law. If such acts are committed by mentally ill persons, the court may order compulsory treatment in psychiatric hospitals with "ordinary, intensified or strict supervision." The latter terms are not specifically defined. According to Amnesty International, the law currently makes it possible for people who are not a physical threat but who are nevertheless considered "socially dangerous" (for example, by "breaking the rules of socialist society" even though a recognizable crime has not been committed) to be subject to enforced confinement under "intensified" psychiatric supervision.[4(p6)] Misuse of psychiatry as well as fear of psychiatry by the citizen is an ever present danger as long as peaceful dissent from established political norms can prove sufficient grounds for forcible confinement.

Patients undergoing compulsory treatment on the order of a court must be examined by a commission at least once every six months. No appeal of such an order appears to be provided for in the new legislation. The possibility of misuse of psychiatry by the courts is considerable under these circumstances. How much conviction will be put into enforcement of the statute declaring that "The internment in a psychiatric hospital of a person known to be mentally healthy is a criminal offense"? Is it possible to enforce such a statute given the fact that a psychiatrist might in good faith hospitalize a person who is not mentally ill? Would the inclusion of language proposed by a committee of the United Nations Commission on Human Rights make a difference? For example, "No person shall be treated on grounds which are not relevant to the person's condition . . . Patients who are liable to be involuntarily hospitalized must be diagnosed by properly qualified mental health practitioners on the basis of internationally accepted classifications of mental illness."[7] In other words, it should be clearly stated that difficulty in adapting to moral, social, political, or other values, in itself, should not be considered a mental illness. In the absence of serious mental illness, "breaking the rules of socialist society" should not be a reason for involuntary hospitalization.

In March 1988, Soviet and American officials announced a plan to have American psychiatrists visit Soviet psychiatric hospitals and Soviet psychiatrists visit American hospitals. This followed discussions a year earlier between the US Assistant Secretary of State for Human Rights and a senior Soviet Foreign Ministry official.[2(pp17–19),6(p36)] Prior to that time, such proposals were condemned by the Soviets as "interference in domestic affairs." The prospects appear good for an exchange. Such on-site inspection may have a salutary effect on the hospitalization and treatment of mentally ill patients in both countries. Details remain to be worked out: the Americans are insisting on reasonable conditions for the trip, such as having their own interpreters and examining patients in the presence of family members (to prevent substitution of other patients for those the visiting team wishes to examine).[8]

The conclusion is inescapable that important changes are contemplated in Soviet psychiatry as part of the Gorbachev plan to restructure Soviet society. However, while the democratization movement within the Soviet Union must inevitably embrace psychiatry, major reforms may actually be slow in coming. Furthermore, as far as forensic psychiatry is concerned, optimism must be restrained, at least until some of the significant advances in mental health legislation involving involuntary hospitalization are reflected in the criminal procedure law.

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Abuse of the child and anxiety in the adult

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ABSTRACT. We report on the early development and family background of 20 patients admitted to an anxiety disorders clinic serving a disadvantaged population. Sixteen of these 20 patients had been abused or neglected. Nineteen had a diagnosis of at least one personality disorder. We discuss the relationship between the patients' backgrounds and their current disorders. It is our thesis that the traumatic background of our patients is the major factor that allows anxiety to be passed across the generations.

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Child abuse has only recently become recognized as a major social issue and a factor in adult psychiatric illness. The problem of child abuse extends over a spectrum of experiences which include incest, physical and psychological assault, abandonment, exploitation, and neglect. It may be so severe as to result in death. Child abuse generally occurs in families with multiple problems and is often associated with other types of parental dysfunction such as multiple changes in caretakers, maternal deprivation, extended maternal separation, impoverishment, and substandard physical care. The environment of these children is usually highly stressful and lacking in support systems. As a consequence, victims of childhood abuse continue to experience longstanding negative sequelae into adulthood and are over-represented in psychiatrically ill populations. Current studies have shown that abuse of the child can lead to increased severity of personality disorders, panic anxiety disorder, and other psychiatric disorders in the adult.

Our work focuses on the early development of anxious adults, with particular attention to physical and sexual abuse, emotional mistreatment, and parental violence and neglect. We are currently conducting a controlled study of the early childhood experiences of patients in a recently created Anxiety Disorders Clinic. We report here preliminary data on the developmental history of the first 20 patients with anxiety disorders.

PATIENTS AND METHODS

All 20 subjects fulfilled accepted psychiatric criteria[1] for panic anxiety disorder. Additionally, ten patients fulfilled the criteria for agoraphobia. Fifteen of the subjects were women. The average age of all subjects was 38 years. The clinic serves a socioeconomically disadvantaged population. All subjects entering the clinic receive a series of structured interviews, using a grossly disturbed childhood inventory to assess developmental problems. Subjects are interviewed about psychological illnesses in their parents and children. After several weeks, their personalities are rated according to DSM-III R criteria.[1]

RESULTS

Separations and Losses. Nine of the 20 subjects were raised in intact families. One patient experienced the loss of a parent by death before age 18. There were ten instances of permanent separation, nine from a father and one from a mother.

Grossly Disturbed Childhood Inventory. This assessment measures disturbances at the hands of parents or primary caretakers before age 18. Physical abuse was rated present in six cases, typically involving repeated beatings that led to some degree of physical injury in the child. Sexual abuse was rated present in two cases, both male. One boy was forced to perform fellatio on a caretaker, and the other boy was made to watch parental sexual activities.

Emotional mistreatment was rated present in 11 cases based on the fact that the individual was repeatedly ridiculed or humiliated. For example, one girl had her hair cut off repeatedly against her will in a way she found humiliating. In another case, the child was repeatedly told that he was unwanted and a burden.

Parental violence was rated present for 11 subjects; in 10 the violence occurred when the parent was intoxicated. Typical examples included a parent shooting a gun while drunk. In another case, the police had to come into the house repeatedly to prevent the parents from hurting each other.

Neglect was judged present in five cases, including medical neglect, and parents making no response to a child's truancy. Overall, 16 of these 20 individuals (80%) were rated positive for at least one aspect of gross childhood disturbance (Table I).

Personality Disorders. Nineteen of the 20 patients were diagnosed as having a personality disorder and some received more than one diagnosis (Table I).

Illness in the Family. Fifteen parents of our 20 patients were alcohol abusers according to patient histories. Another five had problems with depression, and an additional two parents were treated for problems with anxiety. Fifteen of the 20 subjects had a total of 29 children, ranging in age from infants to adults. According to the parents' reports, 12 children had emotional or learning difficulties that necessitated special programs. Substance abuse was present in four children, all boys. Additionally, three children had been deserted by a parent. A male patient, deserted by his own father, deserted his son. A female patient, deserted by her mother, abandoned two daughters. To our knowledge there has been no physical or sexual abuse between these patients and their children.

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TABLE I. Grossly Disturbed Childhood and Personality Disorders in 20 Subjects

Patient	Physical Abuse	Sexual Abuse	Emotional Abuse	Neglect	Parental Violence	Personality Disorder
1					*	*
2	*	*	*	*	*	*
3	*					*
4			*		*	*
5			*			*
6	*		*	*	*	*
7					*	*
8	*	*	*	*	*	*
9	*		*		*	*
10	*		*	*	*	*
11			*			*
12			*			*
13			*		*	*
14			*		*	*
15						*
16						*
17						*
18					*	*
19				*		*
20						
Total	6	2	11	5	11	19

* Positive criteria and the presence of a personality disorder.

DISCUSSION

Anxiety disorders are the most frequent psychiatric problems in the general population.[2] Problems with anxiety, particularly panic disorder and agoraphobia, are familial.[3-5] In families with these problems there is also an increased incidence of depression and alcoholism.[5,6] If a parent has panic disorder or agoraphobia, up to half of the offspring have a psychiatric disorder. Female children are at risk for anxiety disorders, and male children are at risk for problems with substance abuse. These facts have led to a strong interest in the genetic transmission of anxiety disorders with a relative neglect of the psychological development of these problems. Yet the presence of anxiety, depression, and alcoholism in these families undoubtedly has nongenetic consequences for the developing child.

In a large study of twins, Kendler et al[7] concluded that the genetic transmission of anxiety is due to nonspecific factors. In this study they identified an inborn sensitivity that led to either anxiety or depression depending on the individual's current situation. A clue in identifying these nonspecific factors comes from studies of temperament in children. Children inherit temperamentally distinct ways of responding to the new and unfamiliar. This temperamental factor has been given a variety of labels depending on the investigator. The best known label is introversion-extroversion. It is the introverted child who has difficulty approaching new people and new situations. Introversion and extroversion appear to be enduring characteristics of the individual.[8] Current work also shows that introverted children are more apt to develop problems with anxiety.[9] Rosenbaum et al[10] found that introversion was more frequent in children of a parent with anxiety disorder. However, despite the importance of these findings, introversion appears to be neither necessary nor sufficient to produce anxiety in the adult.

In the Chess and Thomas[9] longitudinal study of predominantly middle class children, the development of anxiety is shown to be related to an interaction between constitutional factors, particularly temperament, and environment. Introverted children are more apt to have problems with anxiety. However, when these children are sensitively handled, the introversion does not lead to adult anxiety. In the Chess and Thomas study, overprotection of the child was a major source of later anxiety. This study also showed that extreme family problems led to the development of emotional difficulties, including anxiety, in the extroverted child.

The literature on environmental causes of adult anxiety is surprisingly small. Lipsedge[11] reported from his studies that 58% of patients with agoraphobia had severe disturbances in their upbringing. These disturbances included parental illness, violence, and separation. Brier et al[12] found a much smaller prevalence of developmental trauma in the childhood of anxious adults. They asserted that 25% had a chaotic childhood. Raskin[13] reported a 70% prevalence of grossly disturbed childhood in adults with panic disorder. Controlled studies comparing anxious adults to normal adults are even rarer. Faravelli et al[14] looked at traumatic life events in agoraphobics and normal controls and found significant differences. Sixty-five percent of the anxious adults, but only 25% of the controls, suffered traumatic losses or separations. Solyom et al[15] found an increased incidence of maternal overprotection in agoraphobic patients. From what is known about development, one would not expect a single type of problem to give rise to adult anxiety. Thus, overprotection at one extreme and neglect at the other can produce developmental problems in the child and anxiety in the adult. Samples from different populations will show different types of problems.

Anxiety occurs when the individual perceives a threat and feels uncertain that he can cope with and overcome that threat. Technically, one must distinguish between fear—a response to an identifiable external threat, and anxiety—a response to a predominantly subjective danger. By definition, anxiety is a disproportionate response to a perceived real and current danger. The backgrounds of the subjects reported here contributed to their over-responsiveness. Their background of abuse and neglect in childhood makes them perceive the world as excessively dangerous and themselves as unable to cope with it. Their backgrounds also amplify existing temperamental bias toward over-reactions. Thus, the high prevalence of personality disorders becomes predictable. Personality disorders occur when the individual's more enduring ways of dealing with the world are maladaptive, as manifested by impaired function, distress, or loss of a sense of well-being.

Recognizing the limited data, we observed disturbing associations between childhood experience and adult behavior. Deserted children often seem to desert their own children, and abused children are more likely to become abusive parents. Parenting is difficult under the best of circumstances, and the child who is himself uncared for can be expected to have even greater difficulties caring for others. It is our thesis that the traumatic background of these patients is the major factor that allows anxiety to be passed across the generations. Moreover, to the extent that this background is present, the outcome and morbidity of anxiety disorders is adversely affected.

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Spouse abuse

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An executive is stopped by police as he tries to force his wife's car off the road "to teach her a lesson." The wife of a surgeon wears sunglasses to a luncheon, explaining that she "slipped in the kitchen and hit her head." A woman accused of murdering her husband tries to invoke self-defense as an explanation.

Some husbands beat their wives, some wives beat their husbands. Some spouses who have vowed to love and honor and cherish each other cause pain and dreadful harm to the very objects of their promises, and may even cause the death of the other. Spouse abuse is not new. We hear of it under many names: domestic violence, family violence, conjugal violence, battering.

Throughout a large part of recorded history, women and children were treated as property. One text comments that, "Early English common law stated clearly that the husband and wife are one person in law" and the very being of the wife was that of her husband. In the 19th century in the United States, says Ann Jones,

> . . . both Margaret Fuller and Elizabeth Cady Stanton thought that wife-beating was widespread . . . indeed, the law authorized husbands "to restrain a wife of her liberty" and to administer moderate correction in any case of misbehavior . . . Wife murder became commonplace . . . it was not until 1871 that Massachusetts courts ruled that a man had no right to beat his wife . . . ."[1]

Even before that ruling, a man could not beat a stranger with legal impunity.

Violence is not restricted to married people. Couples living together or even those in dating relationships sometimes come to blows or worse. Boyfriends have injured their girlfriends, and the affianced have harmed those they say they love. The abuse of ex-spouses is also not uncommon.

Violence is not defined by social or economic classes. The educated, the well-to-do, the so-called social elite are far from free of this calamity. One study[2] states that:

> It is estimated that in one of two marriages at least one episode of violence, probably more, will occur. In one out of four marriages the violence will be ongoing, with five or more episodes per year. At the extreme, episodes will happen monthly, or even more frequently.

These data are echoed by Lenore Walker,[3] who has written extensively on battered women and has said, "Some observers including myself estimate that as many as 50% of all women will be battering victims at some point in their lives." The accuracy of these data is hard to determine, or believe, but the existence of battering is certainly widespread.

The idea of Maggie throwing a rolling pin at Jiggs in the comics may have inspired laughter, but violence on the part of any person toward another is not truly funny. The magazine *Insight*[4] published an article on abuse directed by wives towards their husbands. Sources cited in this article confirmed some of the findings of a Canadian study;[5] for example, "It may come as some surprise to find that a higher proportion of women (22.6%) hit or throw things than men (14.6%)." That article in turn quoted an article written in the United States, which stated that "the number of women who throw things is twice as large as the number of husbands who throw things. Kicking, hitting, and threatening with knife or gun also was higher for wives, but pushing, shoving, slapping, beating up and using a knife or gun was higher for husbands." It seems clear that not all such behavior is in response to the battering man; some women are batterers. Nonetheless, men injure women more often than women injure men.

SPOUSE ABUSE

Violence of person against person is not just an issue of sexual politics, although in many cases that is exactly what is operative. The term "politics" is used here to denote power. For many batterers, the need to demonstrate power over someone not as strong is a prime motive for this behavior. Clearly, one would have to have a persistent and inner sense of being helpless and unlovable, at the mercy of others, in order to need to demonstrate its opposite so forcibly.

A second factor is the sense of wounded narcissism. For some people, the apparent love objects are not seen as truly independent humans beings, but as parts of the self. When, as sometimes happens in separation or divorce, the spouse demonstrates that he or she is in fact an individual, the narcissist responds with rage. For him, it is as if a part of his being, his self, has been ripped away, causing him intolerable pain, which he wishes to inflict by way of re-

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venge on the one who has dared to behave independently.

A third, very important, factor in this type of abuse is the need for control on the part of the batterer. He must control nearly all aspects of his spouse's life. Some women report needing their husband's permission to shop with friends or spend the day with other mothers and children. This need for control of the spouse often appears in the guise of jealousy, which the controlling spouse presents as evidence of the intensity with which he "loves" the other. This need for control is sometimes exerted more strongly by control over the pocketbook: the appearance of a manifestation of financial independence on the part of a wife may be the precipitant for an episode of battering.

Any division between these three areas—power, narcissism, and control—is arbitrary, since they may coexist or overlap.

What of the victim? Too often, the victim is blamed by being tagged with the label of "masochist," as if she invariably provokes her spouse into abusing her. It is certain that this does occur in some cases, but in all likelihood represents the minority of battered wives. Society often asks why a wife stays in a relationship with a batterer. Walker[3] described a syndrome existing in many battered women which she calls "learned helplessness." Three stages commonly occur in battering: the first is tension building in the relationship, in which the husband becomes angrier and angrier. Often, the wife attempts to pacify him while trying to shield her children from his developing rage, but this rarely suffices to avert the coming storm. The second stage is that of the acute battering incident itself. This ushers in the third phase, that of kindness and contrition on the part of the batterer, with repeated expressions of apology and promises that nothing like this will ever happen again. Protestations of love serve to try to make things better, and there is a rise in hopeful expectations that this is finally the end of it all.

For many women, a point is reached at which the promises are no longer enough. The woman may go to the police for help or use other resources. The police may be reluctant to interfere in family violence, in part because of the apparently mistaken belief that they are more at risk in these situations than in usual criminal cases, and in part because of the common societal attitude that what happens within a family setting should remain private. These attitudes are slowly being changed, and some jurisdictions now insist that family violence is a matter for the criminal justice system and not for the family courts alone. The law in New York State includes jurisdiction by both family and criminal courts, and states the following:

> The family court and the criminal courts shall have concurrent jurisdiction over any proceeding concerning acts which would constitute disorderly conduct, harassment, menacing, reckless endangerment, assault in the second degree, assault in the third degree or an attempted assault between members of the same family or household . . .[6]

The laws do not always protect the victim as well as they should. Okun[7] commented that, " . . . many shocking travesties of sentencing occur. A Kansas case in 1982 saw a convicted woman-batterer sentenced to buy his wife a box of chocolates . . . .The legal profession is hardly alone in ignoring or disserving the battered woman." He added the fields of medicine, mental health, and religion to those that often prefer to look the other way. We in medicine, psychiatry, and related fields are often the first to learn of a person's having been battered. In addition to providing appropriate medical care, the doctor should know of the options available to the victim and should make these known. For example, the New York State Department of Social Services publishes a small booklet entitled *The Handbook for Beaten Women*[8] which lists alternative means of action. Emergency shelters are available. There is a Domestic Violence Hotline with a toll-free number which can advise of service programs. Victims service agencies may be called. Police may be called. The person may go to family court to obtain an order of protection or other legal remedies.

Working with the battering man may raise unexpected ethical problems for the treating physician. Episodes of violence occur, sometimes predictably, after drinking or the use of other drugs. Bland and Orn[5] stated that, "Particularly high rates of violence are found in those where alcoholism is combined with antisocial personality disorder and/or recourse to depression." Assuming that the doctor is trying to help the alcoholic or drug-abusing patient, should the spouse be told that she may be beaten? Should law-enforcement agencies be notified? If the doctor knows of the existence of an order of protection and is told by the patient of his intent to go to the spouse's home in spite of such an order, how should the physician deal with this? Should an agency or spouse be told of the batterer's failure to appear for treatment?

These issues involve questions relating to the confidentiality of the physician-patient relationship as measured against the likelihood of harm to another. We are often faced with the possibility that one who is in need of treatment to prevent battering may not enter treatment because of concerns that information or knowledge of his behavior will not be kept confidential.

Some physicians feel that the best advice to give to the battered spouse is to bring the police into the situation as quickly as possible. Experience has shown, repeatedly, that most cases of spouse abuse, if unchecked, not only do not disappear but escalate into increasing danger. Involvement of law-enforcement agencies can demonstrate more forcibly than any other way that spouse beating behavior is taken very seriously and must be stopped forthwith.

Ultimately, as a concerned member of the society, the physician shares responsibility for trying to interrupt cycles of family violence. The child-witness of parental violence may well become the batterer of the next generation, each generation giving rise to increasing injury. We are awash in violence. It permeates our airwaves and television sets, our movies and plays, and, most sadly, our newspapers. The death of an adolescent girl in Central Park at the hands of another adolescent or that of an innocent person by the bullet of a drug merchant are tragic occurrences. Our own sense of responsibility must lead us to do what we can within the family to help treat spouse abuse and to prevent unnecessary and tragic death.

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Torture and health care professionals

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Torture is a significant, and at times devastating medical problem affecting large areas of the globe today. The vast number of refugees flowing from such areas to more hospitable lands makes the concern about torture's effects and treatment even more widespread. This paper attempts to put the various aspects of torture in context for health care professionals.

WHAT CONSTITUTES TORTURE?

In 1975, the General Assembly of the United Nations adopted the *Declaration on the Protection of All Persons from Being Subjected to Torture and Other Cruel, Inhuman or Degrading Treatment or Punishment.* Article 1 states,

> 1. For the purpose of this Declaration, torture means any act by which severe pain or suffering, whether physical or mental, is intentionally inflicted by or at the instigation of a public official on a person for such purposes as obtaining from him or a third person information or confession, punishing him for an act he has committed or is suspected of having committed, or intimidating him or other persons. It does not include pain or suffering arising only from, inherent in or incidental to, lawful sanctions to the extent consistent with the Standard Minimum Rules for the Treatment of Prisoners.
>
> 2. Torture constitutes an aggravated and deliberate form of cruel, inhuman or degrading treatment or punishment.[1(p 29)]

The Standard Minimum Rules (adopted 1958) include Rules 31 and 32:

> 31. Corporal punishment, punishment by placing in a dark cell, and all cruel, inhuman or degrading punishments shall be completely prohibited as punishments for disciplinary offences.
>
> 32. (1) Punishment by close confinement or reduction of diet shall never be inflicted unless the medical officer has examined the prisoner and certified in writing that he is fit to sustain it.
>
> (2) The same shall apply to any other punishment that may be prejudicial to the physical or mental health of a prisoner. In no case may such punishment be contrary to or depart from the principle stated in rule 31.
>
> (3) The medical officer shall visit daily prisoners undergoing such punishments and shall advise the director if he considers the termination or alteration of the punishment necessary on grounds of physical or mental health.[1(p 39)]

Note that psychological torture is given equal footing with physical torture.

TORTURE IN HISTORY

Every nation has practiced torture at one time or another in its history.[2(p27)] To review briefly the history of this practice in the West,[3–5] Ancient Greece and Rome forbade the torture of citizens. However, in Athens a slave's testimony was not considered reliable unless he had been tortured, and likewise in Rome. Civil and criminal courts sanctioned the use of torture on slaves in order to obtain confessions and permitted masters to use torture to discipline slaves. Toward the end of the Roman Empire, the use of torture began to encroach on the non-slave as well. With the rise of Christianity the practice fell into disuse, as the Church opposed it. In medieval times, the emphasis on confessions as uniquely valuable evidence of crime brought the practice back, and by the 11th century it had reappeared in secular law. The Roman Catholic Church's fear of growing heresy led to the Inquisition, and Pope Innocent IV in 1252 formally authorized the use of torture in interrogation. The European witch-hunts culminated in 1560–1660. Moving into the 18th century and the Enlightenment, the voices against torture dominated. A series of actions practically eliminated its use, such as the English Bill of Rights in 1689, the abolition of torture in France in 1789, the same in Russia in 1801, and the formal forbidding by papal bull in Catholic countries in 1816.

It was not until the Second World War that the issue again came glaringly to the fore, with the horrors of the Holocaust. The victors of that war saw themselves as representatives of the best of Western civilization with its humanist tradition, one of its values being the abolition of torture. This principle found its way into the postwar declarations on human rights and laws of war without any dissent or debate.[2(p 30)]

THE WEST

Most Americans no doubt feel the United States, as leader of the Free World, stands untainted by allegations of torture. Several countries on "this side" of the iron curtain, however, have been called to account in recent years; Great Britain for its handling of prisoners in Northern

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Ireland, and France for torture during its conflict in Algeria.

Indeed, the United States itself bears a certain responsibility for the widespread occurrence of torture in the many countries that it supports around the world, especially in Latin America.

> The main exporter of cold-war ideas, the principal source of the belief that dissent must be crushed by every means and any means, has been the United States. Our indoctrination of foreign troops provided a justification for torture in the jail cells of Latin America. First in the Inter-American Police Academy in Panama, then at the more ambitious International Police Academy in Washington, foreign policemen were taught that in the war against international communism they were the "first line of defence."[6]

Brazil, for example, was the largest recipient in South America of US arms transfers between 1966 and 1975, and of police equipment and training under the US Public Safety Program of 1961–1973.[7(p 70)]

> Serious charges that US advisors working in Brazil and Uruguay in the late 1960s for the Office of Public Safety of the Agency for International Development (AID) had trained Brazilian and Uruguayan security personnel in "harsh interrogation techniques" prompted the US Congress to terminate the agency's Public Safety Program in 1975.[5(p 11)]

SHOULD TORTURE BE ALLOWED?

This question can be answered in a number of ways, and the context in which it is placed will help determine the answer obtained. The legal question and the medical-ethical concerns are addressed in later sections.

The general ethical question is much too complex to do justice to here. In order not to ignore completely such a fundamental issue, however, let me state some of the more commonly used arguments in support of torture: It is expedient for the extraction of information; the interrogator's credence in the information he is getting is reinforced if the victim does not change the story under torture; it is often committed not so much to menace the particular victim as it is to threaten those whom he will tell of his treatment.

Michael Levin,[8] Professor of Philosophy at the City College of New York, stated the following:

> there are situations in which it is not merely permissible but morally mandatory to torture. Suppose a terrorist has hidden an atomic bomb on Manhattan Island which will detonate at noon on July 4 unless. . .(here follow the usual demands for money and release of his friends from jail). Suppose, further, that he is caught at 10 am that fateful day, but—preferring death to failure—won't disclose where the bomb is. . . .If the only way to save those lives is to subject the terrorist to the most excruciating possible pain, what grounds can there be for not doing so? I suggest there are none . . . .Torture only the obviously guilty, and only for the sake of saving innocents, and the line between Us and Them will remain clear. There is little danger that the Western democracies will lose their way if they choose to inflict pain as one way of preserving order.

But what of the following scenarios? A man admits to planting a bomb; torture will save lives. A man is suspected of planting a bomb; torture will reveal it. A man has a friend suspected of planting a bomb; torture will lead us to the suspect. A man has dangerous opinions and might be thinking of planting a bomb; torture will reveal his plans. A man knows the one with dangerous opinions and probably thinks the same; torture will lead us to still others. A man has refused to say where a suspect is; torture will intimidate others who might do the same.[9]

From the point of view of the individual, torture, for whatever purpose, is a calculated assault on human dignity, and for that reason alone is to be condemned absolutely.[7(p 7)]

THE LEGAL STATUS OF TORTURE

Torture is not permitted under international law.[2(pp 37–38)] This is the position arrived at by the New York State Second Circuit Court of Appeals in 1980, stating with reference to the United Nations Charter that although

> there is no universal agreement as to the precise extent of the "human rights and fundamental freedoms" guaranteed to all by the Charter, . . .there is at present no dissent from the view that the guarantees include, at a bare minimum, the right to be free from torture.[10]

The Charter of the United Nations (1945) speaks of a "faith in fundamental human rights, in the dignity and worth of the human person, in the equal rights of men and women and of nations large and small," and further of "promoting and encouraging respect for human rights and for fundamental freedoms for all without distinction as to race, sex, language, or religion."[11]

The human rights provisions of the UN Charter are further defined by many UN documents, among the most important being three which are collectively known as the International Bill of Rights: the Universal Declaration of Human Rights (UDHR) (1948); the International Covenant on Civil and Political Rights (ICCPR) (1976); and the International Covenant on Economic, Social and Cultural Rights (1976). In the UDHR, Article 5 states, "No one shall be subjected to torture or to cruel, inhuman or degrading treatment or punishment."[12] The ICCPR in Article 7 repeats this statement and adds, "In particular, no one shall be subjected without his free consent to medical or scientific experimentation."[12(p 5)]

Other international declarations and treaties ensuring the illegality of torture can be found in Table I.

THE GEOGRAPHY OF TORTURE

Clearly a list of countries in which torture is practiced on a systematic basis will change with time as conditions

TABLE I. Declarations and Treaties Condemning Torture

Global

Universal Declaration of Human Rights (1948) Article 5

Geneva Conventions (1949) Common Article 3

UN Declaration on the Protection of All Persons from Torture and Other Cruel, Inhuman, or Degrading Punishment (1975) Article 3

International Covenant on Civil and Political Rights (1976)

Regional

Council of Europe Convention for the Protection of Human Rights and Fundamental Freedoms (1950) Article 3

American Convention on Human Rights (1969) Article 5 (2)

African Charter on Human and Peoples' Rights (adopted 1981, not yet in force), Article 5 in the countries listed change. This can be seen by the example of Nicaragua. Amnesty International reported in its 1975 worldwide survey of torture[2] that torture was carried out in the Presidential Palace and in the quarters of the national guard in support of the Somoza family and its government. In its 1984 survey,[7] Nicaragua was no longer listed.

To give a sense of the very widespread nature of the problem of torture in this decade, listed in Table II are those 67 countries singled out in the Amnesty International report *Torture in the Eighties*[7] in which systematic torture is tolerated. Due to obvious reluctance to permit the free flow of this information in some countries, even this list cannot be considered exhaustive.

METHODS OF TORTURE AND THE SEQUELAE

The list of torture types (Table III) is taken mainly from Goldfeld et al,[13] in turn a compilation of six series on torture victims in the literature. Goldfeld's article also emphasizes the high percentage of patients who suffered some form of head trauma and the common occurrence of sexual violation among tortured women and female adolescents. While it is hoped that such a list will make more concrete the general term "torture," it is the context in which each of these punishments occurs that is the essential concern. As Roth[14] points out, the aim of all torture is psychological. Torture aims at degradation, insult, humiliation, often in the form of sexual abuse or abuse of a companion or family member. A torture victim from Turkey witnessed a marrried couple being tortured together in 1981:

> In the presence of four or five torturers they were undressed and made totally naked. Their blindfolds were removed. A torturer would play with the genital organs of the wife, squeezing her breasts and caressing her hair while the husband watched. The reverse would be applied to the husband. While his wife watched they would give electric shocks to his penis, hang him by his feet. They would threaten to rape his wife unless he would admit the accusations made against him. This was one of the tortures that the husband could not bear. And because of that he would admit a lot of crimes that he did not commit.[7(pp 19-20)]

There is evidence that children have been made to witness the torture of their parents, and that children have been tortured in front of their parents to "make the parents talk."[15]

Jean Paul Sartre[16] has written that the purpose of torture is "not only the extortion of confessions of betrayal, but, the victim must disgrace himself by his screams and his submission like a human animal. In the eyes of everybody and in his own eyes, he who yields under torture is not only to be made to talk, but is also to be marked as subhuman."

TABLE II. Countries in Which Torture Has Been Reported, 1980–1983[7]

Africa: Angola, Cameroon, Chad, Comoros, Congo, Djibouti, Ethiopia, Gabon, Ghana, Guinea, Kenya, Lesotho, Mali, Mauritania, Mozambique, Namibia, Rwanda, Somalia, South Africa, Uganda, Zaire, Zambia, Zimbabwe
The Americas: Argentina, Bolivia, Brazil, Chile, Colombia, El Salvador, Guatemala, Guyana, Haiti, Honduras, Mexico, Paraguay, Peru, Suriname, Uruguay
Asia: Afghanistan, Bangladesh, China, India, Indonesia and East Timor, Korea (Republic of), Pakistan, Philippines, Sri Lanka, Taiwan
Europe: Albania, Italy, Poland, Romania, Spain, Turkey, Union of Soviet Socialist Republics, Yugoslavia
The Middle East and North Africa: Bahrain, Egypt, Iran, Iraq, Israel and the Occupied Territories, Libya, Morocco and Western Sahara, Saudi Arabia, Syria, Tunisia

TABLE III. Types of Torture[13,14]

Beating, kicking, striking with objects (to torso and/or genitalia and/or head)
Threats, humiliation
Application of electricity (to torso, genitalia, mouth) with pointed electrodes, cattle prods, or shock baton
Blindfolding
Mock execution
Made to witness others being tortured
Submarino—submersion of head in water (often filthy or polluted with excreta) with near drowning
Isolation (over 48 hours)
Starvation (over 48 hours)
Sleep deprivation
La bandera—suspension from a rod by hands and feet
Rape—mutilation of genitalia, insertion of foreign bodies into vagina or rectum
Burning—application of cigarettes, electrically heated rods, hot oil, quicklime, corrosive acid
Falanga—beating the soles of the feet with rods
Rope bondage—tightening of ropes over hours
Telephono—striking of blows at the victim's ears
Forced standing (over 48 hours)
Throwing of urine or feces at victim
Medicine administration (nontherapeutic)
Traction alopecia (lifting by hair)
Needles under toenails or fingernails
Deprivation of water—providing only filthy, salty, or soapy water
Forced extraction of teeth
Prevention of urination and defecation
Deprivation of medical care
Continuous exposure to bright light or noise
Total sensory deprivation
Cold exposure

Since 1982, torture victims, mainly refugees, have been treated at the Danish Centre for the Rehabilitation of Torture Victims. A series of over 200 patients[14] yielded the data presented in Table IV. Virtually all patients seen in the Danish study had emotional difficulties, most commonly poor sleep, nightmares, inability to concentrate, poor libido, diffuse anxiety, and general depression.

In a study by Allodi and Cowgill,[17] 41 refugee torture victims entering Canada between 1977 and 1979 from Latin America were interviewed. The great majority suffered from somatic complaints, anxiety, and depression. Three quarters had objective evidence of physical damage. Four attempted suicide.

Allodi[18] surveyed three studies of the psychiatric effects in children and families of political persecution and torture. He found that the children reacted most commonly with social withdrawal, chronic fear, depressive moods, clinging and overdependent behavior, sleep disorders, somatic complaints, and an arrest or regression in social habits and school performance (most of these children were under six years of age). Remarkably, he noted reactions of anger against the torturers in none of the torture victims. "Most commonly the victims were bewildered by

TABLE IV. Sequelae of Torture[14]

Neurologic or mental sequelae
Anxiety, depression, fear
Lability, introversion
Lethargy, fatigue
Loss of memory
Inability to concentrate
Sleep difficulties
Nightmares
Headaches
Visual disturbances
Vertigo
Paresthesias
Sexual disturbances
Somatic and psychosomatic sequelae
Gastrointestinal disorders
Skin lesions
Joint pains
Cardiopulmonary problems
Gait disturbances
Pains related to specific traumas

the cruelty of their oppressors and deeply ashamed and humiliated, which suggests that the main aim of torture, the humiliation and dehumanisation of the victim, had been achieved."[18]

ARE HEALTH PROFESSIONALS DIRECTLY INVOLVED IN TORTURE?

A Working Party of the British Medical Association Council reviewed evidence addressing this issue, which was published in 1986 in *The Torture Report.*[19] The following review highlights the pertinent findings.

The Working Party found instances in several countries of physicians examining detainees immediately prior to torture. In Chile and Turkey, in some cases the degree of torture a detainee was considered able to withstand was certified. In Uruguay, it was similarly found that examinations were used to avoid deaths from torture, in a sense protecting the torturer from outright murder.[20]

The Working Party found evidence that health professionals were monitoring torture and ignoring clear evidence of torture in their patients. In Uruguay, a male nurse was always on duty during torture sessions, and a doctor was available. In Brazil, Uruguay, and other countries, physicians have falsified examination records and death certificates to hide torture.[21]

The Committee for the Defence of Human Rights in Uruguay reported that a School of Intelligence was set up in Montevideo in 1975 which ran "practical courses in torture." These courses were attended by officers from El Salvador, Guatemala, Costa Rica, and Paraguay. Students took part in torturing detainees brought in for that purpose. The detainees often lost consciousness, and the course doctor, a major in the army, would revive the victims so that they could be tortured again.

The Colegio Médico de Chile has evidence against several physicians and, despite the dictatorship of Pinochet, has taken disciplinary action against physicians found to have engaged in torture. The danger such an approach holds for the physicians of the Colegio was dramatized by the fate of its Secretary General, Francisco Rivas Larrain. In a lecture given in August 1986 in Copenhagen at a symposium on doctors, ethics, and torture he stated, "During the dictatorship, over 80 physicians directly or indirectly participated in acts of ill-treatment, humiliation, or torture; the great majority of those physicians belonged to the armed forces and were attached to the security agencies."[22] This presentation had to be read to the meeting on Dr Rivas Larrain's behalf, as he had earlier that summer been arrested by the Chilean authorities on unspecified charges.

A general practitioner from Damascus, Syria, reported being tortured during 29 months of imprisonment. At one point a physician called a halt to a torture session telling the torturer that the victim could take no more. The next day torture caused him to lose consciousness and he was brought to the al-Mezza military hospital. Two days later interrogation resumed.

The case of Steven Biko of South Africa has become well known. The black rights activist failed to receive adequate treatment for head injuries sustained at the hands of the security police, physicians allowing him to travel hundreds of miles by police jeep instead. Similar evidence of physicians monitoring torture or ignoring its signs has been noted in Colombia, Spain, Brazil, and Tanzania.

The Working Party found evidence that physicians take active part in torture itself. The medical experiments performed by the Axis powers during the Second World War are a constant reminder of the vulnerability of the medical profession. The case of the Nazi doctors at Auschwitz and other concentration camps has been widely reported in the West. On the Manchurian Plain, Japanese army doctors performed experiments on Chinese and Korean soldiers in which bubonic plague, cholera, syphilis, and other microbes were injected to compare the resistance of various nationalities and races to disease. All of the subjects died.

Currently, it is through the field of psychiatry that physicians are most directly involved in abuse. In the USSR, and other countries to a much lesser extent, political and other dissidents have been confined to psychiatric hospitals for varying periods of time. Reasons for this involuntary confinement have included persistently making religious craft articles; criticizing the government in the presence of workmates and other private citizens; and persistently seeking permission to emigrate. Alexei Nikitin, a mining engineer, was confined to a psychiatric hospital after being charged with "disseminating anti-Soviet propaganda" after a series of attempts by Nikitin to protest unsafe working conditions and inadequate overtime pay. While in the hospital, he received a number of drugs including sulfizine, which brings on intense fever, excruciating pain, convulsions, and disorientation.[23]

Nikitin was one of 55 dissidents examined by the psychiatrists Anatoly Koryagin and Alexander Volshanovich, members of the unofficial Working Commission to Investigate the Use of Psychiatry for Political Purposes. Four years after the Commission was founded, the Soviet authorities had imprisoned or forced into exile all the Commission's members, who had insisted on the release of dissidents forcibly confined to psychiatric hospitals for what they claimed were political rather than genuine medical reasons. Koryagin himself suffered many brutal years in prison before emigration to the West.[24]

Psychiatrists in the USSR commonly rely on a system created by Dr Andrei Snezhnevsky, director of the Institute of Psychiatry of the Academy of Medical Sciences, in their approach to diagnosing schizophrenia. They divide the disease into three forms—periodic, shiftlike, and continuous—and these in turn are subtyped. Within the continuous form is the subtype "sluggish," with characteristics including neurotic behavior, self-consciousness, introspectiveness, obsessive doubts, conflicts with parental and other authorities, and "reformism." Since schizophrenia is a disease in which the sufferers are deemed legally "not responsible" for their actions, that section of the USSR penal code allowing compulsory medical measures for such patients can be applied to all persons initially charged with illegal anti-state activities and then diagnosed schizophrenic.[25,26]

Bloch and Reddaway[26] claim to have documented in their book *Soviet Psychiatric Abuse* almost 500 cases of dissenters being forcibly hospitalized. Apart from the above-mentioned drug therapy, beatings, insulin shock therapy, and prolonged isolation have been used in these hospitals.[27]

Reports have come from both Iran and Iraq of medical personnel draining blood from prisoners just prior to execution. In Iraq, a physician states he was aware of approximately 1,000 such operations in 1982 and 1983.

> I found there two persons in a shocked state. . .and who exhibited air hunger with rapid thready pulse and cold clammy skin. The prison doctor told me that those prisoners were criminals and that he bled them under the influence of hypnotic drugs in order to benefit from their blood before they are executed. The doctor also told me that he has directives from security headquarters to use this method with important political persons so as to give the reason for the subsequent death as "heart failure."

HEALTH PROFESSIONALS AS VICTIMS

Physicians can themselves fall victim to human rights violations and even torture as a consequence of their work. The fate of Dr Koryagin was noted above. Dr Joel Filártiga runs the largest private rural health clinic for the poor in Paraguay. "I believe that all people have a basic human right to medical care and, given the reality that all but a handful of doctors refuse to leave the comforts of the cities to practice medicine in the countryside it was the only decision I could make and still retain my integrity."[10(p 83)]

In March 1976, the police inspector of Asunción, Américo Peña-Irala, suspecting that Filártiga was an accomplice of a Paraguayan guerrilla group, took the physician's 17-year-old son, Joelito, to a police station and taped an interrogation session. Peña and three other policemen beat and whipped the boy, then attached electrodes to his fingers and penis and gave him electric shocks, finally causing his death (no evidence against Filártiga was obtained). The mutilated corpse was given to Joelito's sister. In the ensuing legal case one witness disappeared, as did a Filártiga family member trying to find her, two other family members were jailed, and Filártiga's lawyer was disbarred. Through a unique confluence of events, Filártiga was able to bring suit against Peña in New York State, and in the years 1980 to 1984 Filártiga won a series of legal decisions (the Second Circuit ruling referred to above being one of these) and finally a large monetary award.[10(pp 79–100)]

A 1988 circular of Amnesty International stated that Joel Filártiga has been receiving anonymous telephone calls threatening him and his family. All his medical instruments were stolen in late 1987. On February 22, 1988, a bus collided with his ambulance, causing him numerous injuries. A call shortly thereafter said this was a sample of what awaited him.[28]

WHAT CAN BE DONE AGAINST TORTURE?

A number of actions can be taken specifically by health care workers, alone and as a group. As the recent review by Goldfeld et al[13] attests, many physicians have taken a serious and long term interest in the torture victim and his or her treatment.

There are several medical centers around the world that deal specifically with the torture victim. Major international centers for research and treatment are located in Copenhagen, Toronto, and Minneapolis-St Paul.[29] A center for the rehabilitation of torture victims is currently being organized in New Jersey under the direction of Dr R. Krakauer.

Many scientists have been able to put their specialized skills in the service of human rights workers fighting torture. Clyde Snow, a US forensic anthropologist, has aided the search for the "disappeared" in Argentina, even testifying about the identity of remains of victims, sometimes found in mass graves, and the probable causes of their death.[21(pp 110–113),30] An international group of geneticists has been assisting the Grandmothers of the Plaza de Mayo, a human rights group in Argentina, search for children who were abducted with their parents or born in detention and later separated from their mothers (who were often killed). These children and neonates were on occasion adopted by the very tormentors who held their parents captive. Through sophisticated hemogenetic analysis true grandpaternity can be shown to 99.9% assurance using HLA typing alone, and the children can be returned to their true families.[31]

National medical organizations can take a stand against torture. In June 1986, the American Medical Association adopted Resolution 91, "Persecution of Physicians for Political Reasons and Participation by Doctors in Violations of Human Rights," which endorses the Declaration of Tokyo of the World Medical Association, opposes physician participation in torture, and expresses solidarity with physicians who have been subjected to imprisonment or torture because of their humanitarian efforts to improve the health of their patients.

Extremely courageous medical associations in countries such as Chile and Turkey have spoken out against unjust practices not from afar or after the fact but while still living under oppressive and brutal regimes.

Several international professional associations have recently adopted Codes of Ethics, often explicitly dealing with torture. The Declaration of Tokyo (World Medical Association, 1975), the Declaration of Hawaii (World Psychiatric Association, 1977), and the Resolution of Singapore: Role of the Nurse in the Care of Detainees and Prisoners (International Council of Nurses, 1975) are statements by doctors, psychiatrists, and nurses calling for respect for patients' human rights and condemning torture.

More specifically focused on human rights concerns are such organizations as Amnesty International and Physicians for Human Rights. Amnesty International, active for over two decades and now comprising over 500,000 supporters in some 160 countries and territories, has as its mission universal opposition to torture and execution, fair trials for political prisoners, and release of "prisoners of conscience," people detained on the basis of their beliefs, color, sex, ethnic origin, language, or religion who have not advocated violence. Amnesty International/USA currently has a health professional network open to all concerned health care workers.

Physicians for Human Rights is a new organization open to all concerned with the work of health care professionals in supporting human rights. It has already sponsored missions to human rights trouble spots around the globe.

Thus, even this brief overview suggests the extremely varied activities each of us can engage in to bring an end to torture in the world today.

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Fetal neglect and abuse

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In a modern society, the phrase "fetal neglect and abuse" has the connotation of violent acts being committed against the fetus. Criminal or unlawful harm to a pregnant woman with secondary damage to her growing embryo is not rare. However, improper actions on the part of the mother that can injure the fetus are more common. Acts of this nature may be unintentional, iatrogenic, or deliberate.

The intrauterine environment is designed to protect the fetus from external injury and to assure proper nutrition and disposal of catabolic products. Modern developments such as food processing, "technological progress," and even medical therapies, however, have been able to overcome the uterine protective mechanisms. Fetal homeostasis is heavily dependent on the maternal habitat and is quite susceptible to profound damage. After fertilization

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and throughout pregnancy, the fetus may be subject to the effects of poor maternal nutrition or maternal use of drugs including alcohol and nicotine from cigarette smoking. It may also be the victim of harmful teratogenic effects.

Injuries can damage the developing fetus in varying degrees, depending on the stage of development of the embryo.[1] For example, maternal rubella infection during the sixth week of gestation results in fetal cataracts; during the ninth week and thereafter, in deafness; and during the fifth to the tenth weeks, in cardiac defects and dental anomalies; and abnormalities of the central nervous system are seen during and after infections in the second trimester.[2,3]

DEVELOPMENT OF BRAIN MATURATION

Human beings are differentiated from other species by their intelligence. Thus, it seems useful to detail the process of neuronal and glial proliferation and neuronal migration. The neuroblast or primitive cell undergoes major proliferative events between the eighth and 16th weeks of gestation.[4] Glial multiplication peaks from the 20th to the 40th week.[5,6] The neuroblast develops into the bipolar neuroblast, the multipolar neuroblast, and finally into the adult cell or neuron. The cerebral cortex develops from the pallidum (near the corpus striatum). The neuroepithelial cells release wave after wave of neuroblasts from the subependymal region, also known as the germinal matrix. These neuroblasts migrate and the cells differentiate into fully mature neurons. Teratogenic or environmental factors, such as radiation, anticonvulsants, alcoholism, maternal smoking, and metabolic errors, may have a deleterious effect during this crucial proliferative stage.

In neuronal migration, millions of cells move from the subependymal area to their zone of permanent residency. The peak migration occurs from the third to the fifth month, but it may be detected as early as the second month and beyond the fifth month. It is a highly ordered process that may be profoundly changed under the action of exogenous factors.[7] By the 24th week of gestation, neuronal distribution in the human cerebral cortex is essentially completed, but injury or death of neurons may occur up to the moment of delivery and beyond. Insults that affect the fetal brain, including hypoxia-ischemia, malnutrition, and/or the effects of different drugs, have been well documented in animals[8-10] and in man.[11]

Since the embryo and the fetus are very susceptible to harm, there is an understandable concern for accountability. This may lead to a moral and legal conflict between the constitutional rights of the parents on the one hand and those of the unborn child on the other.

CRIMINAL VIOLENCE AGAINST THE PREGNANCY

During 1986, 46 fetal deaths were reported in New York State due to physical assault of pregnant women, leading to the involuntary termination of the pregnancies. These violent acts are rarely reported, despite the fact that feticide is a crime[12] and third parties responsible for the negligent or criminal destruction of fetuses are criminally liable. Although laws have been developed to protect pregnant women's interests, such laws potentially extend rights to an unborn fetus. Civil courts in almost every state recognize the rights of children to bring claims for injuries inflicted prior to birth if the child survives the abuse and is born alive. Because the third party is often a member of the family, claims are rarely brought to the courts. Proper education and information gathering may provide a more accurate appraisal of this type of violence.

DELIBERATE ACTS AGAINST THE FETUS

A woman has constitutional rights to autonomy and privacy with respect to procreation.[13] Nevertheless, like any other right, it is not absolute. Once a woman decides to conceive and chooses not to abort the conceptus but to continue the pregnancy, she becomes legally and morally obligated to bring the child into the world as healthy as possible, and the viable fetus acquires certain rights.[14] The mother's life style becomes the life style of the fetus, and it may be the right of the fetus to have the mother conduct her life in ways that do not adversely affect or injure it. At the same time, it is unreasonable to force a woman to refrain from all contact that might remotely injure her fetus. An individual is under an obligation to refrain from those actions that are unreasonably dangerous to others. How much knowledge do we expect a reasonable person to have concerning the risks to an unborn fetus? Unless facts are properly balanced, a woman might be held civilly or criminally liable for fetal injuries that are due to maternal negligence, such as automobile or household accidents; failing to eat properly; exposing herself to infectious diseases or workplace hazards; engaging in inappropriate or ill-timed sexual intercourse; and residing at high altitudes for prolonged periods. All of these possibilities might cause the mother to live in constant fear. Nonetheless, deliberate actions that result in severe fetal injuries, such as the use of heroin or cocaine, chronic alcoholism, and heavy smoking should be considered unreasonable and injurious in light of the well known risks to the fetus.

Drug Use. Narcotism is primarily caused by heroin, methadone, and cocaine abuse. Other abusive substances, however, such as phencyclidine (angel dust) and airplane glue are potentially dangerous to the fetus. A recent review of birth certificates during 1986 by the New York City Department of Health Division of Biostatistics revealed that maternal narcotism, heroin, methadone, and cocaine abuse, was mentioned in 2,068 births for a rate of 17.0 per 1,000 live births. For 1978–1984, mortality for those infants was 40.0 per 1,000 live births, 2.5 times the overall rate for New York City (15.5/1,000 live births). The same review indicates that neonatal morbidity expressed as the percent of infants born under 2,500 grams to women with maternal narcotism is almost three times as high as it was citywide (personal communication, New York City Department of Health). The dysmorphogenesis caused by narcotism is manifested as severe intrauterine growth retardation and in many instances as microcephaly.[15] Postnatal withdrawal symptoms in the fetus are almost always present, and reports of sudden infant death syndrome following prenatal drug abuse are increasing. Cocaine-abused fetuses may be born with renal malformations, and even one single intravenous administration of cocaine may cause fetal cerebral cortical atrophy.[16]

A major new concern is the high frequency of human immunodeficiency virus (HIV) seroprevalence in drug addicts. A 1987 study of newborns conducted by the New

York State Department of Health revealed that of the first 19,157 newborn blood specimens taken in the City of New York, 164 infants were HIV antibody positive.

Alcoholism. Alcoholic women are known to give birth to infants with a conglomerate of symptoms and signs, including growth retardation, microcephaly, mental deficiency, facial abnormality, and malformations of the limbs and organs.[17] Fetal alcohol syndrome is believed to occur once in every 750 to 1,000 births.[18] Although there are no reported cases in children of moderate drinkers, there is a broad spectrum of alcohol-related birth defects, with the full-blown syndrome and perinatal death at one end of the spectrum, and subtle behavioral anomalies at the other.

New York State's Division of Alcoholism and Alcohol Abuse published a prevalence report in October 1982[18] in which it stated that there are more than 250,000 females in New York State considered problem drinkers, in the age range 18 to 40 years. Alcohol abuse is the most common drug use problem among American youth. Teenagers begin drinking as early as ten years of age, and recent national surveys indicate that more than 50% of youth report drinking at least once a week, 14.8% are classified as heavy drinkers, and 50% have an alcoholic sibling or parent.

There are one million teenage pregnancies in the United States every year. In 1981, more than 10,000 babies were born in New York to mothers ten to 17 years of age, an age group known to be at high risk because of heavy alcohol abuse. Yet, the glamorization of drinking via the advertising media is expanding, and some commercials dealing with alcoholic beverages are voted as most appealing in viewer surveys.

Smoking. A surgeon general's warning printed on cigarette packages reads, "Smoking By Pregnant Women May Result in Fetal Injury, Premature Birth, And Low Birth Weight." It is now generally accepted that maternal smoking has deleterious effects on the fetus. Smoking increases the maternal heart rate and blood pressure. The fetal heart rate also increases and may become irregular. These changes are associated with a decrease in placental blood flow. The cardiovascular changes are probably secondary to the nicotine effect, but a rise in maternal carbon monoxide also increases fetal carboxyhemoglobin saturation producing secondary fetal hypoxia.[19]

Twenty-five percent or more of infants are born with moderate to severe intrauterine growth retardation if the mother smokes ten or more cigarettes a day. Although it is a well documented fact that cigarette smoking is hazardous to human health, a glance at one's favorite newspaper or magazine still reveals advertising that can have a significant impact and influence on one's smoking habits.

The United States courts have begun to process lawsuits involving wrongful, deadly, or handicapping conditions attributed to narcotism, alcoholism, and smoking.

IATROGENIC FETAL DAMAGE

During the course of a pregnancy, maternal illness may require the administration of medications. Historically, we learned from thalidomide and tetracycline how indicated medications present in the mother's bloodstream could cause serious fetal malformations. Some of the noxious effects may occur years after prenatal administration, as was seen with diethylstilbestrol (DES).

A delicate balance between the benefits to the mother and the teratogenic or adverse effects on the fetus must be individualized for each patient before a prenatal treatment is prescribed. A special report by the Perinatal Collaborative Projects[20] was widely distributed and contains much important information relevant to this issue.

The fetus is most susceptible to the teratogenic effects of drugs prior to the 45th day after conception. Antimicrobials, antiparasitics, antipyretics, sedatives, anticonvulsants, diuretics, vitamins, hormones, and radiologic diagnostic contrast material should be used with caution because all may adversely affect the fetus.

Gingivitis and dental caries are not uncommon occurrences during pregnancy. To avoid them and the need for dental anesthesia, young women of child-bearing age should be advised to maintain dental fitness and good oral hygiene. Relative analgesia (nitrous oxide sedation) is commonly used in dentistry but has been reported to cause teratogenic effects in animals if used in the first trimester.[21] The federal Food and Drug Administration,[22] in discussing the occupational hazards of chronic exposure to nitrous oxide for female health workers of child-bearing age, notes an increase in spontaneous abortion rates among this population. A similar increase in spontaneous abortions has been reported in wives of dentists using nitrous oxide anesthesia in their practices. At present, the use of nitrous oxide during the first trimester of pregnancy should be discouraged.

ENVIRONMENTAL CAUSES OF BIRTH DEFECTS IN HUMANS

Ionizing radiation was the first environmental agent known to cause teratogenic complications in humans. People exposed to radiation at Hiroshima and Nagasaki reported a higher than expected frequency of congenital abnormalities in their offspring. Exposure of pregnant women to x-rays should be avoided.

Methyl mercury and polychlorinated biphenyl (PCB) are found in ocean and lake fish, and mothers who eat large amounts of fish, although not affected themselves, may give birth to infants affected by the chemicals. Thus, during pregnancy, women should avoid swordfish, marlin, tuna, halibut, red snapper, mackerel, fresh water trout, bass, and pike.

Lead may be a contaminant of drinking water, and recent studies have shown that low-level fetal lead exposure may have a negative effect on neurobehavioral development during early infancy.[23]

EFFECTS OF AEROBIC EXERCISES ON THE PREGNANT WOMAN AND FETUS

The quest for physical fitness lures lay people as well as the medical profession. It is common practice to advise women who have exercised prior to pregnancy to continue to do so and women who lead a more sedentary life style to begin exercising. However, hormonal changes cause connective tissue and ligaments to stretch more easily, making articulations more prone to injury. Studies on the effects of aerobic exercise in pregnancy have shown a decrease in the uterine blood flow during exercise; an increase in arterial epinephrine and norepinephrine levels; an increase in oxygen consumption; a significant decrease in blood glucose; and an increase in maternal temperature.[24] Consequently, exercise should be attempted only after receiving the proper medical supervision. Brisk walking, swimming, and bicycling are probably the safest, if done in moderation.

NUTRITION AND PREGNANCY

The recommended weight gain for the entire pregnancy is 20 to 30 pounds for normal-weight women. It is important to have a properly balanced diet, but unfortunately the eating habits of Americans, especially the young, are not necessarily optimal for a healthy pregnancy. The tendency to eat "fast" foods may be complicated by the fact that in the next several years American companies will be introducing a number of synthetic food additives to the market which are primarily substitutes for sugars and fats.

We incorporate coffee into our regular diet with the misconception that it is a safe drink. However, a 1987 study of the relationship between low birth weight and caffeine consumption during pregnancy indicated that maternal caffeine intake affects the birth weight of the child, causing growth retardation, but only if the mother ingests more than 300 mg of caffeine daily (three cups of coffee, tea, soft drinks, etc).[25] The number of products on the market that contain caffeine without any warning notice is very high.

FIGURE 1. Poverty is the most important cause of abuse to the fetus, child, or person of any age (courtesy of David Burnett, Contact Press Images © 1988).

Nutritional disorders such as anorexia nervosa and bulimia have been shown to cause fetal malnutrition. However, malnutrition among pregnant women, especially in the low socioeconomic group, accounts for the most common and important cause of fetal malnutrition, and it may have catastrophic effects on the developing embryo. Poverty is abusive to fetuses, children, and people of all ages (Fig 1). Hence, it is our responsibility to maintain an effort to eliminate the social illnesses that affect the impoverished in our communities.

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Child abuse: The physician's responsibility

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Child maltreatment is not a new phenomenon. It has existed since the beginning of time. The tales of children being maltreated and battered by their parents abound in myth, legend, and literature. The Bible has references to divine destruction of the firstborn, offerings of children to a capricious god, and the child as a nuisance to be abandoned or a chattel to be used and abused. Both Greeks and Romans consigned infants to death by exposure or cast them adrift on a river. In other civilizations, children were mutilated by their parents to inspire sympathy and thereby be more successful beggars. Even today there are countries where infants are discarded at birth because the family has too many mouths to feed, the child is defective, or the child is unwanted.

The problem has seen many brief periods of awareness in the last 100 years. However, it was not until 1962 that the topic of child abuse gained medical attention when C. Henry Kempe and his associates[1] coined and publicized the term "battered-child syndrome" and proposed that such abuse was a frequent but unsuspected cause of permanent injury or death to these children. In a 1963 paper entitled "The maltreatment syndrome in children,"[2] the scope of the problem was widened with an all-encompassing description of child abuse and neglect. In that article it was indicated that the "battered child" phenomenon is the last phase of the child maltreatment spectrum.

DEFINITION AND CLASSIFICATION OF CHILD ABUSE

Child maltreatment as defined today spans a wide spectrum of commissions and omissions by parents and guardians. We encounter emotional or verbal abuse, physical abuse (Table I), and sexual abuse (Table II). The broad term "neglect" has come to include emotional neglect or maternal deprivation (Table III), physical neglect, malnutrition, inappropriate clothing, lack of supervision, medical neglect, educational neglect, and abandonment (Table IV).

In all these cases, inadequate parenting is the major issue. Considering the maltreatment of children in the context of the various signs of abuse and neglect outlined in the tables, it is clear that today, in the 1980s, child maltreatment is a major problem confronting a large number of parents caught in the web of a multitroubled family unit. Child abuse is not confined to impoverished families, it is an issue that affects just as many middle and upper income families.

The incidence of abuse and neglect of children has been increasing at the rate of 15–20% every year throughout the United States. The neglect and abuse of children has become a major pediatric problem.[3] The National Center for the Prevention of Child Abuse and Neglect estimates that 4,000 children die annually in this country as a result of child abuse and neglect. According to official estimates from the National Center in Washington, DC,[3] a million children are being maltreated by their parents each year, as many as 200,000 are sexually exploited, and probably 200,000 to 300,000 are psychologically abused. Child maltreatment is a major medical-social problem that is becoming more entrenched in our society. Physicians in private practice, hospitals, and clinics serve as the front line of protection for the thousands of children who are being endangered by inflicted abuse and neglect. Recognition of the maltreated child is the foundation stone on which all future happenings depend.

Physicians are mandated under New York State and other state child abuse laws to report all suspected cases of

TABLE I. Indicators of Physical Abuse of Children

Unexplained bruises and welts:
- on face, lips, mouth
- on torso, back, buttocks, thighs
- in various stages of healing
- clustered, forming regular patterns, reflecting shape of article used to inflict (electric cord, belt buckle)
- on several different surface areas
- regularly appear after absence, weekend, or vacation

Unexplained burns:
- cigar, cigarette burns, especially on soles, palms, back or buttocks
- immersion burns (sock-like, glove-like, doughnut shaped on buttocks or genitalia)
- patterned like electric burner, iron, etc.
- rope burns on arms, legs, neck or torso

Unexplained fractures:
- to skull, nose, facial structure
- in various stages of healing
- multiple or spiral fractures
- swollen or tender limbs

Unexplained lacerations or abrasions:
- to mouth, lips, gums, eyes
- to external genitalia
- on backs or arms, legs or torso

Human bite marks

Frequent injuries that are "accidental" or "unexplained"

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TABLE II. Indicators of Sexual Abuse of Children

Child's Physical Indicators
- Difficulty in walking or sitting
- Torn, stained, or bloody underclothing
- Pain or itching in genital area
- Bruises or bleeding in external genitalia, vaginal or anal areas
- Venereal disease (especially in pre-teens)
- Pregnancy

Child's Behavioral Indicators
- Unwilling to change for gym or to participate in physical education class
- Withdrawal, fantasy, or infantile behavior
- Bizarre, sophisticated, or unusual sexual behavior or knowledge
- Poor peer relationships
- Delinquent or runaway
- Reports sexual assault by caretaker

Parent's Behavioral Indicators
- Very protective or jealous of child
- Encourages child to engage in prostitution or sexual acts in the presence of caretaker
- Misuses alcohol or other drugs
- Is geographically isolated and/or lacking in social and emotional contacts outside the family
- Has low self-esteem

child abuse and neglect that come before them in their professional capacity.[4] To encourage physicians to recognize and report cases of child maltreatment all 50 states and the District of Columbia have provided physicians with immunity from suit if reports prove to be unfounded. What is not well known is the fact that a doctor who knowingly fails to report a possible case of child abuse or neglect can be charged and fined and be liable to malpractice suits. A California supreme court ruling,[5] the first of its kind in the nation, established the basic principle that a battered child, through an attorney, may ask a jury to award damages for a physician's negligence if further injury follows the doctor's failure to report observed or even only suspected abuse. This type of malpractice legal action will become more frequent as lawyers become more active in recognizing negligence among physicians who fail to report actual or suspected cases of child maltreatment.

TABLE III. Indicators of Emotional Neglect of Children

Child's Physical Indicators

Exhibits one or more of the following:
- Speech disorders
- Lags in physical development
- Failure to thrive

Child's Behavioral Indicators
- Habit disorders (sucking, biting, rocking, etc)
- Conduct disorders (antisocial, destructive, etc)
- Neurotic traits (sleep disorders, speech disorders, inhibition of play)
- Psychoneurotic reactions (hysteria, obsessions, compulsions, phobias, hypochrondia)
- Behavioral extremes: compliant, passive, shy, aggressive, demanding
- Overly adaptive behavior: inappropriately adult or inappropriately infantile
- Developmental lags (mental, emotional)
- Attempted suicide

Parent's Behavioral Indicators
- Treats children in the family unequally
- Doesn't seem to care much about child's problems
- Blames or belittles child
- Is cold and rejecting
- Withholds love
- Behaves inconsistently

TABLE IV. Indicators of Parental Neglect of Children

Child's Physical Indicators
- Consistent hunger, poor hygiene, inappropriate dress
- Consistent lack of supervision, especially in dangerous activities or long periods
- Unattended physical problems or medical or dental needs
- Abandonment
- Often tired or listless

Child's Behavioral Indicators
- Begging or stealing food
- Extended stays in school (early arrival and late departure)
- Attendance at school infrequent
- Constant fatigue, listlessness, or falling asleep in class
- Alcohol or drug abuse
- States there is no caretaker

Parent's Behavioral Indicators
- Misuses alcohol or drugs
- Has disorganized, upsetting home life
- Is apathetic, feels nothing will change
- Is isolated from friends, relatives, neighbors
- Has long term chronic illness
- Cannot be found
- Has history of neglect as a child
- Exposes child to unsafe living conditions
- Evidences limited intellectual capacity

The American Academy of Pediatrics over the years has encouraged the mandatory reporting of suspected child abuse cases and has encouraged doctors in private practice who are reluctant to report, to refer children that they suspect of being abused or neglected to local hospitals. The hospital then assumes the responsibilities of protecting the child, reporting the case to the child protective agency in the community, and providing the needed human support services for the family. This type of physician-to-hospital referral can only be successful if the hospital is staffed with a multidisciplinary team of trained individuals assigned to provide child abuse case management. Physicians who have ambivalent feelings about reporting cases of child abuse and/or are unwilling to ask for help allow a child to suffer. Making a diagnosis of child maltreatment can be equally life saving as making a diagnosis of Wilm tumor—immediate intervention and treatment may prevent unnecessary suffering and death in both cases. Child abuse is far more common than many other life threatening pediatric problems and the fatality rate many times higher.

The maltreated child is often brought to the hospital or private physician with a history of failure to thrive, malnutrition, poor skin hygiene, irritability, psychological abnormalities, and other signs of obvious emotional and physical neglect. The more severely abused children are seen in the emergency rooms of hospitals with external evidences of body trauma, bruises, abrasions, cuts, lacerations, burns, ocular injuries, soft tissue swellings, and hematomas. Hypernatremic dehydration, following periodic water deprivation by psychotic mothers, has been reported as a form of child abuse. Inability to move certain extremities because of dislocations and fractures associated with neurologic signs of intracranial damage can be additional signs of inflicted trauma. Other clinical signs and symptoms attributed to inflicted abuse may include injury to viscera. Abdominal trauma may result in unexplained ruptures of stomach, bowel, liver, or pancreas, with manifestations of an acute abdomen. Chest injuries caused by abuse usually take the form of rib fractures and resultant complications. Any laceration, bruise, or injury to the genital area of a child should cause suspicion of sexual abuse. Children manifesting the maltreatment syndrome give evidence of one or more of these complaints. Those children with more severe maltreatment injuries arrive at the hospital or physician's office in coma or having convulsions. Manual shaking to quiet a crying infant can lead to the "shaking baby syndrome" which can result in major central nervous system injury and death, especially in an infant under 15 months of age. In these cases, the diagnostic and treatment ability of the physician, coupled with child protective community services, can prevent more serious injuries of inflicted battering that are significant causes of childhood deaths.

Nonaccidental poisoning in children must be considered a possibility in any child with unexplained symptoms. Parents who poison their children may have severe psychiatric problems or may be drug abusers. Child neglect encompasses a spectrum of parental omissions which in many cases can be as serious as inflicted abuse.

Although parental neglect and abuse may occur at any age, the most severe physical injuries are inflicted on children under six years of age. In the last decade, the average age of the abused victim has increased significantly. More adolescents are becoming victims of some form of physical, sexual, and emotional abuse in their homes but are often reluctant to report their abusive parents. Of the children reported to have been maltreated, 32% are under six years of age, 27% are between nine and ten years, 27% are between ten and 14 years, and 14% are between 15 and 18 years of age.[3]

Many of these abused children are perceived by their parents as being different, bad, selfish, or hard to discipline. Some children are more vulnerable than others, particularly low birth weight children, handicapped children, and children with defiant or hyperactive behavior. The term "handicapped children" covers a wide range of developmental disabilities that include mental and physical retardation, cerebral palsy, epilepsy, autism, and severe dyslexia.

A child should be suspected of being abused or neglected if several of the following factors are in evidence:

- The child seems unduly afraid, especially of his or her parents.
- The child is kept confined—as in a crib or playpen or cage—for over-long periods of time.
- The child shows evidence of repeated skin or other injuries.
- The child's injuries are inappropriately treated in terms of bandages and medication.
- The child appears undernourished.
- The child is given inappropriate food, drink, or medicine.
- The child is dressed inappropriately for weather conditions.
- The child is passive and avoids parental confrontation.
- The child is aggressive, demanding, and hyperactive.
- The child shows evidence of overall poor care.
- The child is constantly irritable and cries incessantly.
- The child takes over the role of parent and tries to be protective or attempts to take care of the parent's needs—"role reversal" behavior.

The abusive parent is most often the mother. One parent is usually the "active" batterer, and the other parent passively accepts the battering of the child. The average age of the abusive mother has been reported to be 26 years; and the average age of the father is 30 years.

The maltreated child is usually the victim of emotionally crippled parents, beginning with the unfortunate circumstances surrounding the parent's own childhood. The abusive parent frequently has a history of having been brutalized as a child so that pattern of violent behavior, if unbroken, is transmitted from generation to generation.

Divorce, separation, alcoholism, drug addiction, teenage pregnancies, mental retardation, recurring mental illness, unemployment, financial distress, and social isolation are important factors in the family structure of the abusing parents. These stress factors all play leading roles that cause potentially abusive parents to strike out at a "special child" during a crisis situation. These three components—a potentially abusive parent, a target child, and a sudden crisis—are the necessary ingredients that can trigger a child abusing and battering situation.

RESULTS OF CHILD ABUSE

If not properly managed, this distorted relationship between parents and children can be life threatening with profound consequences to the child, family, and society that are both immediate and long term. As a result we are faced with the prospect of thousands of children growing up unhappy, maltreated, deprived, and suicidal. The teenage delinquents, alcoholics, drug addicts, and prostitutes on the streets are for the most part products of a multitroubled violent home environment in which they were the victims of inflicted abuse and neglect. These are the throwaway children, the runaways, and the "kicked out" kids. These are the children that assault, rob, and murder. There are thousands of such troubled, emotionally damaged youths that are finding it difficult with or without their parents. Normal lines of communication between these parents and their children are nonexistent and they have been replaced by harsh words, unfair punishment, and physical abuse which intensifies and perpetuates a vicious cycle of violence breeding violence from one generation to the next.

THE PHYSICIAN'S RESPONSIBILITY

Many maltreated children are not taken to a physician or hospital for medical care until the child is in acute distress or the parents become alarmed about the possibility of the child's impending death. If the infant or child is brought to a physician or hospital, the history related by the parents is often at variance with the clinical picture and the physical findings noted on examination of the child. The physician often discovers that the mother has taken the child to various hospitals and doctors in an effort to negate any suspicions of parental abuse. Difficulty in obtaining an accurate history is often encountered, and the diagnosis is therefore dependent on the physical examination, x-ray findings, and social service investigation.

Once an injury has occurred, the physician's first and immediate responsibility is to the child. When the parents bring their abused or neglected child to the physician, early diagnosis and treatment are essential. The main reason for admitting the child to the hospital, other than for assessing the degree of injury, is to protect the child. It should be a straightforward admission for the purpose of evaluation. The medical and physical evaluation of the child must be handled thoroughly and expeditiously. Emergency care is often required if the child is acutely ill.

Accidental trauma is common in young children and should always be considered in the differential diagnosis in order to prevent faulty and unwarranted accusation of parents.

When the appropriate laboratory testings are evaluated and the medical and surgical consultations have been completed, the physician is confronted with the difficult task of making a diagnosis. If child abuse is suspected, it is essential for the physician to take color photographs of the body injuries at the time of hospitalization since such photographs are very helpful in documenting findings to various law enforcement agencies and the courts, after definitive diagnosis has been made.

The complex nature of child abuse as a symptom of family distress requires that no one person or profession be solely responsible for the management of these cases. Child abuse is no longer "one man's turf." A team of professionals representing a variety of disciplines is best equipped to manage the problems of child abuse and neglect.

Every hospital should have a child abuse committee providing a readily available multidisciplinary team of consultants, headed by a pediatrician, and including a nurse, a psychologist, a psychiatrist, a social worker, and a hospital administrator. This team must also assume the role of educating their colleagues and the members of allied fields in the community.

CONCLUSIONS

In suspected cases of child abuse and neglect the physician should: identify the child who may have been abused and/or neglected; admit the child to the hospital and take emergency measures needed to prevent further injury; provide medical evaluation and treatment—history, physical examination, skeletal survey, and photographs; request a social worker's report and appropriate surgical, psychological, and medical consultations; report the case to the appropriate department of social service and child protective unit or central registry; confer within 72 hours with members of the child abuse committee; and arrange a program of care for the child and the parents.

A comprehensive treatment plan for the family can include traditional psychotherapy and counseling for the parents, residential treatment, "reparenting" educational programs, substance abuse rehabilitation, and self-help programs such as Parents Anonymous. Multidisciplinary treatment and preventive programs, based on our current understanding of parental misbehavior, have been found to be effective with more than 70% of abusive parents.

Child abuse can be prevented. Parents can be reached before they harm their children. Programs that promote parental self-help, including crisis nurseries, drop-in centers, crisis intervention services, and Parents Anonymous, can reach parents missed by the reporting and investigation system.

Last but certainly not least, the best child abuse preventive is to teach people to be effective parents. If we are serious about effecting positive change in the fabric of family life and reversing the plight of our maltreated children, we must advocate a family life development curriculum in our primary, intermediate, and secondary schools. Parenting skills and child rearing techniques can be taught in conjunction with classes in human sexuality. Parent education can be as much a cure for child abuse as public health programs have been for tuberculosis, venereal diseases, and the contagious diseases of childhood.

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Abuse of the adolescent

Reverend Bruce Ritter, OFM CONV

Covenant House is an international child care agency with crisis shelters for homeless and runaway youths in New York, Toronto, Houston, Ft Lauderdale, New Orleans, Anchorage, Guatemala, Panama, and Honduras. Covenant House was founded on the lower East Side of New York City in the late 1960s and today offers food, clothing, shelter, medical and legal services, and counseling 24 hours a day to approximately 26,000 needy youths each year.

In this article I would like to describe why Covenant House got started, why these youngsters leave home, and what happens to them on the street. When I moved to the lower East Side of New York City in 1968 I had no intention whatsoever of caring for children. I was simply trying to be useful. The usefulness became apparent to me one night at about 2:00 in the morning in the middle of a blizzard when six children knocked on my door—four boys and two girls, all quite young, under 16. They asked if they could sleep on the floor of my apartment. I had no choice! The next morning one boy went outside and brought back four more youngsters and said to me,

> This is the rest of us—the rest of our family. They were afraid to come last night. They wanted us to check you out first. I told them that you didn't come on to us last night so that it was probably OK.

These ten children had been living in an abandoned building about three doors from where I was living. They were living with a bunch of junkies who were pimping them—that's how the junkies were supporting their habit—and these ten children were forced to make a pornographic film in Yonkers because they were hungry and they could not obtain any food unless they did so. The kids hated that so much that they ran away from the junkies and came down the street to my place. I could not find a single child care agency in New York City that would accept these children. I called at least two dozen agencies—public and private, Catholic, Protestant, and Jewish—and nobody would touch them. The children were either too old or too young, too sick or not sick enough, or they were from out of state. The real reason was (there were two, actually) they were not reimbursable (no one would pay for them). Essentially nobody in New York City operated a program for young prostitutes and hustlers, so I kept them. That was the beginning of Covenant House.

The Children at Covenant House

Those first ten children knocked on my door 20 years ago. This year, more than 26,000 children, adolescents, and teenagers just like that will knock at my door in New York and in the branches of Covenant House in various cities. They are not the traditional runaway kids that we usually think about. They are not Tom Sawyers: bright, energetic, creative, adventuresome boys or girls who leave a good, warm, loving home where they are really wanted and cared for. They do not run away because they are bored or want some excitement or want to learn more things. There are as many as one million children in the United States who every year leave their homes but the traditional runaway child never goes more than 50 miles from his hometown, rarely stays away more than two or three days, and almost always returns home to his family. The traditional runaway is very glad to be back home and his parents are very glad to receive him back. That is the Tom Sawyer kind of child. We at Covenant House see very few of such children and adolescents.

Almost all of the 26,000 children that will come to us this year are American street kids. They are the Huck Finns in our society. If one recalls the story of Huckleberry Finn, one remembers that he was literally almost beaten to death by his alcoholic father. Huck Finn received repeated, habitual beatings. When he was on the street, very few good things ever happened to him. He drank a lot. He very probably engaged in deviate sexual relationships. He was a very unhappy youngster. Those are the kinds of kids that come to Covenant House, the Huck Finns, not the Tom Sawyers.

The family profile of the average Covenant House youngster is, almost invariably, that of a single parent family, a child of divorced parents or of no marriage at all. These youngsters, mostly adolescents and teenagers, are almost always a product of the child welfare system and repeated placements, have long histories of involvement with the police and of failure in school, and long histories at home of physical and sexual abuse, alcoholism, and drug abuse. There is absolutely no mystery whatsoever as to why these youngsters leave home. They leave home when they are 13, 14, and 16 because it is simply too terri-

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fying a place for them to remain; it is too painful, it is too dangerous, and they run out into the streets. They are not runaway kids. They are walkout kids. They are throwaway kids; they are literally abandoned kids.

THE LIFE STYLE OF STREET CHILDREN

If one thinks of a runaway youngster as a boy or girl who essentially relates to his family and will eventually return home, one should think of a street youngster as a boy or girl who has definitely separated himself from his family, has abandoned any pretense that he has a family, and now relates almost entirely to the street. Such a youngster quickly discovers that the street is an even more brutal environment than a home with brutal parents. If one is 14, 15, or 16 or even older—17, 18, or 19—and has no place to live, and if one is cold, hungry, and scared and has no job skills and cannot find work and has nothing to sell except oneself, the most common choice is that of selling oneself and one's body. Without hyperbole or exaggeration, by the hundreds of thousands these homeless street youngsters turn to prostitution in order to survive. Very conservatively, the universe includes two to three hundred thousand American youngsters who habitually live on the streets and engage in prostitution as a life style. It is safer and easier than being at home.

When these youngsters first become so involved, they really do not understand what is about to happen to them and they try very hard to hide from themselves what is actually going on in their lives. Teenage girls often say to me, "Bruce, he ain't no pimp. He's my boyfriend and he needs me." And the boys will say to me, "Bruce, I ain't gay; I ain't no hustler. I'm just trying to make a few bucks."

In the beginning these youngsters try very hard to separate out in their minds what they are from what they do with their bodies, but after 100 johns, 500 johns, 1,000 johns—that's less than a year's clientele for most of these youngsters—it becomes increasingly difficult and eventually impossible for them to separate what they are from what they do. They become what they do and they no longer even care. At that point, the question of one's sexual identity becomes a matter of complete indifference. Curiously, while the girls always seem to show it first in their faces, the boys hide it longer. However, the boys die sooner. The girls can survive longer because it is simply more acceptable for the girls to "work the streets" than the boys. Furthermore, once a boy turns 18, 19, or 20, he grows a beard and hair grows on his chest. The johns no longer want him. He becomes what is called "rough trade," and anybody who buys sex from him is "crazy." These youngsters lead very tragic lives.

SOME CHARACTERISTICS OF COVENANT HOUSE CHILDREN

A descriptive definition of a typical youngster at Covenant House is that of a boy or girl 17 or 18 years of age who during nearly every 24-hour period uses a combination of drugs and alcohol and almost certainly engages in commercial sex. A further description of the collective profile of the teenagers who come to Covenant House is the following: The median age is late 17 or 18. Two thirds of the youngsters are boys and one third are girls. The boys test out at about the fourth grade academically; the girls test out at about the fifth grade. Despite the fact that almost always the youngsters have completed the tenth grade, they are functionally illiterate, with absolutely no skills. The chief complaint of the boys seeking help at Covenant House is physical assault. Forty percent of the boys at the main Covenant House center in New York have either been shot or stabbed at some time in their lives. The chief complaint of the girls seeking help at Covenant House centers throughout the United States is sexual assault. Rape is simply an ordinary experience in their lives.

Columbia University conducted a study of Covenant House youngsters. The researchers were quite shocked and disconcerted at the results and findings which were well known to the Covenant House staff: 50% of all the youngsters coming to the New York center admitted to serious physical abuse at home. Twenty-five percent of the girls admitted to sexual abuse, although that was not the purpose of the study. The youngsters that come to Covenant House off the streets from these devastated homes after incredible abuse are so sad and so desolate that more than one third of the girls have already attempted suicide and another one third of the girls seriously consider it. One sixth of all of the boys have already attempted suicide and another 25% seriously consider it. In the aggregate, more than half of the youngsters that go to Covenant House have either tried to kill themselves or think about it seriously as a way out of a very desolate, extraordinarily sad life.

SEXUALLY TRANSMITTED DISEASES

Sexually transmitted diseases are pandemic among Covenant House youngsters, all of whom are sexually active. Their rate of exposure to acquired immunodeficiency syndrome (AIDS) is among the highest in the United States. At Covenant House, the staff began encountering many youngsters with AIDS during the summer of 1987. Prior to that time, the recognized cases were very few and scattered. Suddenly, last summer, youngsters with AIDS-related complex (ARC) and AIDS and others who tested positive for the human immunodeficiency virus (HIV) began flooding into the centers of Covenant House throughout the country. Covenant House is now engaged in a large-scale double-blind testing program. It is anticipated that by the end of 1988 more than 2,000 youngsters will have been tested. Analysis to date indicates disheartening facts. The good news is that the 16-year-old youngsters are almost entirely free of the AIDS virus which suggests that an intense educational effort undertaken early can perhaps be quite successful in helping these clients avoid AIDS. But when one examines the data for the older teenagers, the 17-, 18-, 19-, and 20-year-old clients, the rate of HIV infection increases dramatically. For example, among the 20-year-old boys in the test group at Covenant House, 17% or more are infected with the virus. That is an extraordinarily high figure. This infection is not caused by intravenous drug abuse. It is a life style disease. It occurs almost invariably because of the youngsters' involvement in commercial sex. It represents a very serious potential danger to our broad heterosexual population. It is not certain that these preliminary results will be borne out by more extensive testing but certainly in a year or so more definitive information will be available.

COVENANT HOUSE SERVICES

The services provided by Covenant House in the first instance are of the crisis intervention variety. We operate on a totally and absolutely open-intake basis, which means we have no intake criteria; there are no screens. Any boy or girl, any young man or woman who comes to us and asks for help must receive it. It is a condition of employment for the staff at Covenant House that they never turn a youngster away.

In the first instance we, of course, respond to the emergency needs for food, clothing, shelter, medical help, protection from pimps. We try to provide a quick opportunity to return home if that is possible and if that is a good idea for the individual youngster. For the long term, when we can, we provide selected youngsters who are not too severely damaged with job training opportunities in residences where the youngsters can stay for a year, two years, even longer. They complete their education by going back to school and then obtain jobs and rejoin society as happy, active citizens. We have, thank God, a very high success rate in those long term programs.

The youngsters that come to us have a very simple thing in common. They are good and they are brave and they desperately want to make it off the street. What they also have in common is that very few succeed in getting off the street unless they receive the kind of help they need in time. For many thousands of youngsters it is already too late and no intervention of any kind has any success.

We have outreach programs in all our centers in New York, Toronto, New Orleans, Fort Lauderdale, and Anchorage where staff and vans go out every night to find kids on the street. We cruise the high risk areas of the city from 10 PM to 6 AM. In New York City we have identified thousands of kids who are on the streets who will not come in to our program although they almost always know about it and respect it. They have heard very good things about the Covenant House program but they are just not ready. That is the reason they give. The real reason is they are so hopelessly, totally enmeshed and captured by street life that they cannot escape it. Most of these youngsters really do not survive that experience.

It is not easy to reach these youngsters. Many of them have been so damaged and so abused at home and then so incredibly traumatized by their experience on the streets that the damage is so profound it has become almost irreversible. In our experience at Covenant House we must reach these kids if we wish to have any realistic hope of helping them, within a month or two or at most three months. Three months is a long time on the streets. Six months is forever. After a year, for all practical purposes, these kids will not be able to make it back to normal living. We are able to help only about one third of the youngsters that come to Covenant House for help.

Our knowledge and experience indicate that the others die quite young. We believe that most of our youngsters die at an early age—by the time they are 30. Some die from alcoholism. Many of our youngsters are murdered and even more commit suicide. If it is true that one out of every 15 people who attempt suicide actually at some time in their lives succeed in doing so, then literally thousands of Covenant House youngsters eventually kill themselves.

There are no mysteries about why Covenant House teenagers are on the streets. There are no mysteries about what happens to them when they go there. They become part of the American sex industry inevitably, inexorably, but of necessity. They have become the merchandise, the commodities that are bought and sold. They are among the most tragic of the youngsters in our society. When one sees them on the street, one very often sees them with clean clothes and haircuts and they appear to be having a good time, seemingly enjoying themselves. But the reality is that I have never met a young prostitute—boy or girl—among the thousands who have come to Covenant House who wanted to be a prostitute. The reality is that if a girl walks up to a man on the street and says, "Hey, mister, are you looking for a good time? Do you want to party?" she cannot be crying or the man will not "buy her." She cannot enter the john's fantasy that he is a good person doing a good thing. If a boy walks up to someone and says, "Mister, are you looking for some friendly company? Are you looking for some action?" he cannot have tears in his eyes or the man will not "buy him." He cannot enter into the man's fantasy that he is a good person. And so these youngsters must look happy; they must appear to enjoy themselves; they must desperately simulate that they like their clients or they go hungry.

Conservatively speaking, there are about 200,000 such youngsters—a subpopulation of the United States that is not even recognized to exist. They are not even counted among the population of the homeless. By some incredibly strange quirk of United States counting procedures, the homeless are not counted until they become 21 years of age. More and more people are at least becoming aware that the problem exists and on both the federal and state levels some important legislation has been passed. The Child Protective Act of 1984 and New York State's Runaway Youth Act passed almost entirely because of Covenant House efforts. What these youngsters have in common is how good they are, and how brave they are, and how much they desperately want to make it back into society off the street. Very few of them actually succeed.

Abuse of the elderly: Misuses of power

ERIC J. CASSELL, MD

At first thought it would seem that no particular ethical issues are raised by the abuse of the elderly. Abuse of anyone is wrong, and abuse of the elderly seems particularly offensive. It is this intuitive feeling of outrage about intentionally injuring, misusing, or wronging old people that provides a clue as to what we really mean by the word "abuse." In the ensuing discussion, I express the belief that sometimes when physicians employ their power in a manner they believe to be in the best interests of sick, elderly persons, the physicians are abusing their patients.

When one is old enough, kitchens, bathrooms, and even bedrooms are traps for failed memories and unsteady bodies. Yet we do not consider old persons to have been abused by a bathtub or a shower if they are injured in them. Even the old person who is attacked on the street and beaten or mugged has not been abused in the sense in which we usually employ the term. We reserve the term abuse primarily for those situations in which a person, object, or relationship is wrongly used. This derives from the idea that to use something is to have a relationship with it. To abuse someone means, therefore, to violate the terms of the relationship, to take advantage of the relationship, to use it to the advantage of the user and the disadvantage of the used. The elderly are abused when others in relationship to them use them to their disadvantage.

Relationships of all types, whether with persons or medical instruments, restrict our freedom. To be in a relationship with another person is in some sense to be responsible to the person. To use an instrument is to be responsible for the instrument, although to a different degree than is understood in human relationships. In this era we have come to believe that we have enormous freedom of choice and few restrictions on that freedom. But we have mistaken our enormous political freedom as citizens—which has given us rights and, therefore, power in relationship to the state, its agents, and other citizens that would have been unheard of even a generation ago—with freedom from responsibility for persons, relationships, and objects. It is as though the possession of our enormous individual power to make things happen, multiply the force of our muscles, span distance and time, reduce inequities of personal endowment, increase the computational power of our brains, have access to knowledge and information, and defeat disease and death, entitles us to use this power without restraint.

It is human nature for the individual and the group to be more concerned with the exercise and extension of political and technologic power than they are with the restraint of such power. When persons acquire power, they not only want to employ it, but are jealous of it and guard against intrusions or limitations. In the arena of individual freedom this translates, for example, into the use of law and lawsuits as instruments of personal aggression, and rejection of governmental authority no matter how benignly (as in the United States) it had been previously regarded. In the same manner, a population composed of persons intent each on his or her own individual power might be expected to reject authority in general. It should be no surprise that many of the abuses of the elderly stem from our concentration on our powers with neglect of the restraints imposed on us by the web of our relationships.

An examination of some of the kinds of abuse currently suffered by the elderly will make it possible to see how these generalizations about individual power in the modern era apply to the problem.

ABUSE OF THE ELDERLY BY THEIR CHILDREN

Reports of the physical abuse of old persons by their children or other relatives are more common than one might have thought possible. The elderly are sometimes found to have been beaten regularly, tied to beds, or confined to small spaces. They do not complain to authorities because they are frightened of further physical punishment and because they do not see alternatives to their situation. As with battered children, the awfulness comes not only from the infliction of corporal punishment, but from the lack of justification for any punishment for the offenses described. Battering parents frequently cite the child's crying as the reason for the battering. In the case of the elderly, their complaints about ill treatment are given as the reason for the abuse.

How can a person beat up an old man or woman at all, much less a mother or father? While parents may be perceived as wicked and unloving to their children, with bad memories and life-long resentments a natural consequence, most would not find this to be a justification for retaliatory abuse a generation later. It was wrong when done to the child, and it is wrong when done to the parent.

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There are many reasons acceptable to society for a child to refuse responsibility for an aged parent. This is implied in the current welfare laws that sharply limit a child's responsibility for an indigent parent. What makes the abuse particularly noxious is that the child has already accepted responsibility for the dependent parent.

Notice the distinction our society makes between the elderly person who is beaten up by a street thug and the elderly person beaten up by their offspring. The former violence is not cited as an instance of abuse, but the latter is. In the one, there is sympathy about the bodily injuries, the implied lack of safety for the frail elderly, and the sense of injustice and outrage that a young thug should pick on a defenseless old person. In the latter case, there is sympathy for the pain and bodily injury, but also empathy for the anguish we believe is in the old person's heart, sadness that an old person should be so helpless, and outrage that a child could do such a thing to a parent in an apparent betrayal of their relationship.

If there are instances of children battering their elderly parents, then lesser forms of physical and deprivational abuse must also exist and are in all probability more common. Here the child takes some responsibility for the parent's bodily needs, but abuses his or her greater power over a now powerless parent. That it is frank abuse and not merely mean-spirited, small or nasty behavior comes from the relationship of child to parent. As one does not expect parents to abuse their power over their young and helpless children because of their relationship, we do not expect mature children to abuse their power over elderly parents because of this relationship.

One may make convincing arguments for the utility of the social rules that govern relationships, but they are teleological. The social rules of relationships cannot be proven or derived scientifically, because they arise in the moral dimension of human behavior based on ideas of right and good, and science does not recognize the axes of right and wrong or good and bad. Such matters are indifferent to nature, but they are vitally important to humankind. The social rules of relationships are followed because they exist, and because their very existence testifies to common acceptance. While these rules are not derived from argument or science, they can provide the basis for further discussion. The rules of relationships are largely concerned with the relative exercise of the powers of the participants and their responsibilities. It is part of the political history of Western civilization that progress has been marked by the protection of the weak from the strong. The great equalizer is the rule of law in a country dedicated to individual freedom. But law and regulation have distinct limitations as means to control the abuse of powers within a relationship. They cannot supplant the need for a sense of obligation and responsibility instilled in the participants in any relationship. It is impossible to legislate or write regulations to cover every instance of the interaction of one person or group with another person or group. Laws and regulations change slowly and, in the very nature of their genesis, lag behind changes in social rules and customs. There is, in fact, some reason to believe that when law and regulation are over-abundant, the individual sense of responsibility suffers and perhaps even atrophies.

Abuse of the Elderly in Nursing Homes

The elderly are abused not only by their children, but also by representatives of society in positions of power. One has only to visit a welfare office or Medicare office to see how the power of a bureaucrat can confuse, stymie, or even injure the powerless elderly. The elderly, like many citizens, may expect little else from the bureaucracy and its civil servants; it is a common belief that the functionary is in fact primarily responsible to his governmental superior, and not to the beneficiary. The nursing home, however, is believed to be different in concept, if not (sadly) in outcome. Here again, apart from the better institutions, abuse of the elderly is common. Inadequate attention, physical inconvenience, disregard of the humiliation imposed by incontinence and incapacity, and other insensitivities, including physical abuses, are well known. Equally common is excessive emphasis on compliance with regulations concerning medication, accident reporting, bedrails and the like, while human needs are neglected and patients "warehoused."

That poor care and abuse are not inevitable in a nursing home is easily seen in those run by religious groups. Here the treatment of elderly patients is marked by responsiveness to individual need with compassion and kindness. Religious nursing homes excel in providing this because they are dedicated to the service of God through the care of the sick elderly. The inconveniences posed by the incapacities and infirmities of these patients provide an increased opportunity for each caregiver to demonstrate service to God. Obligations to God are exemplified by obligations to sick persons.

Much of the actual care of the elderly in nursing homes is rendered by individuals who are of lesser power in society. The abuse of sick old persons in nursing homes comes about through the actions of the relatively powerless against the almost completely powerless. In these arenas, expressions of power can be mean-spirited, unless they are held in check or given purpose by overriding obligation. Obligation demanded by regulations and laws is a poor substitute for personal obligation.

Obligation and responsibility are reciprocal. The personal responsibility that marks the best nursing home care must be directed primarily toward the old person. Alternatively, the nursing home caregiver may demonstrate responsibility for old persons' bodies apart from the old persons themselves. Most of us believe, however, that our bodies are extensions of ourselves, and that they cannot be taken care of or treated in disregard of ourselves without offending us. That is to say that, "to do something to my body without my permission is to wrong me." It is on that basis that consent to treatment is required from patients. The doctrine of informed consent implies that, "I am the only one who should have the final say about what can be done to my body in my best behalf," and acknowledges that there are personal differences in this regard. The concept that what is done to or for a person's body is for the person to decide, implies that the body belongs to the person rather than to the state or to God. It is possible that only in this century, in the United States, persons are given almost absolute right over their bodies (except while in the armed services). The change in attitude can be seen in the recent modifications in law in which attempting suicide is no longer a crime. (Members of some religious groups believe that the body is God's dominion, and this view sharply limits a person's right to withhold consent for its treatment.) Thus, in the nursing home setting, poor care provided to the body abuses the person. Humiliations that come about because of the treatment of the body are humiliations of the person. These abuses are least present in settings such as religious nursing homes, in which caregivers feel responsible and have obligations to the patients which limit the exercise of power over the powerless elderly.

ABUSE OF THE ELDERLY IN HOSPITALS

It follows from what has already been discussed, that the elderly are also abused in hospitals; but here the issues are more complex. The abuses may arise during the course of medical treatment which is, in itself, apparently proper. The problem arises not with old persons who have the capacity to give consent for treatment, but with those who lack capacity. Many hospital beds all over the United States are occupied by irreversibly incompetent old people in whom cognition is progressively impaired and who would surely die but for effective medical treatment of infections and organ failures. Because the treatment that sustains their lives is frequently given despite their previously expressed wishes to the contrary, or contrary to the wishes of their surrogates, they are being abused. Here the abuse arises not from the intentional exercise of power over the powerless, but from the failure to allow physicians' responsibilities and obligations to old persons to provide checks on the technologic power employed in their treatment.

To avoid misunderstanding, let me clearly define the patient population of which I write. These old persons have several characteristics apart from the presence of their current acute illness. Their incompetence results from chronic, irreversible mental incapacity. Their incompetence is not the result of acute illness; it will not disappear when the present illness is over, and there is no effective treatment for the incompetence. Incompetence has usually come on gradually and has been present for many months to years. The usual diagnoses of the mental incapacity are Alzheimer's disease, multi-infarct dementia, strokes, or other organic dementias. All of these patients are known not to have other diseases the treatment of which might restore their competence. I am restricting my comments to patients whose usual state is such that they cannot hold a conversation, respond appropriately to questions or gestures, and exhibit no evidence of comprehension. There may or may not be evidence of consciousness, but these patients usually do respond to painful stimuli.

It might be argued that I have misrepresented the case. One might contend that incompetent elderly are not being abused; rather, they are being properly treated because they are being kept alive. However, is keeping them alive proper treatment or abuse? Since the issue exists solely because of our current technologic power, we must ask ourselves whether it is an abuse of this technologic power to use it for this purpose. I have pointed out that power is best held in check by obligations and responsibilities, and that these arise primarily in relationships—that, in fact, it is obligations and responsibilities that maintain the balance of power within personal relationships. Physicians maintain relationships with the public, the law, other physicians, the hospitals in which they practice, the profession itself, and patients. While all of these relationships affect the exercise of physicians' power, they derive from the principles that guide the relationship of an individual doctor with an individual patient.

In the care of the incompetent elderly, no actual relationship with the patient is possible, and the doctor's actions are guided by idealized notions of the doctor-patient relationship and the associated responsibilities and obligations of the physician. Nowhere is there warrant for the belief that obligations or responsibilities to patients are discharged by the care solely of the diseased body, particularly when the treatment is in disregard of the patient's wishes. Care of the diseased body is not necessarily care of the patient. When treating patients such as these, physicians state that they are doing it for the benefit of the patient; that if they do not support failing organs or treat infection, the patient will surely die. It is worthwhile, therefore, to question why any episode of disease is treated.

LEGAL CONSIDERATIONS

Many physicians say that they are legally obligated to treat incompetent elderly patients, and that they may be sued for malpractice or prosecuted if they do not provide treatment. Unless the patient has left directives requesting such treatment, or the patient's legal surrogate requests treatment, physicians are under no such obligation. If patients' moral surrogates ask that they be treated, despite their lack of standing as legal surrogates in New York State, physicians are probably correct in continuing treatment. Too often, however, the family is put in the position of being asked, "Is it all right if we let your mother die?" after which it takes a brave family to insist that senseless treatment be discontinued. More often, the recourse to legalisms is merely an excuse not to exercise judgment in the treatment of a patient who requires the physician's best judgment of when it is truly wise to treat or not treat. Treating the patient because the hospital counsel insists that the hospital be protected, or because physicians want to protect themselves, regardless of the good of the patient, is in itself a definition of patient abuse.

Why is any episode of disease treated? Treatment traditionally occurs because a person asks to be treated, a physician concurs that a reason for treatment exists, a treatment actually exists, more good than harm is anticipated, and the physician is not being asked to do harm, damage normal tissue, or become involved in a probable cause of death (as in high-risk surgery). Doctors also act when patients are not able to request treatment, that is, in emergencies or with an unconscious or acutely incompetent patient. Here the essence of the situation is its immediacy; it is an event, a complete circumstance of its own. In these situations, doctors treat because a medically recognizable problem exists which would lead to permanent disability or death.

If no urgency exists, physicians usually wait until the patient can ask for treatment. Further, treatment is considered to be possible when it will not do harm, and it is directed towards at least a meaningful possibility that a viable person will emerge at the other end of the acute situation (for example, in trauma, no one would transfuse a patient whose skull had been crushed simply because anemia is present).

Traditionally, in emergencies and otherwise, three conditions are met. The first of these is that a relationship exists between the patient and doctor, in fact or in custom. In nonemergent care, the patient has become a patient by choice and request, responding in some manner over time, with the opportunity to end the relationship. (In emergencies, the implicit expectation is that the patient would, if able, desire the intervention and become a patient, and the doctor will feel responsible for and to the person no matter what the state of consciousness. The emergency is expected to be over at some time, the recovering patient then being able to respond to what was done on his or her behalf.) The second condition is that in treatment the good of the patient is intended. The good of the patient is not automatically the same as the good of an organ or body part. (Being equated with their body parts is universally disliked by patients.) The third condition is that some concept of the future is inherent in the treatment. It is possible to see the treatment of an episode of disease as an isolated event, but to do so depersonalizes the event as surely as treating merely the body depersonalizes the patient. When the goal is the good of the patient, it is always implied that no matter how bad the future may seem, it exists, and will be better with than without the intervention.

The definitive treatment of acute episodes of illness in the hopelessly sick, hopelessly incompetent elderly patient meets none of these tests. The patient does not request the treatment. The presumption that the patient would request the treatment if able is extremely doubtful, because a significant number of persons would not wish to live given these circumstances. Treatment is possible but does nothing for the underlying problem. It is difficult to know if the treatment is doing good or harm. No restored individual will emerge at the other end of the acute situation. No relationship is involved at the time of treatment or in the future. The doctor may feel some sense of responsibility but will be in conflict as to whether his or her treatment discharges the obligations. The good of the person is not necessarily intended. And the actions are largely in disregard of the future.

Medical Therapy as a Form of Abuse

To abuse someone is to violate the terms of your relationship with them and to take advantage of the relationship. The elderly are abused when others in relationship to them use them to their disadvantage. The incompetent elderly are abused by treatments meant to keep them alive in the face of the inexorable progression of their underlying disease. It is difficult to apply the term "abuse" in these circumstances because, with rare exceptions, the intent of physicians treating such patients is good. Why, then, does the abuse come about?

There are three reasons. First, it is not primarily the sick person that is being treated, but rather events that happen to the patient. For example, the events of sepsis, hypotension, renal failure, and carotid sinus dysfunction are perceived as separate occurrences, rather than as an episode in the ongoing history of a sick person. Because of this, there is no sense of a life that came before sickness and into which the sickness, the death, and the events preceding death must be fitted. There is also no sense of the future to which the patient will be returned. Some therapies are fitting; however, others are not, because these treatments do not fit the story of the person's life and terminal illness. Medical therapy that is oriented towards events rather than patients is, in fact, an abuse of the person. Pernicious effects stem not from fate but from those who take care of the person when he or she is most defenseless.

The second reason is that the treatment reflects what the technology of medicine allows physicians to do, rather than what the patient requires. In this regard, the technology should not be seen as merely an inanimate tool, but rather as an extension of the personal power of physicians. We do not utilize technology because of what it will do, but because of what we can do because of it. We do not support failing renal function when it serves no long term purpose because a dialysis device can do so, but because the device extends our power to support the failing kidney. It is the inappropriate use of power that must be resisted, not technology itself.

The third reason that elderly patients are often abused in hospitals by physicians who have good intentions is that if the proper restraint of power were exercised, physicians would have to express their obligations and responsibilities to the sick patient instead of merely to the patient's body. But what is good for the body is generally a straightforward matter, while the good of the patient may be uncertain, difficult to ascertain, and ambiguous. There is little wonder that decisions are made on the basis of the physiologic parameters that flow from labsheets and monitors rather than the personal determinants. Doing the job right requires careful history taking, intensive and difficult questioning, and painstaking mediation between family members with differing opinions.

Conclusions

Abuse of the elderly occurs primarily because of the exercise of power by others unchecked by obligation or responsibility. Law and regulation are poor sources of restraint of the powers of others over the helpless elderly. In general, it is the web of mutual obligations and responsibilities that regulate the power of the participants in social relationships. In medicine, it is the obligations and responsibilities of physicians in relationships with their patients that have regulated their actions over centuries. When no actual relationship is possible, as with unconscious or completely incompetent elderly patients, obligations and responsibilities flow from the traditional and ideal conceptions of the relationships developed over the centuries and still current in medicine. When these are forgotten or their uncertainties and ambiguities forgone for the greater certainty and simplicity of purely technical medicine, then the power of physicians over individual patients is left unchecked. It seems to be in the nature of the human condition that in the absence of checks and restraints, power is almost invariably abused. Based on the treatment of the incompetent elderly, this seems true of medicine also, despite the profession's underlying benevolent purpose. Obligations and responsibilities to ill persons remain the primary defense against the abuse of the sick.

Confronting child abuse and neglect in New York State

BARRY R. SHERMAN, PHD

Child abuse and neglect, or maltreatment, include a wide spectrum of commissions and omissions by parents and guardians. They encompass emotional, verbal, physical, and sexual abuse. In general, most cases of child abuse and neglect are the result of inadequate or inappropriate parenting.

The social and economic consequences of child abuse and neglect have been well researched and documented. Numerous studies have linked child abuse to juvenile delinquency and adult criminal behavior.[1] Abused and neglected children show impaired physical, psychological, and neurological functioning, as well as a range of behavioral disturbances, including poor impulse control, noncompliance, and aggressive and violent reactions.[2,3] These victimized children also manifest a variety of educational deficits resulting in poor academic achievement and maladaptive behaviors in adolescence.[4]

Based on reported statistics, abused and neglected children, although represented across socioeconomic groups, most often come from families of low socioeconomic status characterized by frequent changes in residence and parental figures.[5] The abusive parents, frequently abused and neglected themselves, are often socially isolated from family and friends as well as other social supports in the community. Numerous research studies strongly suggest that a history of being abused is one of the major factors predictive of an individual's risk of abusing or neglecting his/her own children.[6-8]

Research has shown that 80-90% of all incarcerated felons were victims of child maltreatment.[9] Society incurs a significant financial cost to retain juvenile delinquents in public detention facilities and criminals in state penal facilities. By late adolescence, a disproportionate number of neglected and abused children are sufficiently physically disabled or mentally ill to make them dependent on the public system of care. The long term impact of child maltreatment will overwhelm the system of schools, courts, social services programs, and mental health facilities in the coming years unless preventive efforts are undertaken.[10]

INCIDENCE OF CHILD ABUSE AND NEGLECT

The actual incidence of child maltreatment is unknown. Reported incidents of child abuse and neglect continue to rise at an alarming rate to the point at which child maltreatment is now recognized as a major public health problem. In the United States alone, more than two million children are seriously abused or neglected, and about 5,000 die from related causes every year. Between 1% and 2% of all children in the United States are reported as subjects of abuse and neglect, an annual rate of 10-15 per 1,000 population.[11] During 1986 in New York State, for every 1,000 children under the age of 18 years, there were 17 reports of abuse or neglect.[12]

According to a survey by the National Committee for the Prevention of Child Abuse, 45 of 50 states witnessed an increase in child abuse in 1983, and 38 states indicated that the severity of cases had also increased.[13] A 1986 survey, to which 34 states responded, showed an increase of 23% in childhood fatality from 1985 to 1986.[14] The impact of these figures is compounded over time, because abuse and neglect negatively affect a child's growth and development and cannot heal in the same manner as do physical injuries.

Most experts agree that the greater number of reports is evidence of a heightened public awareness of the problem and a function of increased utilization of the reporting system. While the percentage of substantiated cases has dropped from 47.5% in 1976 to 40% in 1986, the actual number of reported cases has risen 44% during that same period.[14]

Some additional illustrative facts are as follows:

- Since 1980, there has been a 51% increase in child abuse and neglect reports with a concomitant increase of only 2% in funding for child abuse and neglect prevention programs.[15]
- Seventy-five percent of child fatalities due to child abuse and neglect occur before the child is 2 years of age.[16]
- One third to one half of the fatality cases are already known to the mental health and criminal justice systems, as well as to child protective services, which had investigated but not substantiated a previous allegation of abuse or neglect.
- Approximately 70% of families are known to be abusive by their informal social networks including relatives, neighbors, and friends.[17]

That's the bad news. The good news is that there has been significant progress in coordinating a response to the problem in New York State.

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PREVENTION ACTIVITIES IN NEW YORK STATE

The Children and Family Trust Fund Program, as established through Chapter 960 of the Laws of 1984 and amended by Chapter 57 of the Laws of 1985, made available $3 million as a new source of funding in New York State for prevention of all aspects of family violence including child abuse, spouse abuse, and elder abuse. Funds are distributed annually by the New York State Department of Social Services through a request for proposals to local programs. The majority of funded programs were in full operation by 1987 and include parent education, community awareness and education, counseling, advocacy, respite, and a Hispanic domestic violence film project.

Also in 1984, under a grant from the United States Department of Health and Human Services, National Center on Child Abuse and Neglect, the State of New York Department of Health and Department of Social Services jointly promulgated two sets of official guidelines, one concerning the identification and management of suspected child abuse and neglect in hospitals and clinics and the other regarding the identification and management of sexual abuse and maltreatment in hospitals and clinics. These two documents have been lauded by the medical community as extremely useful in expediting timely and appropriate reports to Child Protective Services.

Given the complexity of the system of services to children and the barriers to effective interagency communication and collaboration, Governor Cuomo, as part of his declaration of the "Decade of the Child" in his 1988 State of the State Message to the Legislature, established a Task Force on Children and Youth to address systematically the needs of children in New York State.

The Citizens' Task Force on Child Abuse and Neglect, co-chaired by First Lady Matilda Cuomo and the Commissioner of Social Services, includes representatives from both the public and private sectors with a variety of expertise and backgrounds. The mission is two-fold: 1) to educate the public about the problem of child abuse and neglect: and 2) to promote the development of parenting education programs.

Since the 1982 joint resolution of Congress, every April has been nationally proclaimed as Child Abuse Prevention Month. "New York Loves Parents" was the theme of the Citizens' Task Force campaign in 1988. The campaign launched the new Pre- and Post-Natal Parent Education Hospital Based Parenting Programs (PPPEHP). The "Welcome to Parenthood" booklet was published; it offers hints for coping with the birth of a child and the stresses of childrearing and lists community-based programs. The booklet is being distributed to all new parents by hospitals throughout New York State.

New York made significant progress in the area of child abuse and neglect prevention by enacting new laws in 1988 that permit the courts to remove an allegedly abusive or neglectful parent from the home instead of removing a child (Chapter 673; Assembly Bill No. 10857A) and establish new training programs for mandated reporters who are new or current employees of mental hygiene facilities, hospitals, school districts, and the police (Chapter 544; Assembly Bill No. 10835A).

The Medical Society of the State of New York's Committee on Child Abuse has developed a protocol for physicians' management of child maltreatment. Additionally, the committee is engaged in a project to produce a video cassette and manual for the education of physicians and other professionals who are involved in providing court testimony in trials where physically, and especially sexually, abused children are being questioned regarding the circumstances of their abusive situations.

HOME-VISITOR PROGRAMS: A PROMISING PUBLIC HEALTH MODEL OF EFFECTIVE PREVENTION

Historically, the approach of the majority of child abuse and neglect intervention programs has been treatment of individuals and families (perpetrators and victims) who are identified after an incident of maltreatment has already occurred.[18] There has been a paucity of research regarding the efficacy of prenatal preventive interventions. An exception has been the research of Olds and colleagues[19] which empirically demonstrated the effectiveness of home visitation by public health nurses for poor teenage parents in Elmira, NY, from the prenatal through the postnatal period. In a two-year follow-up, the home visited group evidenced significantly fewer subsequent reported incidents of child abuse and neglect (4% as compared to 19% in the controls; $p < 0.05$), and the babies were seen less frequently in the emergency room. The study also verified the assertion that the encouragement of the early formation of secure attachments between mother and infant facilitates mothers' appropriate interpretation of babies' signals, ultimately protecting the child from abuse and neglect.[19]

A study, currently being conducted by this author in collaboration with the New York State Division of Social Work and the Rochester Adolescent Maternity Program (RAMP) at the University of Rochester Medical Center, is empirically testing the home-visitation model utilizing the services of indigenous paraprofessionals, instead of nurses, who function as case managers for at-risk, inner-city primiparous adolescents. Services are provided primarily by closely supervised paraprofessionals in the clients' homes on an average of once a week. The goal is more frequent and longer visits than in the public health nurse model, which enable the development of strong relationships between providers and pregnant teens. Services are targeted to women under the age of 18 and are initiated prior to 28 weeks' gestation. Specific activities include preparation for childbirth, prenatal mother-infant bonding, household management, family planning, parent-child stimulation, and support groups.

Young teenagers are randomly assigned to the experimental and control groups during their second trimester of pregnancy. An experimental design is being employed to assess empirically the effectiveness of the paraprofessional home-visitor treatment intervention. The control group receives all aspects of comprehensive prenatal and pediatric care except for the home visits. A family's involvement in the program extends until the child's first birthday.

At the six-month follow-up, 23 of 24 (95.8%) of the home-visited teens with babies were still receiving health and social services and were identifiable by project staff. Only 18 of 31 (58%) of the control group, receiving no home visitation, could be located by either the health or social services delivery system. Other expected outcomes of the project include reduction in the incidence of child abuse and neglect, less demand for foster care placements, improved infant development, and an enhanced quality of life for participant families.

RECOMMENDATIONS

The following policy recommendations are proposed in full knowledge of the many bureaucratic barriers that make implementation a very difficult challenge:

- The community response system needs to be redefined to include more than Department of Social Services Child Protective Services. In order to assume a greater public health perspective, training of frontline responders in the community must be undertaken, including police, paramedical, medical and, particularly, school personnel. Child abuse is not just a social services problem, it is everybody's problem.
- Bureaucratic boundaries that categorize and constrain the way that child abuse is addressed must be eliminated. If the recently publicized death of Lisa Steinberg, resulting from physical abuse by her adoptive parents, taught us nothing else, it pointed out the desperate need for removing barriers to interagency communication and coordination.
- Risk assessment cannot be an instant snapshot of a family in crisis but rather, valid risk assessment should be based on a personal, ongoing supportive relationship with families.
- Since available statistics can predict geographic areas where child abuse and neglect occur most frequently, comprehensive community-based home health visiting programs hold tremendous promise for implementing support systems to families in those targeted high-risk areas.

In summary, child abuse and neglect are only part of a more general public health problem of increasing family violence involving more intentional injuries such as homicide and suicide. Significant resources have been expended on after-the-fact adjudication and incarceration of perpetrators as well as medical and psychological rehabilitation of victims. Instead, what is truly needed is a collaborative, interagency strategy to prevent child abuse and neglect before it occurs. Children are our future, and it is the responsibility of all of us to protect their lives from harm.

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Anorexia nervosa: The destructive pursuit of thinness

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In the last few years, we have witnessed a marked proliferation of the literature on eating disorders. As a result, physicians are now much more knowledgeable and also more successful in the treatment of patients with these problems. Anorexia nervosa primarily manifests itself in the adolescent female but it can also continue into the middle as well as the older years[1] if treatment is not undertaken. In severe cases it may be fatal. It can also be encountered in the young male but to a very much lesser degree. Although the expressed goal is for slenderness, the pathology soon becomes evident as the patient progresses to a state of emaciation. It must be emphasized that this depriving oneself of food is not due to any loss of appetite but is consciously adhered to solely for the purpose of being thin. What is being expressed here is the underlying fear of being fat.[2]

This fear of being fat which is experienced by the anorexic patient should not be confused with the discomfort or unhappiness of an overweight young woman. Here it is directly related to deep seated fears as she is physically maturing and beginning to experience stronger sexual feelings. The growth of her breasts, a rounding of her body contour, and the onset of her menstrual periods confront her with an awareness of her sexual maturity. The patient who resolves this conflict by developing anorexia will frequently also reveal a family history of eating problems and fears of being overweight.

HISTORICAL BACKGROUND

Self-starvation was already referred to in the Middle Ages but at that time it was attributed to demons and devils who had taken possession of a person's body. In 1689 these symptoms were dramatically and accurately described by Morton[3] in his paper, "Phthisiologia: Or a Treatise of Consumptions," for at that time he considered it to be a variant form of tuberculosis. Two centuries were to elapse, however, before anorexia nervosa was actually identified as a clinical entity. It was the English physician Gull[4] and shortly thereafter the French physician Lasegue[5] who recognized and understood that the causes were not physical but emotional. This was challenged, however, following the publication in 1914 of Simmonds'[6] work on cachexia and the pituitary gland. For several years, anorexia nervosa was mistakenly viewed as an endocrine disturbance until further studies resulted in differentiating it from Simmonds' disease.

ETIOLOGY

Although cultural and societal factors play a part in this desire for thinness, the nuclear problem revolves around the mother-child relationship. Unresolved sadomasochistic conflicts in the oral phase of development set the stage for further difficulties in the successive phases of development. The early history of these patients usually presents a surface picture of a good and obedient child who is doing well in school until approximately the time of puberty, when these problems suddenly begin to appear. However, when these patients undertake analytic treatment, a somewhat different picture emerges, revealing a host of problems that the child had been experiencing. These angry, confusing, and unhappy feelings are exhibited symbolically by means of self-starvation.

PSYCHODYNAMICS

The patient usually is totally unaware of her emaciated state and fully convinced and satisfied that this is her ideal weight. She has no interest in or wish to change this body image. Only the concerned and continued efforts by family and friends may serve to overcome the persistent resistance to seeking medical care. Bruch[7] was aware of this basic problem when she wrote that a realistic body image is a precondition for seeking help and recovery in anorexia nervosa. However, a great deal of psychoanalytic work will have to be done before there is an understanding of what this emaciated body image represents.

The primary symptoms exhibited by these patients have now become quite familiar: severe weight loss caused by a starvation diet, amenorrhea, and a program of strenuous athletic activities constitute the classical triad. Although these symptoms are in the foreground, the physician soon becomes acquainted with other problems the patient is experiencing, such as feelings of omnipotence, demands for immediate gratification, obsessions, and the ever present underlying need for control. The unsuccessful struggle for control which began in the early relationships with her mother is now continued more successfully as the patient restricts her food intake. Here she is able to achieve this control. Sperling,[8] one of the early and respected investigators in this area, points out that this also represents a more overt expression of even greater rebelliousness and

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hostility toward the mother. However, since this is an illness, the patient feels safe and can exhibit this aggressive behavior without fear.

With the onset of menstruation, the unresolved oedipal conflicts serve to intensify the patient's fears of sexuality. As a consequence there is a regression of the sexual impulses and their displacement from the genital to the oral level. Although the forbidden sexual feelings are now disguised, the basic problems remain unchanged. Unless these unconscious fears are resolved, the patient will remain incapable of assuming the role of an adult female or of accepting future motherhood. Simply increasing the patient's weight by subjecting her to a high caloric diet unfortunately will not provide the solution. Before the patient begins analytic treatment, methods and goals must be carefully explained, while also emphasizing that the gaining of weight is an objective but not the primary one.

Psychologically, the cessation of menstruation provides two important goals for the patient. First, the regression to an earlier phase of development frees her of these threatening sexual feelings and second, not menstruating also serves to fulfill her earlier unconscious fantasies of being pregnant. These earlier findings by Deutsch,[9] Lorand,[10] and Sperling[8] continue to be confirmed in treatment with patients today. When these unconscious fears are understood and resolved there is again the resumption of a regular menstrual cycle and the amenorrhea ceases to be a problem. It should also be pointed out that, contrary to earlier beliefs, amenorrhea may appear as an initial symptom rather than only later when the patient is severely emaciated.[11]

Possibly even more troubling for the patient with anorexia is the constipation and the added difficulties she may create in her endeavors to deal with this problem. The abuse of laxatives, diuretics, and enemas can easily alter the electrolyte balance and result in a life threatening situation. It demands a constant and careful attention to the physical aspects but what should not be forgotten is that the basic problem, the constipation, is still psychologically determined. It has its roots in the early confusion and fears surrounding eating and excretion and also provides the matrix for the development of fantasies of oral impregnation, pregnancy, and anal birth. Only by understanding the dynamics of her misinterpretations and fears will the patient be able to relinquish these symptoms of defense.

THERAPY

Diagnostically, anorexia should be considered as a neurosis, although at times episodes of severe disorganization can occur which may appear to resemble a frank psychosis. However, these are usually of short duration and can be dealt with without medication unless life threatening. Although analytic therapy is preferred by some, other modalities of treatment are also utilized at the present time. Foremost among these are psychotherapy—nonanalytic, the cognitive and behavioral therapies, family systems therapy, and hospital or residential treatment. Although the patient may respond to any of these forms of treatment and appear symptom free, unless the deep seated underlying personality problems are resolved, further difficulties can be anticipated. Specifically, anorexia nervosa should be regarded as an unsuccessful attempt at solving unconscious sexual conflicts concerning sexual identity. If one ignores this area, treatment will remain incomplete.

Our ability to treat anorexia nervosa more successfully now confronts us with another challenge, and that is learning how to prevent it. Since the pathology in the mother-daughter relationship plays a major role in this problem, it may be rewarding to utilize this as a public health program of education. This mental health program should be directed at three target groups: prepubertal and adolescent females and young married couples. It will provide a new understanding for many and may prove to be effective in reducing the present day pathology even more than it is possible to anticipate at this time. By carefully designing such a program, one may also create a blueprint for achieving that elusive healthier emotional life for each and every member within the family structure.

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The physician's management of child maltreatment

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Stanley Schwartzberg, MD; Lucia Boselli; Daphne Gleit; Jule Fontanetta; Lila Wolff;
Barry Sherman, PhD

The child maltreatment syndrome as listed in the International Classification of Diseases is mainly regarded as referring to physical abuse. However, other forms of maltreatment include emotional abuse, sexual abuse, and neglect. New York State law mandates reporting by persons involved with child care of all reasonably apparent cases to the proper authorities. Furthermore, it assures the reporter of immunity from prosecution or other legal action if a case is proven to be unfounded. Professionals should be able to question properly and to recognize with a reasonable degree of certainty inflicted trauma as distinct from accidental injuries, as well as evidence of sexual molestation.

Most victims of abuse are brought to a hospital emergency room by either a social worker, a law officer, or the abusive parent; or are referred there by a clinic or primary care physician who would like to have a second opinion. The management protocol should be as follows:

All alleged physically or sexually abused or severely neglected minors under 18 years of age should be admitted to the in-patient pediatric or adolescent unit without delay. (An exception should be made when the accompanying social worker from an agency such as Special Services for Children officially requests only an out-patient physical examination of the child, and a documented opinion prior to the child's appearance in court and placement in a shelter or foster home.)

An immediate telephone report by a responsible hospital representative should be made to the New York State Child Protective Services Registry's 24-hour hotline (1-800-342-6720), unless this has already been done by the social agency authorities. A hospital pediatric social worker should be assigned to the patient. Within 24 hours, or by Monday if a weekend intervenes, the worker should fill out and submit the official form DSS2221A, for submission to Child Protective Services. This must contain the examining physician's diagnosis and signature.

A house officer or attending physician should thoroughly examine the child with special alertness for traumatic lesions. Photographs of obvious lesions, and x-rays of all possible or actual fractures, including long bone or skull series films, should be taken for the record, if warranted by the history or the suspicion index of the physician. Medical and surgical procedures should be instituted promptly as indicated, and thoroughly documented.

The legal definition of sexual abuse or molestation of a minor under 18 years of age pertains to incest or intrafamilial acts; or to intrahousehold acts, including digital manipulation of genitals, fellatio (oral sex), sodomy, and intercourse. These cases generally fall within the clinical purview of the hospital's department of pediatrics, and suspected victims should be examined, treated, and managed by the house staff under the guidance of the chairman and/or the attending physician. Sexual attack or rape of a female outside of the household by an adult is usually a subject for police action, and the victim should be transferred directly to a certified rape center.

On the pediatric/adolescent unit, the sexually abused minor must be handled calmly and nonjudgmentally by the examiner obtaining the history of the actual events of the molestation. If the girl is pubertal, a careful menstrual record should be obtained and a urinary chorionic gonadotropin (UCG) test for pregnancy should be performed. Special inspection for traumatic and other lesions of the genitals, anus, hymen, vaginal orifice, and throat is important. Smears, cultures, and blood tests for sexually transmitted diseases should be performed. In cases of recent sexual contact, or when the discharge is suggestive, smears should be examined for semen and sperm. When a serious gynecologic condition or pregnancy is apparent, a gynecologic consultation should be requested.

A physically, sexually, or otherwise abused minor, whose case is reported officially and who is admitted to a hospital, should be retained by legal decree, even beyond medical or surgical indications. This should be effective until the court or the official agency decides and designates when and to whom the minor should be discharged. The hospital social worker should maintain contact with the patient, with the official agency involved, and, if indicated, with the minor's family.

Address correspondence to Dr Blumberg, Jamaica Hospital, 89th Ave and Van Wyck Expwy, Jamaica, NY 11418.

* Prepared as an educational and informational report by the Committee on Child Abuse of the Medical Society of the State of New York, Lake Success, NY.

The role of physicians in America's sterilization movement, 1894–1925

JOHN S. HALLER, JR, PHD

> I am firmly convinced that if the many who decry and protest against sterilization, could be brought into direct contact with warped mentalities as are we who live among them, they would come over to our side; just as have those who, in theory opposed to lynching, when their own families are defiled at once become enthusiastic advocates of lynch law.
>
> Martin W. Barr, MD, *Alienist and Neurologist*, 1912

> It seems, to put it none too strongly, that America is simply hell-bent on taking a brief biological joy-ride with the definite policy of later turning over its vast intellectual conquests to the morons.
>
> George Worthington, *Journal of Social Hygiene*, 1925

During the late 19th and early 20th centuries, statistical evidence indicated that the numbers of individuals identified as feeble-minded, defective, and criminal were increasing at a proportionally higher rate than the rest of the population. In addressing this issue, prominent as well as less than prominent physicians contributed to the discussion. Their observations and recommendations carried significant weight in the mind of the ordinary citizen and in the corridors of government. As respected confidantes to the rich and influential, they found their ideas heeded, debated, and accepted as credo for many who were seeking solutions to the troubling evidence before them. Physicians became a moral and political force out of proportion to their numbers but certainly in keeping with both their expectations and their increased status within American society. Their ability to define the parameters of the problem, provide surgical options, and carry out the surgical solution brought them into the forefront of the sterilization movement in America.

ALARM BELLS

In both Europe and America, physicians, scientists, and social scientists announced that defective classes were multiplying more rapidly than the total population. Statistics published at the turn of the century and based on the United States census of 1900 suggested that some 1,250,000 individuals were "insane," "feeble-minded," "deaf and dumb," "blind," confined to prisons and reformatories, or supported in benevolent institutions such as industrial and reform schools and almshouses, all requiring some form of long term care or assistance. According to T. Alex Mitchell, MD, of New York, "a wave of degeneracy is sweeping the land, and its development threatens the physical vitality of the nation."[1,2] In England, T. B. Hyslop, MD, characterized mental degenerates in his country as a large "sperm whale" propagating the East End of London with the castoffs of society.[3(p25)] Similarly, Arnold White predicted that unless England did something practical to reduce the number of "inefficients," the year 1925 would mark the end of England's sovereignty.[3(p25)]

Increasingly, philanthropists and criminologists, state boards of charities, state boards of health, and state legislatures focused their attention on the so-called "unfit." Operating on the assumption that heredity, congenital and acquired disease, and unfavorable environmental influences had spawned vice, crime, and disease, they both raised the issue and simultaneously suggested solutions. Enrico Ferri's volume on *Criminal Sociology* (1897),[4] and Cesare Lombroso's books on genius and insanity (1864),[5] criminal man (1876),[6] and criminal anthropology (1899),[7] built on an impressive foundation established by earlier physiognomists and phrenologists and the more recent works of L. S. Forbes Winslow (1844–1913),[8] Henry Maudsley (1835–1918),[9] Henry Mayhew (1812–1887),[10] and Henry Clarkell.[11] Together their research convinced legislators that governments must erect a defensive system that was social in purpose, preventive in accordance with the latest scientific studies of heredity, and repressive in contrast to prevailing laissez-faire systems and solutions.

Classifiers used such terms as "defective," "dependent," and "delinquent" which they called the "three D's," or added the terms "deficient" and "degenerate" to make the so-called "five D's." Recognizing that the cost of caring for these classes ran into hundreds of millions of dollars, reform-minded physicians, scientists, and social scientists required little argument to conclude that future generations faced the burden of maintaining "this vast army of dependents and criminals" unless society planned some form of corrective action.[12(p17)]

Eventually, many of those groups that advocated reform settled on ten separate cacogenic varieties: feeble-minded, pauper, inebriate, criminalistic, epileptic, insane, constitutionally weak, diathetic, physically deformed, and cacesthenic. These categories were derived from a variety of legal, medical, and biological criteria. Within each of

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these categories more specific subgroups were identified. For example, Bleecker Van Wagenen of the Eugenics Record Office in Cold Spring Harbor (Long Island, NY) counted within the feeble-minded class the subgroups of "moronic," "microcephalic," "epileptic," "amaurotic-idiotic," "cretinic," "mongolic," "anaesthetic," "toxic," and "traumatic." The pauper class contained "tramps," "beggars," and "ne'er-do-wells," while the inebriate class contained "dipsomania," "chronic alcoholism," and "pharmacomania." Similar subgroups existed within other major categories. In like manner, physicians, scientists, and social scientists divided the criminal classes into those of sound mind who were driven to crime by environmental influences and those of unsound mind who inherited from ancestors certain degenerate tendencies. The problem for the experts lay in discerning the causation for each individual.[12(pp17–21),13]

Hereditary drunkenness ranked high among the causes most frequently mentioned as producing the class identified as "defective." Of 1,744 inebriates in a 1901 study on heredity published in the *Journal of Insanity*, the author counted nearly 1,100 as having a history of drunkenness in the family. Hereditary drunkenness seemed prevalent in the criminal degenerate as well as in imbecility, idiocy, epileptic offspring, and insanity.[14] As W. O. Henry, MD, noted in a speech read before the Nebraska State Health Association in 1913, the insane gave a "hereditary taint of weakness and mental instability to their offspring." Moreover, in many instances the insane were themselves the offspring of parents who evidenced alcoholism, epilepsy, or other nervous disorders, including the excessive use of morphine, cocaine, and tobacco.[15]

Like many physicians who cared for defectives in state asylums, James R. Bloss, MD, Assistant Physician to the West Virginia Asylum, turned his attention to the prevention of defectives. Believing that the good of the individual ranked second to the welfare of society and preservation of the race, he focused his attention on the potential ability of the habitual criminal, ignorant degenerate, imbecile, and insane person to produce offspring. Bloss reported that in the period from 1870 to 1908, the ratio of insane people under state care had increased from one for every 2,135 of the population to one for every 583. Fearing the continued increase in the number of defectives whose upkeep had to be borne by the state and recognizing that the cost of their upkeep would continue to drain critical resources, Bloss recommended that the state initiate steps to prevent the continued increase of this element.[16]

The nature-nurture debate dominated the discussion of defectives, delinquents, and criminals in both the popular press and the professional literature. Articles abounded on such topics as marriage between first cousins, often concluding that consanguineous marriages tended to generate idiocy, insanity, and even sterility. Interestingly, American physicians, scientists, and social scientists remained divided on whether the majority of the insane, idiots, and feeble-minded were products of hereditary transmission or of the environment. Many social scientists retained a cultural bias for the environmentalist's view of change, while physicians and scientists increasingly sided with heredity. Even those who insisted that environment really shaped the career of the individual admitted that "heredity invites an environment that will favor its continuance." Although unclear with respect to the actual mechanism of hereditary transmission, physicians and scientists were not shy in suggesting Spencer's physiological units, Darwin's pangenesis, Galton's stirpiculture theory, Weisman's germ plasm, Lamarck's theory of inherited characteristics, or Mendelian genetics as possible hypotheses. Exemplary of this influence was the American Breeders' Association, which came into existence as a direct result of the rediscovery of Mendel's law. Organized in 1905, it changed its name in 1913 to the American Genetic Association.[14,17]

By the early 20th century, physicians, scientists, and social scientists had traced various family histories, compiling evidence that mental defectiveness was transmitted from parent to offspring. Significant among these genealogical trees were the Juke (Dugdale),[18] Kallikak (Goddard),[19] and Zero (Jorger)[20] families as well as the Tribe of Ishmael (McCulloch).[21]

Notoriety regarding the Juke family made a considerable impact on the sciences and social sciences. Written by Robert L. Dugdale, the book *The Jukes, A Study in Crime, Pauperism and Heredity* recorded the history of a family of Dutch heritage beginning in New York about 1730. Although the family did include some "honest workers" among the 1,200 descendants of Ada and Max Juke, it also contained 709 criminals, prostitutes, vagabonds, and paupers. Criminals accounted for 76 of the 709, and of the females, 176 were characterized as "disreputable" or "abandoned women." Dugdale estimated that the Juke family had cost New York State $1.25 million and, while he stressed environment as a determining factor in the lives of many of the Jukes, most readers preferred to believe that heredity played a far more important role in the creation from generation to generation of so many paupers, feeble-minded, prostitutes, and criminals.[15,22,23]

The Kallikak family, whose descendants of Martin and Deborah numbered 480, had among them 143 known feeble-minded, 36 illegitimate, 33 sexually immoral, 24 confirmed alcoholics, 3 epileptics, 3 criminals, 8 keepers of brothels, and 83 children so constitutionally feeble that they died in infancy. H. H. Goddard, author of *The Kallikak Family*, demonstrated the effects of heredity and the negligible influence of environment on crime, feeblemindedness, and pauperism in American society.[19]

Believing that eugenics amounted to nothing more or less than the civilizing of the reproductive instinct, H. E. Jordan, professor of histology and embryology at the University of Virginia at Charlotteville, urged its use as a form of "applied evolution in the interests of mankind." Attributing to Sir Francis Galton the responsibility for founding the science of eugenics with his books *Hereditary Genius*,[24] *Inquiries Into Human Faculty and Its Development*,[25] and *Natural Inheritance*,[26] Jordan urged that eugenics become, like religion, an essential component of the national consciousness. Although voluntary celibacy or abstinence from parenthood sufficed for some in securing society's protection from economic burdens and racial decay, he recommended more repressive efforts for those "unwilling or incapable of response to internal appeals." Jordan applied the designation "grossly and obviously unfit" to the feeble-minded, imbeciles, idiots, insane, epileptics, confirmed criminals, inebriates, vagrants, and paupers. These were either racially unfit or unworthy to reproduce. In either case, science left little doubt regarding the inheritance of their characteristics. "Probably no all-round great man or woman has ever come from such stock," he wrote. "Their offspring is of practically the same type and can do naught to abet—although much to hinder—human progress." Although this "unfit portion" might constitute only 10% of the population, their economic burden and unusual fecundity made them a serious impediment to the needs and aspirations of the state.[27]

Even Charles W. Eliot, the president of Harvard University, urged a closer examination of social defectives and advocated improved methods for their isolation to prevent procreation. Recognizing that research into the history of certain families had demonstrated a clear propensity among some for criminal activity, he warned that leaving them free would simply allow them to perpetuate and multiply their tainted inheritance. "The habitual criminal, who has demonstrated his quality to the public authorities, should be effectively prevented from propagating his kind," Eliot wrote. "The democratic governments must demonstrate that the free pursuit of happiness which they guarantee to their citizens does not include freedom to commit crimes or to propagate degeneracy."[28]

OPTIONS

Physicians suggested a variety of options including prohibition of marriage between first cousins; tighter controls over scandalous publicity, criminal activity, child labor, and "cheap novels;" legislation requiring compulsory education and a healthier social and moral environment for children; restrictions on the use of alcohol; prohibition of marriage of all persons legally judged insane, idiotic, epileptic, or feebleminded; and sterilization in the event that legislators found the prohibition of marriage too severe. Those supporting sterilization typically argued that the defective classes became a menace to each successive generation by reason of their degenerative heredity; that sterilization of degenerates was sound public policy; that the right of self-defense applied to both nations and individuals; and that habitual criminals and defectives were manifestly unfit for rearing children.[29]

According to Austin Flint, MD, the several options available to the state, including emasculation, regulation of marriage, segregation, and colonization, were really not options at all. Because emasculation worked such a psychical and physical change in the individual, Flint felt that few would countenance its use as state policy. And although numerous reformers recommended the regulation of marriage, skeptics recognized very quickly that regulation (including prohibition) did not guarantee the control of propagation. In fact, Flint feared that restriction on marriage would simply increase the number of illegitimate children and spurious relationships. Segregation prevented commingling of the sexes but, here again, few believed in its effectiveness since it required virtual imprisonment. As for colonization, this solution applied only to those countries having overseas colonies and, even then, simply transferred the problem to another setting.[1]

Reformers also recommended the establishment of "backward classes" in the public schools. This grouping or "typing" of students promised to facilitate more intelligent inspection as well as special training. Of those characterized as "irretrievably backward," reformers advised asexualization and, if necessary, permanent sequestration. For Martin W. Barr, MD, of Pennsylvania, castration and oophorectomy offered the most preferable forms of asexualization. When performed on the young, he argued, sexual desire "almost entirely ceases, or is at least held in reasonable abeyance." If, for sentimental reasons, society preferred a less drastic measure, then he recommended vasectomy in the male and fallectomy or tuberectomy in the female. These latter operations did not deter sexual desire or sexual power, only the ability to impregnate or conceive.[30]

According to Barr, who was chief physician for the Pennsylvania Training School for Feeble-Minded Children, there were some 10,000 cases of imbecility in his state, of which only 3,500 were sequestrated. "As sexual impulses dominate their lives," he wrote, "the removal of this excitation, as has been proven, not only makes them more tractable, as does gelding for the ox, but the general health improves, and numerous disorders, to which many are subject, become more amenable to treatment." He found that asexualization improved the physical vigor of the individuals and they "became contented and happy."[30]

Not all physicians were convinced that sterilization alone would protect society from the unimprovable criminal, the hopeless delinquent, or the incurably insane. For some, the state had to both sterilize and separate the individual from society. Anything less left society with a false sense of security from venereal disease or other equally dangerous social evils. By itself, sterilization actually increased the danger to society unless accompanied by appropriate controls over the individual's movement. Thus, the need for penitentiaries, reform schools, and other forms of institutionalization was by no means lessened by the sterilization.[31]

Of the available options, H. E. Jordan recommended effective isolation during the procreative years of the "genetically tainted individual," or sterilization. In the case of the epileptic or tuberculous male, Jordan recommended vasectomy. In instances of sexual perversion and rape, however, he advised castration. For the feeble-minded, the idiotic, and other mentally deficient types, he favored isolation. In the latter instance, however, he also favored vasectomy. Important in every case was the need to provide for a thorough scientific investigation to determine whether the defects were the result of heredity or environment.[27]

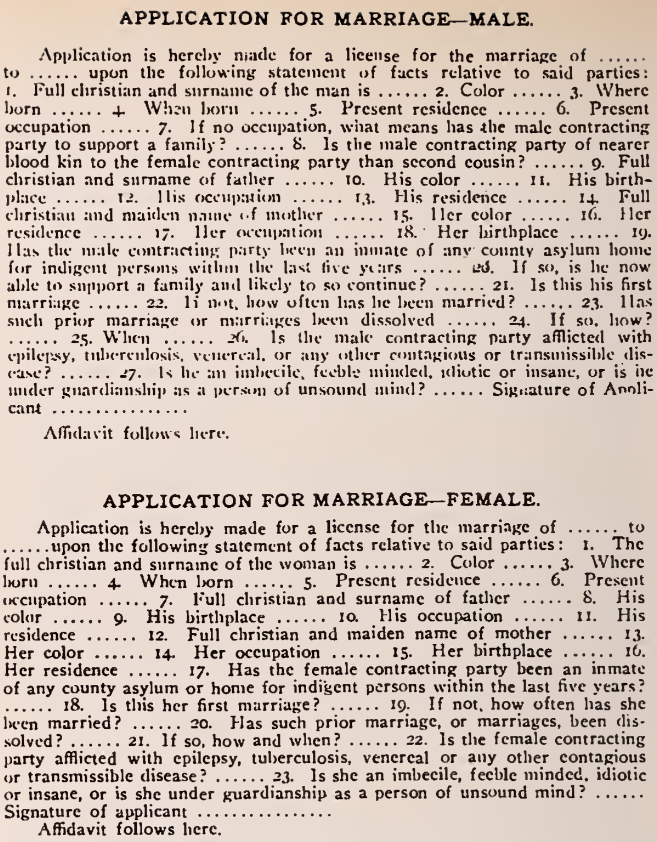
In 1903, Robert R. Rentoul, MD, author of the book *Race Culture; or Race Suicide*, proposed that governments consider prohibiting marriage for any idiot, imbecile, feebleminded, epileptic lunatic, chronic inebriate, habitual vagrant, habitual criminal, drug user, sexual pervert, or deaf-mute, or anyone markedly neurotic. He also proposed that these types be sterilized as well since they were not legally responsible for their actions. His basis for proposing these steps resulted from his belief that governments had been responsible enough to institutionalize certain degenerates but derelict in preventing their ability to produce children. Studies of unmarried feebleminded females assigned to workhouses indicated that they continued to produce idiot children.[29]

Reformers were often reluctant to use the power of the state to restrict procreation through tighter controls on marriage. While some held that the state had the right to interfere in these matters, others rejected this intrusion as a violation of personal liberty. "I do not believe that the state should regard its citizens as the stock breeder regards his stock," remarked Burnside Foster, MD, of St Paul, Minnesota, "and pick out for breeders only the finest specimens, although there is no doubt but that if this were done there would result a race of human beings stronger physically than any race the world has ever seen." Foster did not oppose sterilization any more than he opposed laws prohibiting marriage. However, he noted that laws prohibiting marriage based on physical examination would be unenforceable unless such laws were uniform in all states.[32]

Although physicians, scientists, and social scientists agreed on the seriousness of the problem and the impact which it had on race survival, economics, and the condition of society and government, they were less than unanimous on possible solutions. Arkansas, Arizona, Colorado, Oregon, Louisiana, Ohio, Indiana, Nevada, Washington, and Montana passed legislation prohibiting marriage between first cousins. Other states, such as Michigan, prohibited the marriage of persons with syphilis or gonorrhea, while in Connecticut the prohibition of marriage extended to epileptics, imbeciles, and the feeble-minded. Connecticut also prohibited sexual intercourse for women younger than 45 who were identified as epileptic, imbecilic, or feeble-minded. Minnesota passed a similar law, which stated that "no woman under the age of forty-five years, or man of any age, except he marry a woman over the age of forty-five years, either of whom is epileptic, imbecile, feeble-minded, or afflicted with insanity, shall hereafter intermarry or marry any other persons within this state. It is also hereby made unlawful for any person to marry any such feeble-minded, imbecile or epileptic person, or any one afflicted with insanity.[14,33]

The information physicians considered essential for certificates of marriage included knowledge regarding any transmissible disease (ie, syphilis or tuberculosis), and disabilities or hereditary taints such as insanity or epilepsy. This information ensured that the state placed a premium on health, that it exalted the diginity of parenthood, preserved the health of generations, provided untold economic benefit to the state, and promoted the "survival of the fittest" in a physical, civil, economic, and social sense if not also in a moral and religious sense. Accordingly, Delaware (1893), Minnesota (1904), New Jersey (1904), and Ohio (1905) prohibited certain "degenerates" from marrying without a prenuptial certificate of good health and sterilization.[3,15]

Following passage of a 1905 law, the State Board of Health in Indiana prohibited the issuance of marriage licenses to its citizens except on completion of a written and verified application. The law stipulated that "No license to marry shall be issued when either of the contracting parties is an imbecile, epileptic, or of unsound mind, or under guardianship of a person of unsound mind, or to any male person who is or has been within five years an inmate of any county asylum or home for indigent persons, unless it satisfactorily appears that the cause of such condition has been removed, and that such male applicant is able to support a family and likely to so continue, nor shall any license be issued when either of the contracting parties is afflicted with a transmissible disease, or at the time of application is under the influence of an intoxicating liquor or narcotic drug."[1]



APPLICATION FOR MARRIAGE—MALE.

Application is hereby made for a license for the marriage of ...... to ...... upon the following statement of facts relative to said parties: 1. Full christian and surname of the man is ...... 2. Color ...... 3. Where born ...... 4. When born ...... 5. Present residence ...... 6. Present occupation ...... 7. If no occupation, what means has the male contracting party to support a family? ...... 8. Is the male contracting party of nearer blood kin to the female contracting party than second cousin? ...... 9. Full christian and surname of father ...... 10. His color ...... 11. His birthplace ...... 12. His occupation ...... 13. His residence ...... 14. Full christian and maiden name of mother ...... 15. Her color ...... 16. Her residence ...... 17. Her occupation ...... 18. Her birthplace ...... 19. Has the male contracting party been an inmate of any county asylum home for indigent persons within the last five years ...... 20. If so, is he now able to support a family and likely to so continue? ...... 21. Is this his first marriage ...... 22. If not, how often has he been married? ...... 23. Has such prior marriage or marriages been dissolved ...... 24. If so, how? ...... 25. When ...... 26. Is the male contracting party afflicted with epilepsy, tuberculosis, venereal, or any other contagious or transmissible disease? ...... 27. Is he an imbecile, feeble minded, idiotic or insane, or is he under guardianship as a person of unsound mind? ...... Signature of Applicant ....

Affidavit follows here.

APPLICATION FOR MARRIAGE—FEMALE.

Application is hereby made for a license for the marriage of ...... to ......upon the following statement of facts relative to said parties: 1. The full christian and surname of the woman is ...... 2. Color ...... 3. Where born ...... 4. When born ...... 5. Present residence ...... 6. Present occupation ...... 7. Full christian and surname of father ...... 8. His color ...... 9. His birthplace ...... 10. His occupation ...... 11. His residence ...... 12. Full christian and maiden name of mother ...... 13. Her color ...... 14. Her occupation ...... 15. Her birthplace ...... 16. Her residence ...... 17. Has the female contracting party been an inmate of any county asylum or home for indigent persons within the last five years? ...... 18. Is this her first marriage? ...... 19. If not, how often has she been married? ...... 20. Has such prior marriage, or marriages, been dissolved? ...... 21. If so, how and when? ...... 22. Is the female contracting party afflicted with epilepsy, tuberculosis, venereal or any other contagious or transmissible disease? ...... 23. Is she an imbecile, feeble minded, idiotic or insane, or is she under guardianship as a person of unsound mind? ...... Signature of applicant ....

Affidavit follows here.

FIGURE 1. Marriage license application, Indiana, 1913 (from George RR: Sterilization of the unfit. *N Engl Med Gazette* 1913; 48:472–473).

In 1894, F. Hoyt Pilcher, MD, who supervised the Kansas Asylum for Idiotic and Imbecile Youth, reported on the asexualization of 58 feeble-minded boys and girls.[34,35] In 1907, in an article in the *American Journal of Surgery*, M. E. Van Meter, MD, of San Francisco recalled his own experiments in 1896 which were intended to eradicate the curse of certain hereditary problems. His sterilization of both men and women led him to suggest sterilization as a means of preventing procreation by those predisposed to crime and those with hereditary problems. He concluded that it "will make mankind a larger, stronger, healthier, happier and better race of beings."[36]

According to Van Meter, American society had a duty "to compel a contaminated man to submit to sterilization [rather than] allow him to become the father of a half dozen children with mental or physical defects that will blight their whole lives and, in turn, which they will transmit to still other innocents." He included among those to be sterilized all habitual drunkards, kleptomaniacs, moral perverts, drug offenders, those who were guilty of penitentiary offenses, and those who "either from incapacity or lack of will [had become] a pauper or public charge."[37]

H. C. Sharp, MD, of Indianapolis, who served as physician to the Indiana State Reformatory at Jeffersonville, performed the first known vasectomies on criminals in 1899. Performed with the consent of the convict, vasectomy was used in 176 cases over a period of several years before the practice became enacted into law. Sharp recalled one inmate of the Indiana State Reformatory who requested the operation in order to decrease his sexual desires and avoid the possibility of having children. Following his vasectomy, the inmate claimed to have "improved in mind and body," gaining weight, strength, and the power to concentrate. Another inmate claimed to have gained 40 pounds following the operation.[38,39]

In 1908, Charles V. Carrington, MD, surgeon to the Virginia State Penitentiary, urged the sterilization of "habitual criminals" as a means of ultimately reducing the size of the criminal class. By "habitual," Carrington included all criminals returning to prison for a second offense, and those convicted of murder in the first degree, arson, rape, or train-wrecking. In studying the prison rolls of Virginia institutions for a period of 50 years, Carrington discovered that certain families were "regularly represented" in the prisons and would continue being represented unless the state decided to stop the "breed." He admitted to having sterilized two habitual criminals at the Virginia Penitentiary. Carrington described one of the criminals as a "consistent masturbator" who, upon sterilization, became "a slick, fat, docile prisoner, a trusty about the yard." The other, Carrington described as "a notoriously lusty, beastful Sodomist and masturbator" who on returning to prison as a second termer was sterilized and became "a docile, model prisoner."[40]

Physicians ranked the various forms of sterilization in order of their efficiency: double orchiectomy with amputation of the glans penis, double testectomy, double oophorectomy, vasectomy, and x-ray. Double testectomy with amputation of the glans penis involved total mutilation of the male sexual organ. Considered the most radical choice, it destroyed both the desire and the ability to procreate and had the additional effect of changing the mental, moral, and physical status of the individual. Double oophorectomy sterilized the woman through removal of the ovaries; unlike the double orchiectomy, however, the operation did not destroy either the sexual ability or desire. Yet another operation, clitoridectomy, removed the woman's ability to experience pleasure in sexual intercourse, although it did not destroy her sexual ability.

Most physicians preferred vasectomy as the least destructive yet most efficient remedy for the male. For females, physicians urged the use of salpingectomy or oophorectomy. Vasectomy, or ligation of the vas, caused sterility without destroying the desire or ability to copulate. Nor did it change the physical, mental, or moral attributes of the individual. A simple operation, vasectomy required but a few minutes of a surgeon's work and resulted in little inconvenience to the patient. During copulation, the male ejaculated semen but not spermatozoa.[41]

The x-ray, discovered by Wilhelm Conrad Roentgen in 1895, offered still another form of sterilization as did radium treatment, developed by Henri Dominici. Both were known to temporarily prevent conception without lessening the desire or ability to copulate. Physician Rex Duncan of Los Angeles recommended a single intrauterine application of 2,500 or more millicurie hours of radium over a period of four to 24 hours to produce sterility in the female. X-ray treatment over the pelvis accomplished the same result in one or more one-hour applications. Most physicians, however, considered x-ray and radium too dangerous, and few actually used them in the treatment of criminals and degenerates.[42–44]

LEGISLATION

Although the issue was discussed in France, Germany, England, and Switzerland, the United States became the first nation to legislate the sterilization of certain "degenerates." In 1905, the Pennsylvania legislature passed a sterilization bill that empowered every institution in the state entrusted with the care of idiots and imbecile children to examine their mental and physical condition. Further, it created a committee of experts who, in conjunction with the institution's board of trustees, determined when the individual was non-improvable and procreation thus inadvisable. Despite legislative approval, Governor Samuel W. Pennybacker vetoed the bill. Similarly, the legislature of the state of Wisconsin discussed a sterilization bill but tabled any action in 1906 while, in 1908, the Oregon legislature passed a bill which was not signed by the governor.[29]

In 1907, the Indiana legislature passed a bill authorizing sterilization. The Indiana bill (H 364), approved March 9, 1907, by Governor J. Frank Hanly, stated the following:

> Preamble. Whereas, heredity plays a most important part in the transmission of crime, idiocy and imbecility; therefore, Be it enacted by the General Assembly of the State of Indiana, That on and after the passage of this act it shall be compulsory for each and every institution in the state entrusted with the care of confirmed criminals, idiots, rapists and imbeciles to appoint upon its staff, in addition to the regular institutional physician, two (2) skilled surgeons of recognized ability, whose duty it shall be, in conjunction with the chief physician of the institution, to examine the mental and physical condition of such inmates as are recommended by the institutional physician and board of managers. If, in the judgment of the committee of experts and board of managers, procreation is inadvisable and there is no probability of improvement of the mental condition of the inmate, it shall be lawful for the surgeons to perform such operation for the prevention of procreation as shall be decided safest and most effective. But this operation shall not be performed except in cases that have been pronounced unimprovable; provided, that in no case shall the consultation fee be more than three ($3.00) dollars for each expert, to be paid out of the funds appropriated for the maintenance of such institution.[22]

During the first year following passage of the Indiana law, physicians operated on 412 male inmates in the reformatory at Jeffersonville. Although applied only to male offenders, the Indiana law did permit the operation on women as well.[32] Within two years, nearly 800 persons underwent sterilization, and the so-called "Indiana plan" passed in Oregon and Connecticut. By 1912, 1,200 men had undergone vasectomies. The *Journal of the American Medical Association*, the Chicago Physicians' Club, the Southern District Medical Society, the Chicago Society of Social Hygiene and the Chicago *Evening Post* went on record supporting the "Indiana plan" and similar legislation across the country. According to the Chicago *Tri-*

bune, "sterilization of defectives and habitual criminals is a measure of social economy."[38,45]

According to Dr Hart, Director of the Department of Child-Helping of the Russell Sage Foundation, the statistics on sterilization were misleading. By his own tally, Indiana physicians performed 119 operations in 1907–1908 and 39 in 1908–1909. None occurred in the following two years. Moreover, according to Hart, there was little enthusiasm to implement sterilization in those states such as Connecticut, Washington, New Jersey, Iowa, New York, and Nevada where the legislation was already in force.[46]

H. C. Sharp claimed to have kept 236 cases of vasectomy under postoperative observation and determined that there had been no atrophy of the sexual organs, no cystic degeneration, and no disturbed mental or nervous condition. Most articles that reflected on the positive results of vasectomy referred directly to Sharp's work in Indiana. In his published papers, Sharp often described how patients acquired a "more sunny disposition, [were] brighter of intellect," ceased "excessive masturbation" and demonstrated a "decided increase of energy and well-being."[1,47]

As a result of Sharp's postoperative descriptions, authors often misattributed the results of castration to those of vasectomy. Physicians who performed a vasectomy often spoke of the patients' lowered sexual desires as a result of the operation, of becoming more content, having fewer erections, and less desire for masturbation.[48] H. E. Jordan of the University of Virginia described one "sexual pervert" as having gained weight, ceasing his "foul practices," and becoming "cheerful and more tractable." Although unsure whether the result stemmed from mental suggestion or some indirect effect on the sex glands, Jordan concluded that vasectomies often achieved the effect of castration without the risk or inconvenience.[27]

Following the reputed success of the Indiana plan, California legislated its own act on April 26, 1909, which authorized the asexualization of inmates of state hospitals and the California Home for the Care and Training of Feeble-Minded Children and of convicts in the state prisons. Unlike the Indiana law, which placed the implementation of the act in the hands of a committee of two surgeons and the institution's physician, the California act required the superintendent or resident physician to bring the issue before the general superintendent of state hospitals and the secretary of the State Board of Health. Moreover, the California act included individuals who committed sexual offenses outside the United States. Neither Indiana or California stipulated exactly what operations should be performed, preferring as California did to simply use the term "asexualised." To some critics, the broadness of the term allowed operations that could rob the patients of their sexual characteristics rather than simply their procreative powers.[29]

On August 12, 1909, the state legislature of Connecticut enacted legislation that permitted the directors of the state prison and the superintendent of state hospitals for the insane to appoint surgeons to examine individuals for whom procreation would be "inadvisable."

> Such board shall examine the physical and mental condition of such persons and their record and family history so far as the same can be ascertained, and if in the judgment of the majority of the said board procreation by any such person would produce children with an inherited tendency to crime, insanity, feeble-mindedness, and idiocy or imbecility, and there is no probability that the condition of any such person so examined will improve to such an extent as to render procreation by such person advisable, or if the physical or mental condition of any such person will be substantially improved thereby, then the said board shall appoint one of its members to perform the operation of vasectomy or oophorectomy, as the case may be, upon such person.[29]

Two years later, an Iowa law went into effect that legalized the prevention of procreation of certain habitual criminals, idiots, feeble-minded individuals, imbeciles, drunkards, drug fiends, epileptics, syphilitics, and moral or sexual perverts who were then confined in state institutions. The law authorized the operating officer in each of the institutions housing these classes of individuals to examine their respective mental and physical condition and determine whether it was advisable or inadvisable to allow any of them to procreate. Following consultation with the parole board as well as with the surgical superintendent of the institution, the state permitted the surgeon to perform a vasectomy or ligation of the fallopian tubes.[41,49]

In 1912, the New York State Senate and Assembly created a board of examiners consisting of three individuals appointed by the governor (a surgeon, a neurologist, and a practitioner of medicine) to examine the feeble-minded, epileptic, criminal, and other defective inmates in the state hospitals for the insane, state prisons, reformatories, and charitable and penal institutions. The examination board was to determine which persons would produce offspring that would inherit a tendency toward crime or insanity. Those so judged could then be sterilized.[50]

By 1913, eight states had passed sterilization laws which authorized or required surgical sterilization of certain classes of defectives or degenerates. These states included Indiana in 1907; Connecticut, Washington, and California in 1909; Iowa, Nevada, and New Jersey in

TABLE I. Analysis by States of Sterilization Laws Enacted Prior to January 1, 1920*

Indiana	1907
Washington	1909
California	
First statute	1909
Second statute	1913
Sterilization provision in act establishing Pacific Colony	1917
Connecticut	1909
Nevada	1911
Iowa	
First statute	1911
Second statute	1913
Third statute	1915
New Jersey	1911
New York	1912
Michigan	1913
Kansas	
First statute	1913
Second statute	1917
Wisconsin	1913
Nebraska	1915
Oregon	1917
South Dakota	1917

* From Laughlin HH: Eugenical sterilization in the United States. *Social Hygiene* 1920; 6:531.

1911; and New York in 1912. The laws permitted sterilization for confirmed criminals in all of the states; for rapists in five; for idiots, imbeciles, and the feeble-minded in six; for the insane in four; and for epileptics in two. In all of the states, the decision to sterilize was the responsibility of a commission or board. In 1914, sterilization bills were before the New Mexico, Michigan, Ohio, and Utah legislatures. In Nevada and Washington, the laws limited sterilization to individuals judged guilty of carnal abuse of females under the age of ten years or of rape, or to those who were judged to be habitual criminals.[27]

The sterilization laws were held constitutional in California and Washington but declared unconstitutional in Wisconsin, Connecticut, New York, North Dakota, and Michigan. Moreover, an additional 20 states attempted but failed to secure sterilization laws through their legislatures. Of the 12, eight were in the South. The governors of four states—Pennsylvania, Oregon, Vermont, and Nebraska—vetoed sterilization legislation and, in Oregon, where the statute was subsequently passed and approved, the people later revoked it by a referendum.[51]

G. Frank Lydston, MD, professor of genitourinary surgery at the medical department of the University of Illinois, became one of the most vocal apologists among physicians for the sterilization movement. A prolific writer on a variety of social topics, he attempted to demonstrate the relationship between medicine, the individual, and the body politic. He believed that the study of sociology developed out of social pathology which, in turn, revolved around individual pathology. Beneath all social ills, he argued, lay individual and social degeneracy. Degeneracy in "physical fibre" preceded degeneracy in morals. Thus, measures which lessened the number of individuals associated with degeneracy directly lessened the level of social problems. "The most effective attack upon degeneracy and its evil social brood," he wrote, "must be aimed at the reproductive powers of defective social integers. It is here that we may dam the tainted social streams at their very sources."[52]

TABLE II. Sterilization Procedures in the United States, 1894–1921*

State	No. of Procedures
California	2558†
Connecticut	27
Indiana	120
Iowa	49
Kansas	54
Michigan	1
Nebraska	155
Nevada	0
New Jersey	0
New York	42
North Dakota	23
Oregon	127
South Dakota	0
Washington	1
Wisconsin	76
Total	3233

* Between 1894 and 1921, some 3,233 operations had taken place under the sterilization laws in place in 15 states. Of that number, the feeble-minded represented 403, the insane some 2,700 and the criminalistic 130.

† The State Hospital for the Insane at Patton was responsible for 1,009 of the cases.

From Eugenical sterilization. *J Heredity* 1923; 14:308–309.

Like others before him, Lydston praised Indiana for its progressive legislation in having adopted sex mutilation as a remedy for crime and degeneracy. However, Lydston did not credit Sharp with the so-called "Indiana plan." Instead, he attributed the genesis of the idea of asexualization of criminals to Gideon Lincecum, MD, of Texas, who proposed sterilization to the Texas State Legislature in the late 1870s as a substitute for capital punishment. As early as 1893, Lydston himself had proposed castration as a solution to the sexual crimes committed by southern blacks. Again, in 1905, Lydston presented sterilization and asexualization in both sexes as a solution for social disease. In his book *Diseases of Society*, Lydston argued that most of the crimes against society were "more or less remediable by castration or resection of the vasa deferential and the Fallopian tubes."[52,53]

According to Lydston, society "began its self-contamination at the marriage license window." There the "stream of degeneracy" first started its movement into the social system. Accordingly, he found it difficult to accept that so important a step for both the individual and the society would be taken without any seeming regulation, control, or supervision by the state. In other words, it was the marriage license that set in motion both the individual and social machinery that permitted the "manufacture of degenerates."

> The license window is the place where the honest citizen and the criminal, the sane and the insane, the diseased and the healthy, the pauper and the millionaire, the learned and the ignorant, the intellectual and the weak minded, may meet upon common ground—providing always that the important consideration of the license fee is forthcoming. The criminal, the insane, the epileptic, the syphilitic, and the drunkard are here authorized by law to begin the procreation of their kind, the number of their progeny being limited entirely by the volition and physical capacity of the individuals immediately concerned.[52]

Lydston urged that states authorize marriages on the same basis that life insurance companies authorized life policies, ie, that certain marriages be prohibited as "bad risks." Although the "sanitary marriage" was probably "an idealist's dream," Lydston hoped that society might eventually insist on some level of control based on "rational scientific principles." For example, society might consider it a crime for persons infected with active stages of venereal disease to infect innocent persons through marriage.[52,54,55]

Already, wrote Lydston, the state had insisted on some regulation of marriage. This included laws stipulating the ages of candidates for matrimony, prohibitions on consanguineous marriages (even to the fourth degree of consanguinity), and miscegenation. It therefore seemed inconsistent that sentimental objections would prohibit further regulation based on "proper physical qualifications." It also seemed inconsistent to Lydston that society demanded examinations and licenses for engineers, lawyers, physicians, dentists, and pharmacists to protect the public yet held back from similar such requirements in matrimony. For Lydston, marriage should be governed by society on "strictly business principles, patterned after those of life insurance companies, in the management of which sentiment is an unknown quantity." In other words, prior to the issuance of a marriage license, the parties should be required to obtain a certificate regarding their "physical condition from a nonpolitical and therefore nonpartisan board of medical examiners." The board, consisting of three physicians, should consider both the physical and moral qualifications of the candidates. Individuals who in the face of unfavorable medical opinion still insisted on marriage could do so provided they undergo sterilization.[52]

Lydston did not support eugenics for the human race. The comparison between scientific breeding of animals and of man yielded artificial qualities that could prove disastrous. Since the qualities that made a "superior" domestic animal could just as easily produce "abnormal" types in human kind, Lydston urged genuine caution in any proposed ameliorative legislation.

> It is obvious that some of the physically unfit are of great value to the world, and if bred with the proper "corrective" blood may possibly reproduce their betters in physique and their kind in value. Many a short-lived genius has accomplished wonders. So many geniuses have been physically or morally degenerate that it is rather easy to estimate the price the world must pay for its geniuses and the price the genius himself must pay for his genius. Genius is largely a loss of physical and psychic balance. Breed genius to genius and, as I have intimated, where the union is not sterile the progeny are likely to be few, short-lived, and degenerate. Not only do physical perfection and overtopping genius rarely go together, but often times the genius is a biological sport. Breed him so as to perfect the physical man and we insure the loss of genius in his progeny. All those things which raise man in the scale of intelligence, beyond a certain point, tend in general to lower him from the standpoint of physical animality.[52]

EUGENICS RECORD OFFICE

In 1914, the Eugenics Record Office at Cold Spring Harbor issued a landmark document entitled "Report of the Committee to Study and to Report on the Best Practical Means of Cutting Off the Defective Germ-Plasm in the American Population." The Committee had as its expert advisors Alexis Carrel of the Rockefeller Institute in medicine, Alexander F. Chamberlain of Clark University in anthropology, Steward Paton of Princeton University in psychiatry, H. H. Goddard of Vineland Training School in psychology, Judge Warren W. Foster from the New York Court of General Sessions in criminology, Franklin H. Giddings from Columbia University in sociology, Robert DeC. Ward from Harvard University in immigration, Louis Marshall from the New York Bar Association in law, and Newell Dwight Hillis in religion and morals.[12(p5)]

The report originated at a meeting of the Eugenics Section of the American Breeders' Association in Palmer, Massachusetts, in May 1911. At this meeting, the Eugenics Section passed a resolution commissioning a study on the means for "cutting off the defective germ-plasm in the American population." Under the chairmanship of Bleecker Van Wagenen of New York City, the committee outlined its investigation, named experts to assist in its work, and presented its preliminary report to the First International Eugenics Congress in London in July 1912. The committee intended to investigate "all phases of the problem of cutting off the supply of defectives" and to publish data that seemed applicable to the issue. The committee likewise took on the role of pointing out those practical means available for "purging the blood of the American people of the handicapping and deteriorating influences of these anti-social classes."[12(pp5-6)]

In its first series of studies, the committee focused on the issue of sterilization as a eugenic means of protecting society and examined it from a variety of viewpoints including medicine, physiology, surgery, biology, thremmatology, anthropology, psychiatry, psychology, morals and ethics, woman's viewpoint, criminology, sociology, political economy, statistics, law, history, public affairs, and international cooperation. From the very beginning the committee relied on Charles B. Davenport, the resident director of the Eugenics Record Office, who served as consultant throughout its deliberations.[12(pp8-9)]

The remedies the committee investigated included life segregation (or segregation during the reproductive period), sterilization, restrictive marriage laws, eugenic education, systems of matings to remove defective traits, environmental changes, polygamy, euthanasia, neo-Malthusianism, and laissez-faire. The committee unanimously endorsed life segregation as "the principal agent used by society in cutting off its supply of defectives." On the other hand, the committee remained undecided on sterilization and proposed that the state sterilize individuals only as a supporting mechanism to the "more important feature of segregation."[12(pp46-47)] While the committee did not give much credence to restrictive marriage laws and customs, it did place considerable importance on eugenic education of both the public and of prospective marriage mates. The committee likewise supported the use of "selective forces" to control mate selection but did not specifically indicate what those forces would be. Presumably it entailed a greater understanding of the principles of heredity and therefore more active effort to control mate selection. The committee discounted polygamy on account of the sacredness of the monogamous marriage and discounted euthanasia—despite the Spartan social custom—as contrary to eugenic principles. Finally, the committee condemned neo-Malthusianism since it failed to cut off the supply of defectives and tended instead to adversely affect the fecundity of the better classes.[12(pp53-54)]

The committee concluded that a successful program for "cutting off the lower levels of the human breeding stock" ultimately meant encouraging increased fecundity among the better classes of people, or what the committee called "aristogenics." To be successful, however, eugenics had to become part of America's "civic religion," financed by the states. "If America is to escape the doom of nations generally," the committee observed, "it must breed good Americans. The fall of every nation in history has been due to many causes, but always chiefest among these causes has been the decline of the national stock."[12(p59),56]

OPPOSITION

The proponents of sterilization were not without opposition. Critics objected to these "esoteric medical practices" by raising ethical aspects, indicating a lack of confidence in the "short-cuts of benevolent despots," and condemning what they called "compulsory mutilation." Any remedy to the problem of degeneracy had to be "scientifically sound, legislatively possible and administratively practicable." Not the least of the problems encountered in such legislation would be the certification by medical men of the need for sexual mutilation. The legal obstacles would be enormous and the time consumed in judging individual cases heavy.[3]

George Bernard Shaw took considerable notice of the sterilization efforts and regretted that the scientific and medical communities had been unable to define "degeneration" with any real specification. "If we abolished the diseased we should lose a number of very interesting people," he wrote. "Might not an epileptic fit be a misapplied form of useful energy? Many 'backward' people were criminal only on account of our deplorable social conditions; were these altered, many now occupying high places would be snuffed out."[3]

Geoffrey Clarke, MD, a medical officer at Banstead Asylum in England, was very much interested in defective heredity and the study of eugenics. Improvements in medical science had resulted in keeping alive the weaklings of society. "By patching up both the physically and mentally defective," he wrote, society had favored "the propagation of the unfit" and tended as a result to weaken one of the basic rules of nature which constantly strived for the "survival of the fittest." Nevertheless, Clarke was not convinced that society was ready to recommend measures from a eugenic point of view. For one thing, increased insanity had been a natural result of advanced civilization. "The more complicated life is made," he wrote,

> the greater will be the number who fail to adapt themselves to the increasing complexity of their environment. Insanity is not a definite disease, but a variation from the average mental state; there is no definite line between sanity and insanity, and even the shadowy line that exists is drawn at a different level according to different social surroundings.

In effect, he found no reason to believe that lunacy was increasing out of proportion to society.[57]

Clarke likewise doubted whether improvements in mental and hygienic science actually favored the survival and multiplication of the unfit, thus moving society toward some level of national deterioration. Although this was a widely held opinion that alarmists continually raised in the popular press as well as the scientific journals, Clarke believed that more enlightened treatment of diseases had no doubt "improved the general stock of the country" and kept from perishing many weak and delicate children who not only survived but grew into strong and healthy adults.

> The arguments based upon the experience and methods of stockbreeders cannot fairly be applied to human beings. Breeders of pedigree animals, biologists, and experimenters on Mendelian and other lines are gradually collecting facts and formulating laws of inheritance; they teach us of normal and abnormal variations, they separate the hereditary characters from those due to nurture and environment, their work is invaluable to the student of eugenics, but they help us but little with the problem of insanity; they shed no light on the transmission of mental qualities, and are concerned purely with the physical. The pedigree heifer is probably a dement, the foxhound is a cheerful imbecile, the fleetest racehorse is often an irritable, dangerous rogue.[57]

The real question in Clarke's mind was whether or not there was sufficient knowledge regarding the transmission of mental disease to allow the state to say to certain of its citizens, "thou shalt not breed." He doubted there was sufficient scientific evidence to justify an artificial lowering of the numbers of the insane without doing an injustice to the individual and his rights.[57] Clarke was not convinced that sterilization would materially reduce the number of insane people. But what he found disconcerting was the popular inclination to advocate sterilization without hard evidence. "If the overwhelming figures which support the utility of vaccination from a national public health point of view fail to convince a large body of antivaccinators that it is justifiable to make vaccination compulsory, what of the figures in support of sterilization?" The truth was, he believed, that there were insufficient statistics on insanity to justify the recommended legislation. Little information existed in the accumulated tables and statistics of the normal relatives or offspring of the so-called degenerate. As a result, most asylum statistics were useless for purposes of addressing the critical public policy issues of the day.[57]

In general, critics found the sterilization laws premature, in obvious violation of the Bill of Rights, complicated by inadequate legal classification describing persons who should be subject to compulsory sterilization, and actually encouraging illicit intercourse and spreading venereal disease. Instead, they recommended further study of family histories, of insanity, and of the pathologic alterations of the brain that accompanied insanity.[46] Charles A. Boston, a New York lawyer, feared that the effort to purify the American people of its feeble-minded would place "one more nail in the coffin of those constitutional safeguards which have been the political cause of the prosperity of this country as the haven of the oppressed of all nations." He objected to making one tenth of the population the target of the other nine tenths by curtailing their right or privilege to have offspring. "I cannot conceive that the American people, whether individually they have defective germ plasm or not, will so long as they remain a free people tolerate beneficent decimation by sterilization."[58]

RETROSPECT

Important as it is to recount the origins of the early sterilization movement in America, there is good reason not to judge it outside the context of the age which gave it birth. This is because America's interest in the sterilization of criminals and defectives coincided with several very similar or connected issues in the press and professional journals—issues that tended to merge with and reinforce one another in the popular culture.

The first issue focused on the Negro and his supposed physical and mental deterioration since emancipation. Statistics gathered from the 1870 and 1880 census reports, along with medical studies carried out during and after the Civil War, suggested that the black population in the United States had increased incidences of insanity as well as all forms of debilitating diseases (particularly tuberculosis and syphilis), leading the majority of physicians, scientists, and social scientists to conclude that the race was actually dying out. Arguing that slavery had protected the Negro in a virtual "hothouse" environment from the rigors of race struggle, they theorized that emancipation had forced the Negro into an unequal struggle with superior races for survival of the fittest.[59–63]

A second related issue of this period centered on the massive immigration of peoples from southern and eastern Europe whose customs, laws, and even physical appearance were deemed alien to "old stock" Americans. Studies on these immigrants frequently referenced their constitutional inferiority and their tendency toward a higher incidence of disease, insanity, and pauperism.[64–66] Tied closely to the issue of the new immigrant was the all-consuming fear that "old stock" Americans were not replenishing themselves at the same rate as these newer "breeds." With this recognition came the belief that "American" customs and laws would face extinction or at least demonstrative restructuring as more immigrants washed ashore with each new wave.[67–69]

The convergence of these issues in the late 19th and early 20th century gave added meaning and substance to the concerns raised over the numbers of criminals and degenerates populating prisons, correctional houses, asylums, and hospitals. Seen in their totality, they gave credence to the opinion held by many Americans that the traditional laissez-faire approach to the problems of immigration, degeneracy, and race deterioration no longer seemed moral or practical. Instead, American society turned to interventionist solutions—immigration restriction, legalized segregation, and, in the case of the criminal and defective population, sterilization. In this context, the sterilization laws conformed with much of the action-oriented legislation debated and discussed at the turn of the century. These laws also represent the impact that hereditarian ideas had on American life and institutions. While the nature-nurture debate did not end with a definitive triumph of the hereditarian point of view, environmental arguments played a decidedly secondary role during these turbulent years.

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FROM THE LIBRARY

SUPREME COURT OF THE UNITED STATES UPHOLDS THE VIRGINIA STATUTE PROVIDING FOR THE STERILIZATION OF MENTAL DEFECTIVES

The recent decision of the Supreme Court of the United States in the case of *Buck vs. Bell*, upholding the Virginia statute providing for the sterilization of mental defectives under careful safeguards, should arouse interest not among physicians only, but with all persons who are interested in the problems of eugenics and the betterment of the race. It should be of special interest to the citizens of New York State where a similar statute a few years ago was declared unconstitutional, and then repealed. . . .

The procedure provided by the Virginia statute is very similar to that of the repealed New York law.

The Supreme Court made short work of the attack upon the constitutionality of this statute, and in an opinion of great brevity but extreme clarity, Mr. Justice Holmes, the distinguished son of the distinguished physician, Oliver Wendell Holmes, outlined the reasons why the Virginia law should be upheld.

"The attack is not upon the procedure but upon the substantive law," declared Mr. Justice Holmes. "It seems to be contended that in no circumstances could such an order be justified. It certainly is contended that the order can not be justified upon the existing grounds. The judgment finds the facts that have been recited and that Carrie Buck 'is the probable potential parent of socially inadequate offspring, likewise afflicted, that she may be sexually sterilized without detriment to her general health and that her welfare and that of society will be promoted by her sterilization,' and thereupon makes the order. In view of the general declarations of the legislature and the specific findings of the Court obviously we cannot say as a matter of law that the grounds do not exist, and if they exist they justify the result. We have seen more than once that the public welfare may call upon the best citizens for their lives. It would be strange if it could not call upon those who already sap the strength of the state for these lesser sacrifices, often not felt to be such by those concerned, in order to prevent our being swamped with incompetence. It is better for all the world, if instead of waiting to execute degenerate offspring for crime, or to let them starve for their imbecility, society can prevent those who are manifestly unfit from continuing their kind. The principle that sustains compulsory vaccination is broad enough to cover cutting the fallopian tubes. . . . Three generations of imbeciles are enough."

LLOYD PAUL STRYKER, ESQ
(*NY State J Med* 1927; 27:792–795)

Secondary syphilis in a prepubertal child

Differentiating condylomata lata from condylomata acuminata

JOHN M. GOLDENRING, MD, MPH

Acquired perigenital syphilis in prepubertal children is unusual. When it occurs outside the newborn period, it is usually evidence of child sexual abuse.[1-3] Manifestations of secondary syphilis may also be confused with lesions of other sexually transmitted diseases. We describe a case of secondary syphilis in a ten-year-old boy, in which the initial lesion was originally thought to be condylomata acuminata.

CASE REPORT

A ten-year-old boy was brought to the emergency room because of a perirectal mass discovered approximately one week prior to admission. He was brought to a private physician who gave him a cream of unknown type, which did not decrease the size of the mass. The mass was somewhat painful, so the patient's mother brought him to the emergency room.

The perirectal lesion was noted to be raised and somewhat convoluted (cauliflower-like) in appearance with a broad base and mild keratinization, resulting in a venereal wart-like lesion (Fig 1). The pediatric emergency room physicians thought that the lesion was most likely condylomata acuminata (human papilloma virus infection). Because condylomata acuminata in the perirectal region is presumed to be a sexually transmitted disease, the child was admitted to the hospital under Child Protective Service Supervision, pending an investigation of probable sexual abuse.

The social history revealed that the child was a good student, though somewhat of a loner, living in a stable family situation with two siblings. He had no history of medical illnesses or current fever, weight loss, or trauma, and denied any previous skin lesions. Both the family and the child vehemently denied sexual abuse. Cultures for gonorrhea and Microtrak chlamydial slides were taken from the rectum and urethra, as well as a gonorrheal culture from the throat (and these were all later reported negative). A complete blood count and urinalysis were unremarkable. The inpatient attending staff thought that the lesion was somewhat atypical in appearance, but condylomata acuminata seemed the most likely diagnosis. Therefore, an excisional biopsy and papilloma virus typing of the biopsy specimen was scheduled.

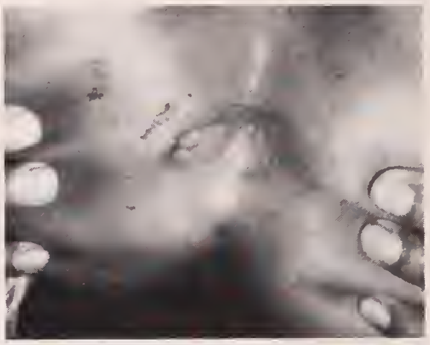

FIGURE 1. Raised, venereal wart-like lesion measuring 6 × 4 cm on admission.

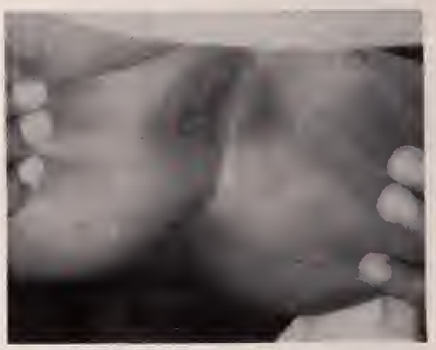

FIGURE 2. Lesion 48 hours after the administration of penicillin.

On the second day of admission, the VDRL, which had been taken as part of the sexual abuse work-up, was reported as positive. A treponemal microhemagglutination test (MHATP) specific for *Treponema pallidum* was also positive with a titer of 1:32. An attempted dark-field examination of the lesion was unsuccessful due to the insufficiency of the specimen.

Because of the positive MHATP, the patient was treated with 2 million units of benzathine penicillin intramuscularly, in addition to a 2-g oral dose of ampicillin, which had been given in the emergency room at the time of admission. Twenty-four hours after the penicillin, the lesion on the buttock which had previously been 6 × 4 cm (Fig 1), had involuted by two thirds. Within 48 hours, it had completely disappeared. A depigmented area remained in the perirectal region at the site of the original lesion (Fig 2).

The patient was presumed to have had condylomata latum, and was treated with 2 g of penicillin intramuscularly weekly for an additional two weeks as treatment for secondary syphilis.

Despite being asked several times, the patient still denied sexual abuse. He had visited Puerto Rico approximately five months prior to admission, the only time when he was away from parental supervision. His body language and eye contact during questioning about this period indicated to examiners that there was a high probability that he was hiding something that had happened during that period. The patient was released in the custody of his parents, to be followed with further VDRL titers over the subsequent months.

DISCUSSION

This case illustrates several important points. It is important when one sexually transmitted disease is found in a child to test for all possible others.[2] The work-up should always include testing for gonorrhea and chlamydia in vaginal and rectal areas, gonorrheal testing of the throat, wet mounts and Pap smears of the vagina in females, urinalysis, urine culture, and a screening test for syphilis. These studies provide collaborative information if further sexually transmitted disease is found, or if other sites of carriage are identified. As in this case, the failure to identify the second sexually transmit-

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ted disease, either symptomatic or asymptomatic, could have serious medical consequences. Though the yaws-like transmission of syphilis has been documented, genital lesions are clearly not likely to be transmitted by any means other than true sexual contact.[2]

Condylomata lata can look very much like condylomata acuminata when presenting in the perirectal area. Classically, condylomata lata are described as large, soft, flat-topped, coalescing papules which are painless and grayish-white in color. However, particularly in the perirectal region, the lesions may be raised well above the skin and convoluted in the manner of condylomata acuminata (see reference 4 for best picture). The rapid disappearance of lesions after the administration of benzathine penicillin was diagnostic, though it would have been useful to have obtained a positive dark-field examination.

If the lesion had been condylomata acuminata and not syphilitic in origin, the management of the case would have been the same except for the administration of penicillin. Perirectal condylomata acuminata constitute a sexually transmitted disease[2,5–7] unless the lesions are atypical in the pattern of presentation (ie, not truly in the perirectal region, but rather on buttocks or in buttock creases).[5–8] In order to be certain that the lesions are condylomata acuminata and to establish the medical/legal evidence, it is suggested that all perirectal and perivaginal condylomata acuminata or penile condylomata acuminata in children be at least partly excised, with the biopsy specimen being sent both for pathologic identification and for wart viral subtyping.[5,7] Lesions of condylomata acuminata are caused only by specific wart virus subtypes (primarily nos. 6, 11, 18).[5–7] The finding of these wart virus subtypes in true perirectal presentation confirms the diagnosis of sexual transmission and child sexual abuse. Of course, it is quite possible to have both syphilis, either primary or secondary, and condylomata acuminata in combination. If the lesion in this case had not disappeared with the administration of penicillin, the child would have been treated for syphilis, and a biopsy of the lesion would still have been performed.

Despite medical evidence of secondary syphilis and the fact that this had to be a sexually transmitted disease, the patient steadfastly refused to admit to an abusive sexual encounter, though interviewing revealed a probable geographic location for such an episode. This is in keeping with the finding that young males are very unlikely to reveal sexual abuse under any circumstances until at least middle adolescence.[9] Physicians must be very alert to the limitations of interview techniques in establishing child sexual abuse, especially in young males. Failure to elicit and deal with the problem may result in significant guilt feelings and other negative sequelae. Physicians should also be suspicious of unusual, nonvesicular cutaneous lesions in children and should consider drawing a VDRL for secondary syphilis work-up in the presence of such unusual rashes.[1]

Acknowledgment. The author thanks Nicholas Tsimbasis, MD, resident in pediatrics at Lincoln Hospital, who took the photographs for this paper; adolescent specialists Martin Anderson, MD, and Peter Mascella, MD; and pediatric dermatologist James Leach, MD; who all aided in the clinical care of the patient.

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See also pp 132–133.

"While we have time, let us do good"

Sometimes God Has a Kid's Face. By Bruce Ritter, OFM. 121 pp. New York, Covenant House, 1988. (paperback)

> No human being has a richer experience than the unlovely, awkward girl who in the course of a few months turns into a beautiful young maiden. It is both a glorious surprise and a fulfilled expectation.
>
> Isak Dinesen (Karen Blixen)[1]

And no human being can have so devastating an experience as the unloved, naive girl or boy who in the course of a few short months turns into a drug addict, a prostitute, a male hustler, a celluloid captive in a porno film, a panderer, a petty thief—or any combination of victim. In some ways that, too, is an expectation fulfilled, but it holds nothing glorious in it at all.

The saddest irony is that try as these youths might—degrade themselves, sell themselves, use, be used, do nearly anything on the streets of the richest nation in the history of mankind in order just to survive—most of them don't succeed. This is the truth Father Bruce Ritter, OFM, has learned as the founder of Covenant House, the most famous runaways' refuge in America. In the introduction to this short volume subtitled "Letters from Covenant House," Father Ritter says:

> In the almost 20 years of our existence we have tried our best to help the more than 100,000 kids we have taken off the streets into residence in our programs. We believe that we have been able to help about one-third make it back.
>
> The rest? I think most of them don't make it. They die young. . . . Kids don't survive very long on the street—at least in any recognizably human way. The distortion of the personality, the erosion of character are swift and massive and almost always irreversible.

Even the ones who make their way to Covenant House are not assured of longevity, Father Ritter writes. "Live Fast, Die Young, Leave a Beautiful Corpse," has been a motto of the devil-may-care set since at least the 1920s, but in Father Ritter's experience, only the first two phrases are true. The corpses these children leave behind are anything but beautiful.

Where do they come from, these street kids? Ultimately, says Father Ritter, they are the byproducts of child abuse. One of the stories Father Ritter tells in this book is about an 11-year-old girl and her two brothers, eight and nine years old. "Our parents beat us a lot," she said. "We had to leave." Another boy told his story: "I ran away when I was 14. My father and mother were alcoholics." Other stories are similar, stepparents who beat, ignored, mistreated; foster homes that gave no shelter; biological parents who disappeared; adults who used their size and strength and callousness to subdue kids until one day the children just could not accept the treatment one hour, one minute more, and ran away, seeking something, anything, better.

In Father Ritter's eyes what they're looking for is love.

> Kids come to Covenant House because they have no place to go. Most are in flight from deplorable living situations, hungry and very scared. Some are in flight from themselves. The problem is, for all of them, very simple: Where am I going to sleep tonight? Or eat tonight? Who will take care of me tonight?
>
> In a much deeper and totally unromantic sense the question is even more searing: Who will love me and care about me?

Father Ritter's personal answer—as evidenced by his actions since 1969 and by Covenant House—has been, "I will," but he takes no special credit for that. In describing his first contact with the homeless children of New York he says he took them in because "I didn't have the guts to kick them out and I couldn't find anyone else who wanted them. . . ." But his larger answer would be that we all need to take care of these children because "[Each] is yours and mine. Like it or not, he is part of us."

Father Ritter's volume telling the stories of these kids, these parts of us, recounts the history and mission of Covenant House. But underneath the day-by-day story of Covenant House and some of its boys and girls, successes and failures, is another point, another anger.

Father Ritter has his own point of view, a Christian outlook that seems in some ways out of step with modern America. He castigates Dr Ruth Westheimer, for example, as being the "high priestess of hedonism," and for fostering "practically unlimited sex anytime . . . as long as they use contraceptives." He calls this "insane drivel"—even though many sociologists would argue that if more couples knew the biological facts of life and used contraceptives then there would probably not be as many abused children to wind up at Father Ritter's doorstep.

But whether or not one agrees with every statement of Father Ritter (or of Dr Ruth, for that matter), it does not detract from the basic message. Which is, not only are these children all part of us, but we are very much a part of the system that exploits and kills them. It starts on an individual basis, with parents who don't care about their children, who beat, maltreat, denigrate, and abuse them, who drive these teenagers out into the streets where there are other predators waiting for them.

These predators are the sellers of drugs and the sellers—and buyers—of sex.

> When you're 14 and 15, and you can't read or write very well, and you have no place to live, and it's cold and you're hungry and you have no marketable skills, you market yourself. . . . This year we expect at least another 10,000 [Note: This figure was accurate in the late 1970s when this sentence was originally written; in 1989 the figure would read 26,000] beautiful kids to come through our doors. Most of them will have been involved, on some level, with the [sex] industry. Most of them call it making a few bucks. I have never met a young prostitute, girl or boy—and I've met hundreds—who. . .did not start out as a runaway.

The word on the street is, says Father Ritter, that johns prefer chickens—that is, kids.

> Hundreds of thousands of New Yorkers and out-of-town visitors patronize the sex industry, and make a lot of very unwholesome people very rich. There's no mystery why we have a sex industry. The reasons have always been the same: greed and lust. And our inability to care enough about what happens to the young people who, every year, become enslaved by the industry. . . .

Sex has become a kind of entertainment, he argues, which is why it's no surprise that the sex industry in Times Square is contiguous with the entertainment industry of Broadway. He writes, "The sex business, dominated and controlled by organized crime, has actually formed a continuum with the entertainment industry. . . . And we seem to want it that way." Father Ritter poses the question, What is the ethical or moral or qualitative difference between spending money to see "Oh, Calcutta!" or a porno flick, and he answers that there is no difference at all.

Neil Postman, author of *The Death of Childhood*, social thinker and follower of Marshall McLuhan, points to this as a larger social issue. He hypothesizes that "[American] politics, religion, news, athletics, education and commerce have been transformed into congenial adjuncts of show business, largely without protest or even much popular notice. The result is that we are a people on the verge of amusing ourselves to death."[2]

As an industry, this particular form of amusing ourselves displays all the excesses of America's other industries. It is profit-oriented and resource-wasteful. It preys upon man's lower nature, manipulating both buyers and sellers, and cowing the general populace into accepting its very existence. It chews up raw materials and then discards the leftovers when they no longer make profits.

And the raw material, says Father Ritter, are the children—mostly runaways, but some unwilling captives as well—that end up on his doorstep, or dead.

> It doesn't take long to murder a child, and there are lots of ways to do it. . . . There's another kind of death my kids experience. . . . Three months on the street is a very long time. Six months is forever. A year? Then they're just breathing in and out but dead inside. The poison works very quickly. . . .
>
> In the beginning they can still make distinctions between what they are inside their heads and what they do with their bodies. But after 100 or 500 or 1,000 johns it becomes difficult, and then impossible to separate. . . . Somewhere along the process a child dies. He's been murdered.

This is a heinous crime, he says, and we do little or nothing about it, because of "greed and lust, and our sloth and fear. Who, after all, wants to take on organized crime? Our politicians certainly don't. Our law enforcement groups can give you a thousand reasons why they can't, either. Our prosecutors and our judiciary tell us they have their hands tied. And everybody has a finger to point at the other guy."

Father Ritter has many such stories to tell, stories with enough sorrow and terror to explain why he is sometimes bitter at civil libertarians who defend First Amendment rights, or uphold the criminal's rights in the judicial system, or even people like Dr Ruth Westheimer. But as you read his book, you can begin to wonder about Father Ritter's stance. He is there, in the trenches, fighting sin everyday: "Sin, in the concrete on Eighth Avenue and 42nd Street, is sin in the X-rated lives of children, in their X-rated eyes and X-rated faces and their X-rated bodies. The kids are sinners. We are too," writes Father Ritter. But, at least in *Sometimes God Has a Kid's Face*, Father Ritter seems too cynical to entrust society with cleaning up these sinners.

God, he says again and again, will care for these children. When faced with financial problems at Covenant House he tells us, "That's God's problem." When a certain runaway returns time and again to Covenant House only to be unable to live within the rules, Father Ritter wonders, "Please God, can I—should I—write [him] off and leave [him] to Your mercy?"

And yet, what is that mercy? "Only God," writes Father Ritter, "can help the burned-out, used-up kids. . . . What he usually does is help them die." That seems a cold way to view these dying children. It makes the statement of one whom Father Ritter could not hold on to understandable, in a way. "Your God is okay, I guess," he said to Father Ritter, "But He's sure not part of anything I've been into." Isn't that a cry from the heart that can be understood? It's a plea to do something to stop all this, and an understanding that kindness and charity are not in and of themselves enough.

This is perhaps an unfair criticism. Father Ritter's book does not profess to be a comprehensive plan for solving the problems of child abuse, runaways, lack of adequate public shelters, prostitution, lust, and greed. In fact, *Sometimes God Has a Kid's Face* isn't really a book at all.

It is a chronological collection of Father Ritter's newsletters from Covenant House, and the frank purpose of these letters is to raise funds to keep the several centers now in existence going, and to open new refuges around the United States as needed. The stories he tells are scarifying enough, terrible enough, to accomplish this purpose. Perhaps that is more than enough (and, truthfully, running Covenant House is far nobler and more useful than writing book reviews).

But listen carefully to a story like the one Father Ritter tells about "Mark" who hitchhiked 2,000 miles to tell Father Ritter that he worked as a male prostitute for a Fortune 500 corporation. "I had a company car and an apartment and took care of corporation clients. They would fly me all over the country. The corporation had a representative that would take a portfolio of the kids in their stable, both boys and girls, to their clients. We didn't have any clothes on in the photographs. The clients could pick anyone they wanted. . . ." Mark was pretty popular, but he wanted to quit. "I'm afraid," he told Father Ritter. "They don't like you to leave them." Father Ritter told Mark to stay with them at Covenant House, and he would be helped; but the next morning Mark was gone.

This story raises many questions, not the least of which is, can it be true? Father Ritter has evidence here of a major American corporation keeping a stable of child prostitutes, pimping for them, threatening them. What has he done with the evidence? What happened to Mark? Did this Fortune 500 firm get him back—perhaps right out of Covenant House?—and punish him? Why hasn't this corporation been exposed in the public media?

Reading this kind of story makes one wish that Father Ritter had envisioned a larger purpose in publishing his notes from Covenant House, had had more of the Upton Sinclair muckraker in him, and less Mother Theresa. But this is a small criticism laid next to the enormous gratitude owed to Father Ritter just for being there, for caring enough, for having the guts to not send those first half-dozen refugee kids away.

In his conclusion Father Ritter says, "Kids should not be bought and sold. They should not be exploited. It should not be infinitely dangerous to be alone and homeless on the streets of our cities." He stresses that there must be "a place where they can all get help. And [get help] when they need it: before it's too late." This is the mission of Covenant House; and it should be the mission of us all.

"Dum tempus habemus, operemur bonum!" (While we have time, let us do good) was the final injunction of St Francis. Father Ritter, a Franciscan, offers it as a motto for us all. In an epilogue to *Sometimes God Has a Kid's Face* he makes suggestions for turning those words into action. Suggestion number six is well worth reprinting here:

> Pick out some aspect of the problem of exploited kids and do something about it. Join or start a group. Five people working together can accomplish more than 100 individuals working alone. Become politically streetwise. There are no great mysteries about it: politicians respond to appropriate, persevering pressure. There are laws that need changing and laws that need passing. . . .

Yes, while we have time, let us do good—but let us start right away, for the children's hours are passing quickly.

BRIAN QUINN
Medical Society of the State of New York

[Sometimes God Has a Kid's Face *can be obtained from Covenant House, 460 W 41st St, New York, NY 10036. A $3 donation is requested.*]

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Child sexual abuse

Child Sexual Abuse: A Handbook for Health Care and Legal Professionals. By Diane H. Schetky, MD, and Arthur H. Green, MD. 264 pp. New York, Brunner/Mazel Publishers, 1988. $27.50 (hardcover)

In this book the authors—two child psychiatrists and three other contributing professionals—discuss the issues and pathology of child sexual abuse in a most appropriate developmental context. This is a particular strength of this text. They also give an excellent overview of normal psychosocial development—something that should be taught to everyone—and provide a historical perspective of the problem and discussion of the relevant literature available. The book is divided into four sections: "Overview," "Evaluation," "Legal Issues," and "Treatment and Prevention." I found the chapters on the clinical and physical evaluations particularly helpful as a guide to assessment of individual patients, although this is not the major focus of the book and similar information can be found in newer pediatric and medical text and manuals. This is, however, an excellent guide for a sensitive and complete workup which is mandatory in individual patients.

The legal issues are discussed, including laws and background with respect to child pornography and prostitution. Of particular interest to the physician is the child as witness and the physician as expert witness. These sections will be particularly helpful to physicians and other health care workers who become involved in such cases, although they may be less comprehensive for legal professionals than for medical ones.

The chapter discussing true and false allegations of child sexual abuse is especially worthwhile. It is an excellent preface for the novice health professional who may be asked to make difficult judgments as to whether or not a child has suffered sexual abuse. The reader will be better able to deal with situations in clinical practice after having read this book.

The section on treatment of sex abuse offenders offers an excellent scientific and empathetic approach to these disturbed individuals. The appendix provides good resources for clinical aids to diagnosis and other appropriate educational materials.

Child abuse has been an increasingly recognized problem in the United States for the past several decades, whereas child sexual abuse seems to be a more recently recognized phenomenon, with new sensationalism in the press as each tragic case is uncovered. As recently as 1975 a major pediatric textbook, *Nelson's Textbook of Pediatrics* (ed 10, Vaughan and McKay, eds, Philadelphia, W.B. Saunders), did not mention sexual abuse in its index as a separate topic but rather listed it as a subheading under child abuse with a one-page reference. This is a seven-line paragraph which indicates that sexual exploitation of children is probably the most underdiagnosed type of child abuse. Similarly, *incest* did not appear in the index of the 1975 edition but is widely discussed in the most recent 1987 edition (ed 13). To be sure, this reflects a relatively uneducated profession in the 1970s and a greater appreciation of this problem in recent years. Although incest and its taboo are well recognized, the wider pathology of sexual abuse has been less well identified and studied.

The lay press now abounds with daily headlines that somehow continue at the same time to shock and anger us, but leave us with a sense of frustration at our inabilities to deal with this problem. One may justifiably ask, Is this a medical problem in its classic sense or such an overwhelming societal or social problem that we as physicians are unable to manage it? Like other newer morbidities of childhood and adolescence—eg, drug abuse, violence, suicide, homicide, accidents, eating disorders, etc—we are confronted not with a disease confined to or born of poverty and destruction, but a disease of the middle and affluent classes as well. To wit: The tragedy of Lisa Steinberg which has glared in the lay press for months.

Child abuse and sexual abuse are also seen with recurrent frequency in poor populations as well. In a traditional well child clinic of an adoption agency in New York City, which over the years has largely become a foster care agency, not a week goes by that we do not see victims of childhood sexual and other forms of abuse. Every practitioner faces such patients in his/her practice. There is an urgent need for physicians to become more conversant with the etiology and treatment of this disease. Clearly the treatment rests with physicians and other professionals. The immediate prevention of repetitious behavior by the perpetrators of this disease and the need to halt the inheritance of this legacy in the next generation may require a broader community and political coalition to rid our society of this affliction. Although it is well known that victims of child abuse are more prone to be abusers of their own children, only about one third of current child abusers were abused as children themselves. For this high risk group preventive interventions and strategies should be identified. Providing parents and children with help in enhancing self-esteem may be the single most important preventive intervention. Typically abusers and those abused have poor self-worth and self-image. Classically this is seen in the issue of teenage pregnancy. Teenage pregnancy programs are good places for adolescents to work towards developing parenting skills and behaviors that may prevent the cycle of both repeated pregnancies and the abuse of the children born. Evidence clearly suggests that teenage parents are at a high risk for abusing their children. Other high risk populations should be identified and interventions sought. Mental health clinics, the courts, and drug abuse treatment and prevention programs are some examples of places where potential abusers may be found. Knowledge of child abuse and sexual abuse should be made part of health education curricula in the school systems as well.

Child Sexual Abuse: A Handbook for Health Care and Legal Professionals is concise, very readable, and well indexed so that it becomes a handy reference on the subject for the physician's library. It should be helpful to family physicians, pediatricians, psychiatrists, and other mental health professionals interested in optimizing the physical and mental well being of children who are victims of sexual abuse. Its relatively modest price should also encourage a wide readership.

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Cesarean childbirth in New York State

The rate for cesarean births in the United States has risen dramatically since 1970. In 1985, the rate reached 22.7% of all births, representing the highest level ever observed for the delivery of newborns.[1] In contrast, the cesarean delivery rate during the 1950s was approximately 4% and rose only slightly to 6% by 1970.[1,2] The increase was first observed during 1970–1978 when the cesarean rate rose to 15.2%.[2] Figure 1 shows a similar pattern for New York State. The rate for total cesareans in 1970 was 6.6% and by 1986 it had risen to 24%. The rise in cesarean childbirth is closely tied to the decrease in the use of other operative procedures (eg, breech, forceps, version and extraction, etc). These procedures steadily declined during 1970–1986, from 33% to 6%, while cesareans steadily increased.

Studies suggest that dystocia, fetal distress, breech and other malpresentation were possible factors for the increase in cesarean delivery rates. Another important factor was the larger number of women eligible for a repeat cesarean, a direct result of the increase in the primary cesarean rate. In the 1980s, previous cesarean delivery was cited as the most important contributing factor to the rise.[3] In the state, the percentage of women who had a previous cesarean and a subsequent noncesarean delivery during 1984–1986 was only 7%. This indicates that cesarean deliveries will continue to increase as women who had a primary cesarean in the mid-1980s approach their next pregnancy. The projected cesarean delivery rate in the United States for 1990 is 28.8%; for the year 2000 it is expected to be 40.3% if present trends continue.[4]

The following analysis examines various maternal and fetal outcomes during 1984–1986 in New York State. The overall cesarean rate for this time was 22.8%; 14.4% primary and 8.4% repeat cesarean deliveries.

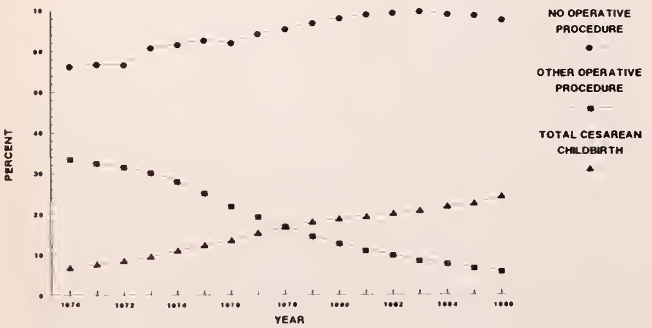

FIGURE 1. Percentage of live births by method of delivery, New York State, 1970–1986. "Other operative procedure" includes breech, forceps, version and extraction, etc.

Maternal Characteristics

The cesarean delivery rate was found to increase with increasing maternal age. Mothers aged 10–17 had the lowest rate (14.7%); mothers aged 40 or more had the highest (33.4%). The increase in the overall rate with increasing maternal age was associated with repeat cesarean sections. The primary delivery rate remained fairly constant for women 10 to 34, but repeat cesareans were increasingly performed in relation to the mother's age.

The cesarean rate increased with the education level of the mother, except for the group with the lowest level of education (1–8 years). The lowest rate (19.1%) occurred in mothers with 9–11 years of education. Mothers with 16 or more years of education had the highest rate of cesarean deliveries at 24.9%.

Subtle differences were observed for the four ethnic groups examined: whites, blacks, non-Hispanic others (nonwhite, nonblack) and Hispanics. Blacks were shown to have a slightly lower overall cesarean delivery rate (20.7%), while whites had the highest rate at 23.6%.

Women with very low birthweight babies (less than 1,500 grams) had the highest rate for cesarean deliveries at 40.7%. Women with high birthweight babies (4,500+ grams) and low birthweight babies (1,500–2,499 grams) had rates of 36.9% and 31.3% respectively. These figures compared to the cesarean rate for normal birthweight infants (2,500–4,499 grams) at 21.9%.

Complications of pregnancy and labor were analyzed through birth certificate data. Women who had complications of pregnancy had an overall cesarean delivery rate of 41.1% compared to 21.2% for women having no complications. The cesarean rates associated with complications of pregnancy varied from 28.7% for abnormal uterine bleeding in the first trimester to 68.7% among women with genital herpes affecting the pregnancy. The cesarean rate for women having complications of labor was 54%, compared to 9.6% for women with no complications. The cesarean rate associated with cephalopelvic disproportion (CPD) was 97.3%, while the rate associated with retained placenta was 8.0%.

Maternal/Fetal Outcomes

Outcomes associated with infant health status related to the birth event include Apgar score and infant and neonatal mortality. Length of hospital stay is used to infer maternal morbidity.

For cesarean deliveries, only 10.2% of hospital stays were fewer than five days as compared to noncesarean deliveries at 91.5%. Of all cesarean deliveries, 32.5% had a hospital stay of one week or longer. Only 2.6% of noncesarean deliveries indicated such a prolonged stay.

Apgar score is a composite score calculated at one and five minute intervals after birth. It is based on observed heart rate, respiratory effort, reflex irritability, muscle tone and skin color. Low scores are associated with neonatal morbidity, low birthweight and neurological abnormalities at one year of age.[5] Regardless of delivery method, the majority of infants had high one minute Apgar scores. The highest percentage of infants with scores between 7 and 10 were in the spontaneous delivery and repeat cesarean delivery groups (93.7% and 91.6% respectively), while primary cesarean delivery births had a much lower percentage of high scores (83.7%). At the five minute interval, little difference existed between the method of delivery groups in the distribution of Apgar scores. Infants delivered by repeat cesarean and spontaneous delivery again had the highest scores, 98.0% and 97.9% respectively. Infants delivered by primary cesarean had only a slightly lower percentage of 96.0%. Births associated with all other operative delivery procedures were at the intermediate level, with 97.0% having high scores.

Infant mortality is defined as death during the first year of life. The highest infant mortality rate during 1984-1985 was observed among primary cesarean births at 15.2 per 1,000, while repeat cesarean births had the lowest (eight per 1,000). Births delivered by other operative procedures had an infant death rate of 13.0 per 1,000. The rate for births delivered with no operative procedure was 9.8 per 1,000. When birthweight was considered, there were significant changes in the infant mortality rates. Infants in the at-risk group (500-2,499, 4,500+ grams) had a higher mortality if delivered by operative procedures other than cesarean at 93.2 per 1,000. Repeat cesarean deliveries had an infant death rate of 47.2 per 1,000. Infants in the no-risk group (2,500-4,499 grams) showed little difference in infant mortality among the various delivery groups. The death rates ranged from 3.4 per 1,000 for operative procedures other than cesarean to 5.7 per 1,000 for primary cesarean deliveries.

Neonatal mortality refers to deaths that occur less than 28 days after birth. These deaths account for approximately 70% of all infant deaths. The highest rate was observed for primary cesarean births at 11.1 per 1,000. Repeat cesareans had the lowest infant mortality rate at 4.5 per 1,000. Among at-risk live births, operative procedures other than cesarean had the highest neonatal death rate at 81.7 per 1,000. Primary cesarean births had a rate of 49 per 1,000.

This article was adapted from *Cesarean Childbirth in New York State: A Statistical Profile*, published by the Information Systems and Health Statistics Group. A copy of the report is available from: Bureau of Information Science and Policy, New York State Department of Health, Empire State Plaza, Corning Tower, Room 308, Albany, NY 12237.

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Prevention of perinatal transmission of hepatitis B

The New York State Department of Health recently recommended (Health Series Memorandum 88-81) that all pregnant women be tested for evidence of hepatitis B infection. This practice will allow newborn infants at risk for acquiring infection in the perinatal period to be identified and treated. Treatment of exposed infants prevents the development of chronic hepatitis B infection.

Hepatitis B is transmitted by contact with infected blood or body fluids. One of the important modes of transmission of hepatitis B is from chronically infected women to their newborn children at the time of birth (perinatal transmission). Approximately half of the infants exposed to the virus at birth will become infected, but the infection rate can be as high as 70-90% if the mother is hepatitis B core antigen positive.[1,2] Among infants infected at birth, 85-90% will become chronic hepatitis B carriers. The chronic hepatitis B carrier state can persist throughout life. The carrier can transmit infection to others and has a risk of approximately 25% premature mortality from cirrhosis, chronic active hepatitis or primary liver cancer.[3]

Perinatal transmission of hepatitis B can be prevented in 85-95% of cases with a series of immunizations beginning as soon after birth as possible.[2] Hepatitis B vaccine and hepatitis B immune globulin should be given to infants of infected mothers within 12 hours of birth. Additional doses of hepatitis B vaccine are given at one and six months of age. The timing of the first treatment is critical, as the efficacy of the treatment falls off markedly after 48 hours of life.

Previous recommendations for prevention of perinatal hepatitis B transmission focused on testing of high-risk women. However, several recent reports published in the medical literature indicate that this may result in failure to identify 50% of infected women whose infants are in need of treatment.[4] Additionally, universal testing of pregnant women for hepatitis B has been suggested to be a cost-effective strategy.[5]

The rate of pregnant women carrying hepatitis B in New York State is estimated to be 1%. Therefore, approximately 2,500 infants each year are at risk of contracting hepatitis B infection from their mothers at the time of birth. The New York State Department of Health has issued the following specific recommendations which have been endorsed by the New York State Medical Society and the state chapters of the American College of Obstetrics and Gynecology and the American Academy of Pediatrics.[6] These recommendations are consistent with recent national guidelines on perinatal hepatitis B prevention.[4]

- All pregnant women should be tested for hepatitis B surface antigen (HB_sAG) during the first prenatal visit to their physician. This testing should be done at the same time as other routine prenatal screening tests (eg, syphilis serology). The test should be repeated late in pregnancy if acute hepatitis is suspected, if there is a history of hepatitis exposure, or if the women engages in high risk activities. These high risk activities include intravenous drug use, having multiple sexual contacts and frequent occupational exposure to blood.

- A pregnant woman's HB$_s$AG status must be known at the time of hospital admission for delivery. Results from prenatal testing for HB$_s$AG should be recorded prominently in the mother's medical chart. If testing has not been performed or if the test results are not available, HB$_s$AG testing should be done as soon as possible after admission. Hospitals where infants are delivered should make test results available within 24 hours.
- All infants born to HB$_s$AG-positive mothers should receive hepatitis B immune globulin (HBIG 0.5ml) intrasmuscularly (IM) when they are physiologically stable, preferably within 12 hours of birth. The efficacy of HBIG decreases significantly if it is given more than 48 hours after birth. These infants should also receive three doses of hepatitis B vaccine. The first dose (0.5ml IM) vaccine should be given concurrently with HBIG (at a different site) or within seven days of birth. Subsequent doses should be given one and six months after the first dose. Hepatitis B immunization will not interfere with routine childhood immunizations. Infants so treated should be tested for HB$_s$AG and its antibody (anti-HB$_s$) at 12–15 months of age to determine the effectiveness of the treatment. In the unlikely event that the infant has not responded to immunization or has not developed hepatitis B infection, additional doses of hepatitis B vaccine may be considered.
- Measures must be taken to ensure that test results are communicated to the physician caring for the newborn children so that medical follow-up can be carried out.
- Household members and sexual contacts of women identified as hepatitis B carriers during the prenatal screening should be tested for susceptibility to hepatitis B infection. Those contacts found susceptible should be immunized with hepatitis B vaccine (three doses at 0, 1 and 6 months).

The New York State Department of Health is developing mechanisms to help ensure effective follow-up of infants of HB$_s$AG-positive mothers. Limited amounts of hepatitis B vaccine will be available for follow-up doses at local health departments. The department also plans to conduct educational activities to increase public and professional awareness of the importance of prenatal hepatitis B screening and of providing hepatitis B vaccine to infants of HB$_s$AG-positive mothers.

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AIDS among New York State children

The number of acquired immune deficiency syndrome (AIDS) cases among children in New York State has grown rapidly since 1985. By September 30, 1988, the New York State Department of Health had received 378 reports of AIDS cases in New York State residents less than 13 years of age. Figure 1 describes these pediatric cases by year of diagnosis. Sixty-two percent of these cases have been diagnosed since the beginning of 1986. (Note: there was incomplete information for one of these cases, and it was not included in the analysis.) Table 1 describes the age, sex and race/ethnicity of these pediatric AIDS cases. Forty-eight percent were diagnosed prior to age one, and nearly 90% were diagnosed by age four. However, it is likely that many children who were infected at birth have not yet developed AIDS because of a prolonged incubation period. As these children develop AIDS at older ages, the age distribution of cases may change. There is no gender predominance in the frequency of diagnosed cases. Minorities are disproportionately affected, with over 90% of the pediatric AIDS cases occurring among nonwhites. Table II describes the most predominant opportunistic diseases diagnosed among children with AIDS in the state. *Pneumocystis carinii* pneumonia (PCP) and lymphoid interstitial pneumonia (LIP) are the most common diagnoses. Ninety percent of all pediatric AIDS cases have been diagnosed with either PCP or LIP. The major risk factor for AIDS among children in New York State is maternal transmission associated with intravenous (IV) drug use (Table II). A total of 291 (77%) pediatric AIDS cases can be linked to IV drug users (IVDUs); 63% of children with AIDS were born to mothers who used IV drugs, and an additional 14% had mothers whose sexual partners used IV drugs. Of the children whose mothers were IVDUs, 54% were black, 37% were Hispanic and 8% were white (data not shown).

Table III describes the pediatric AIDS cases by county

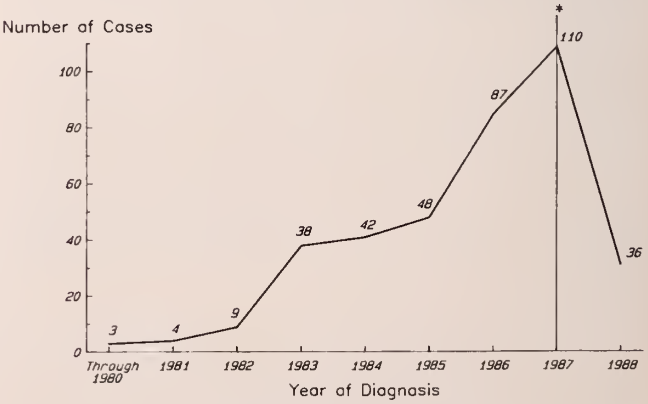

FIGURE 1. New York State pediatric AIDS cases by year of diagnosis; reporting period ending September 30, 1988. Data for the last 12 months are incomplete (Source: AIDS Epidemiology Program, Division of Epidemiology, NYSDOH).

TABLE I. New York State Pediatric AIDS Cases by Age, Sex, and Race (Data through September 30, 1988)

Age (years)	New York City No. (%)	Rest of State No. (%)	Total No. (%)
<1	169 (50.0)	12 (30.8)	181 (48.0)
1–4	137 (40.5)	19 (48.7)	156 (41.4)
5–9	31 (9.2)	7 (18.0)	38 (10.1)
10–12	1 (0.3)	1 (2.6)	2 (0.5)
Sex			
Male	169 (50.0)	19 (49.0)	188 (49.9)
Female	169 (50.0)	20 (51.0)	189 (50.1)
Race			
White	23 (6.8)	12 (31.0)	35 (9.3)
Black	197 (58.3)	20 (51.3)	217 (57.6)
Hispanic	117 (34.6)	7 (18.0)	124 (32.9)
Other/unknown	1 (0.3)	0 (0.0)	1 (0.3)

TABLE III. New York State Pediatric AIDS Cases by County of Residence at Onset of Illness (Data through September 30, 1988)

	Number of Cases	Percent of Cases
New York City	338	89.7
Bronx	98	26.1
Kings	105	28.0
New York	82	21.7
Queens	43	11.5
Richmond	8	2.1
Unknown NYC Residents	2	0.1
Rest of State	39	10.3
Dutchess	1	0.3
Nassau	6	1.6
Onondaga	3	0.8
Orange	1	0.3
Putnam	1	0.3
Rensselaer	1	0.3
Rockland	3	0.8
Suffolk	16	4.3
Sullivan	1	0.3
Wayne	1	0.3
Westchester	5	1.3
Total	377	100.0

of residence at the time of diagnosis. New York City accounts for 338 (90%) of the pediatric cases in the state. This is similar to the proportion of adult cases from New York City (85%). Within the city, the Bronx, Brooklyn, and Manhattan account for 285 (84%) of the New York City cases. Thirty-nine (10.3%) of the pediatric AIDS cases in the state resided outside New York City. Nassau, Suffolk and Westchester counties account for 27 (69%) of these cases.

Cumulative mortality among pediatric AIDS cases is high (Table II). Sixty-five percent of children with AIDS have died, compared with a 52% mortality rate for adult AIDS cases in New York State. Among children, mortality is inversely related to age. Seventy-nine percent of children who were diagnosed before age one have died, whereas 55% of children diagnosed between age one and four have died. Mortality is similar among the different racial/ethnic groups: 71% for whites, 62% for blacks and 67% for Hispanics.

AIDS among children is expected to continue due to the prevalence of human immunodeficiency virus (HIV) infection among IVDUs. It is likely that minorities will continue to bear a disproportionate burden of pediatric AIDS cases. A major share of the Department's prevention efforts are directed to women of childbearing age and to inner-city populations.

TABLE II. New York State Pediatric AIDS Cases by Selected Opportunistic Disease, Risk, and Vital Status (Data through September 30, 1988)

	New York City No. (%)	Rest of State No. (%)	Total No. (%)
Disease*			
Pneumocystis carinii pneumonia (PCP)	168 (49.7)	14 (35.9)	182 (48.3)
Lymphoid interstitial pneumonia (LIP)	158 (46.8)	14 (35.9)	172 (45.6)
Bacterial infections	70 (20.7)	7 (18.0)	77 (20.4)
Mycobacterium avium complex or M Kanasii	36 (10.7)	5 (12.8)	41 (10.9)
Esophageal candidiasis	34 (10.1)	6 (15.4)	40 (10.6)
Cytomegalovirus	30 (8.9)	7 (18.0)	37 (9.8)
Risk			
Mother, IVDU	213 (63.0)	24 (61.5)	237 (62.9)
Mother, sex w/IVDU	51 (15.1)	3 (7.7)	54 (14.3)
Mother, born in Car-Afr or sex w/person born in Car-Afr	22 (6.5)	2 (5.1)	24 (6.4)
Mother, HIV+/AIDS or sex w/person HIV+/AIDS	16 (4.7)	2 (5.1)	18 (4.8)
Blood products	11 (3.3)	5 (12.8)	16 (4.2)
Other/undetermined	25 (7.4)	3 (7.7)	28 (7.4)
Vital Status			
Dead	218 (64.5)	25 (64.1)	243 (64.5)
Alive	120 (35.5)	14 (35.9)	134 (35.5)

* For disease category, numbers indicate total frequency of opportunistic diseases diagnosed. The percent column reflects the percent of AIDS cases with a given opportunistic disease.

Ipecac poisoning: Know the symptoms that suggest child abuse

For accidental poisoning in children, the home remedy of choice is commonly ipecac, a safe drug for inducing emesis. However, chronic administration of ipecac is associated with severe adverse effects on the gastrointestinal, neuromuscular, and cardiovascular systems, and is an easily overlooked form of child abuse.

McClung et al (*Am J Dis Child* 1988; 14:637–639) reported on three children, ages 8, 10, and 21 months, who were victims of intentional ipecac poisoning. The children were brought for treatment of recurrent diarrhea and vomiting. Following extensive diagnostic procedures and therapeutic approaches, suspicions of ipecac poisoning led to screening tests for ipecac and confirmation of ipecac poisoning. Separation of the children from their mothers led to immediate cessation of symptoms.

Emetine, the most active ingredient of ipecac, can remain in the body for as long as 60 days following systemic administration. "Toxicity after systemic absorption of emetine is cumulative . . . .The cardiac manifestations are the most dangerous. They include initial tachycardia followed by flattened and inverted T waves and a prolonged QT interval on the electrocardiogram, which can be associated with fulminant cardiomyopathy and death from cardiac failure."

The authors presented the common features of these patients that suggest intentional poisoning: parents were in general uncooperative and critical of the treatment of their child; children were under two years of age and appeared well nourished; the pattern of vomiting was not suggestive of an infectious, anatomic, or metabolic cause; vomiting continued throughout the hospital stay, yet subsided on separation of the child from the mother.

Many standard laboratory evaluations do not test for ipecac, and, because the drug is accepted as a treatment for accidental ingestion of poison, the potential for acute and chronic abuse of ipecac may be overlooked. The authors concluded with the recommendation that "toxicology laboratories utilize methods that include ipecac in their screens and report its presence in all samples and that physicians who suspect chronic vomiting from factitious causes request urine screening specifically for ipecac."

Clinical picture of child abuse is changing

Increased awareness of child abuse during the last decade has contributed to earlier detection of abuse, and more openness in discussing and recognizing sexual abuse. Consequently, more younger children are being brought for treatment of abuse, and the physical injuries seen are often less severe than the multiple fractures, malnutrition, and violent sexual abuse that was more common several years ago.

Based on their experience with abused and neglected children from 1984 to 1986 in an outpatient clinic, Marshall, Puls, and Davidson summarized the changing spectrum of child abuse: "more children are being brought for evaluation, sexual abuse is the most frequent complaint and most often involves preschoolers, and more clinically subtle abuse is being seen" (*Am J Dis Child* 1988; 142:664–667).

Of the 382 abused and neglected children who comprised this study, 64% were girls. Furthermore, 84% of the sexually abused children were girls. Physical abuse was the more common finding among boys. Half of the victims of sexual abuse were four years of age or younger, as were the majority of children found to have suffered physical abuse and neglect.

Child abuse is much less likely to be covered up or ignored in today's society. Therefore, victims of abuse are, in general, being identified at an earlier stage of long term abuse. Verbal and nonverbal clues that indicate sexual abuse are playing an increasingly important role in evaluating young children. Although most of the children who were evaluated for sexual abuse "had a definite history of abuse," 70% of these patients exhibited no abnormal physical or laboratory findings. The authors stressed that the lack of physical findings in no way rules out abuse. Only five of the 382 children evaluated had falsely claimed to be victims of abuse. Despite the absence of physical damage or disease, victims of sexual abuse must still contend with the emotional consequences of their experiences.

"The new spectrum of child abuse requires less acute medical intervention; . . . alternatives to hospitalization are now available and usually more desirable." In fact, the growing number of child abuse cases and rising health care costs have led to an increased reliance on outpatient care facilities for diagnosis and treatment of the victims of child abuse.

Health care practitioners face malpractice charges for failure to report child abuse

In all but six states, health care professionals can face criminal prosecution for not reporting suspicions of child abuse (*Dental Management* 1988; 28:32). Recent court rulings have now opened the door for malpractice liability charges against dentists in such cases. The dentists would then be "liable for any further harm to his patient whom he suspected may have been a victim of child abuse."

In 1976, a California court charged an emergency room physician with malpractice for failing to file a child abuse report on an 11-month-old patient with a broken leg. The patient was subsequently treated for extensive injuries, and the second physician's report of possible child abuse was substantiated. In 1984, an Illinois jury found a physician guilty of malpractice for not having diagnosed and reported a case of child abuse. Most recently, in 1987, a woman sued her father's psychiatrist for not reporting that his patient was physically and mentally abusing the woman. Although the court dismissed the charges, stating that the psychiatrist had not acted improperly toward the woman, because she was not his patient, a Florida District Court of Appeals upheld the charges. The appeals court based its ruling on Florida's mandatory child abuse re-

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NEWS BRIEFS are compiled and written by Vicki Glaser.

porting statute, which "provides a civil cause of action predicated on violation of its provisions, subject to proof that the violation gave rise to the injury sustained by a member of the protected class."

Training pediatric residents to recognize and manage child abuse

Health practitioners who specialize in the care of children identify approximately 12% of all reported cases of child abuse. As the incidence of child abuse increases, pediatric health care professionals must intensify their efforts to diagnose, treat, and report child maltreatment. Howard Dubowitz, MD, from the University of Maryland Medical School, has stressed the importance of teaching programs and adequate clinical resources in improving the competency of pediatric residents to manage cases of child abuse (*Pediatrics* 1988; 82:477–480).

The author distributed questionnaires to every pediatric residency program in the United States. He requested information on specific features of the hospital and residency training program, the frequency and types of child abuse reports prepared by the hospital, the amount and content of resident training in child abuse, the existence of an interdisciplinary child abuse team, and the respondents' satisfaction with their teaching program concerning child abuse.

Based on data from the 72% of questionnaires that were returned, the author determined that the median amount of training in child abuse was eight hours in the first and third years of the residency programs and seven hours in the second year. This training was judged to be "better than adequate in 27% of programs, adequate in 42%, and less than adequate in 30%." An interdisciplinary team formed to handle cases of suspected child abuse was a component of 81% of the programs. The majority of these teams included a social worker, pediatrician, and a nurse, while some had a child psychiatrist or psychologist and a lawyer.

Dubowitz concluded that "although there are many competing areas for time in residency programs, it appears that there is insufficient training in the area of child maltreatment." He recommended additional educational programs, perhaps in the form of seminars or mini-courses, as well as continuing education programs for practicing pediatricians.

Future effects of abuse differ from child to child

Abuse of a child, whether physical, sexual, or emotional, leaves the child with lifelong scars, whether visible or not. Recent studies suggest that approximately one third of the victims of child abuse will themselves become abusers (*NY Times*, January 24, 1989, pp C1, C6). Although this figure is lower than once thought, it does not portray the whole picture—child abuse also increases the probability of subsequent problems such as depression, alcohol and drug abuse, sexual maladjustment, and multiple personality.

Researchers are now focusing their efforts on identifying the factors that differentiate those abused children that grow up to be abusers from those who overcome their traumatic childhood. "Key factors found to worsen the long-term impact of abuse are: abuse that started early, abuse that lasted for a long time, abuse in which the perpetrator had a close relationship to the victim, abuse that the child perceived as particularly harmful, and abuse that occurred within a cold emotional atmosphere in the family. These factors, researchers say, help identify which children need treatment most urgently."

The emotional support of a friend or relative or the nurturing environment of professional therapy appear to be important in dealing with the experiences of child abuse. These relationships can help the victims admit that the abuse occurred, resist the all too common and unhealthy response of blaming themselves for the abuse, and deal with the consequences of childhood trauma.

Public disclosure of private information about victims of abuse

The Council on Child and Adolescent Health of the American Academy of Pediatrics has approved a position statement on the public disclosure of information about victims of child abuse and their families (*Pediatrics* 1988; 82:387). The academy contends that such disclosures, particularly with regard to sexual abuse, have resulted in serious harm to victims, and recommends that "all states adopt laws forbidding public disclosure in the mass media of information that identifies victims of child abuse, their families, and perpetrators, who may be minors, unless ordered by a judge who has jurisdiction, as in a dependency hearing or in the trial of a perpetrator." Information should be disclosed only when the "public interest in the case is already great . . . and the risk of harm to the child is small." The academy also recommends that the media "exercise great caution in publishing any information that includes the names of victims of sexual or physical abuse."

American Dental Association urges dentists to become more aware of child abuse

In 1987, the American Dental Association (ADA) reported that dentists were not making a great enough effort to diagnose and report suspected child abuse (*Dental Management* 1988; 28:33). Although 5% to 65% of all cases of child abuse involve orofacial injuries, few of these cases are reported by dentists, primarily because of a fear of getting involved. Other reasons cited by the report included "discomfort in confronting parents, lack of confidence in the child's account of the injuries, and reluctance to disrupt the patient-dentist relationship."

Recognizing the need for increased training and exposure of dentists in diagnosing child abuse, the ADA included in its report a description of the most prominent signs and symptoms of abuse: "difficulty in walking, standing, or sitting; an apprehensive or overly aggressive/hyperactive behavior; unnatural fear of a guardian; wincing away from a hand or voice; a vacant, withdrawn look to eye and face (as if they had given up); a child who is chronically ill for no good reason; or just something that makes the child different."

For the benefit of all health care practitioners, there are child abuse telephone hot lines in all 50 states.

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Anthony Thomas Capello, MD, Albany. Died December 4, 1988; age 81. Dr Capello was a 1937 graduate of Middlesex University School of Medicine, Waltham, Massachusetts. He was a Fellow of the American Academy of Family Physicians. His memberships included the New York State Society for Medical Research, the American Occupational Medical Association, the American Geriatrics Society, the Medical Society of the County of Albany, and the Medical Society of the State of New York.

Isadore A. Caputo, MD, Brooklyn. Died December 8, 1988; age 60. Dr Caputo was a 1953 graduate of the State University of New York Health Science Center at Brooklyn. He was a Diplomate of the American Board of Internal Medicine. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Joseph T. Cavaliere, MD, New Hyde Park. Died December 17, 1988; age 72. Dr Cavaliere was a 1941 graduate of the New York Medical College. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Ophthalmology. His memberships included the New York Academy of Medicine, the American Academy of Ophthalmology and Otolaryngology, the Nassau County Medical Society, and the Medical Society of the State of New York.

Edward I. D'Orsogna, MD, Binghamton. Died November 26, 1988; age 79. Dr D'Orsogna was a 1935 graduate of the Facolta di Medicina e Chirurgia dell'Universita di Bologna, Bologna, Italy. He was a member of the American School Health Association, the Broome County Medical Society, and the Medical Society of the State of New York.

Albert Decker, MD, New York. Died November 27, 1988; age 93. Dr Decker was a 1920 graduate of the New York University School of Medicine, New York. He was a Fellow of the American College of Surgeons and the American College of Obstetricians and Gynecologists and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the American Fertility Society, the New York County Medical Society, and the Medical Society of the State of New York.

Armand L. DiFrancesco, MD, Buffalo. Died November 16, 1988; age 67. Dr DiFrancesco was a 1949 graduate of the University of Western Ontario Faculty of Medicine, London, Ontario, Canada. He was a Fellow of the American Psychiatric Association and a Diplomate of the American Board of Psychiatry and Neurology. He was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Isadore Earle Gerber, MD, New York. Died December 21, 1988; age 86. Dr Gerber was a 1926 graduate of the University of Maryland School of Medicine, Baltimore, Maryland. He was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine and the American Board of Pathology. He was a member of the New York Academy of Medicine, the New York Pathological Society, the American Association of Pathologists and Bacteriologists, the New York County Medical Society, and the Medical Society of the State of New York.

Rebecca Elisabeth Gray, MD, Wantagh. Died December 14, 1988; age 35. Dr Gray was a 1980 graduate of the New York Medical College, New York. She was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Gordon Lemont Green, MD, Huntington. Died August 15, 1988; age 84. Dr Green was a 1929 graduate of the Loyola University Stritch School of Medicine, Maywood, Illinois. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

Hilbert L. Harris, MD, Syracuse. Died November 22, 1988; age 80. Dr Harris was a 1934 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a Fellow of the American College of Allergists and the American College of Physicians and a Diplomate of the American Board of Internal Medicine. He was a member of the New York Academy of Medicine, the Onondaga County Medical Society, and the Medical Society of the State of New York.

David Abraham Harrison, MD, Syracuse. Died November 28, 1988; age 92. Dr Harrison was a 1921 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a Fellow of the American College of Chest Physicians. His memberships included the New York Academy of Medicine, the American Thoracic Society, the Medical Society of the County of Oneida, and the Medical Society of the State of New York.

Ralph Jones, MD, Pocono Lake, Pennsylvania. Died November 18, 1988; age 70. Dr Jones was a 1943 graduate of the University of Pennsylvania School of Medicine, Philadelphia. Dr Jones was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. He was a member of the American Association for Cancer Research, the Medical Society of the County of Niagara, and the Medical Society of the State of New York.

Seymour Robert Kaplan, MD, New York. Died September 20, 1988; age 66. Dr Kaplan was a 1945 graduate of Duke University School of Medicine, Durham, North Carolina. He was a Diplomate of the American Board of Psychiatry and Neurology. He was a member of the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Harry Durwood Kingsley, MD, Ft Pierce, Florida. Died November 12, 1988; age 73. Dr Kingsley was a 1941 graduate of the University of Rochester School of Medicine-Dentistry, Rochester. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. He was a member of the New York Academy of Medicine, the New York State Society for Medical Research, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Robert W. Kohn, MD, New York. Died July 24, 1988; age 40. Dr Kohn was a 1974 graduate of the Cornell University Medical College, New York. He was a Fellow of the American Board of Physicians and a Diplomate of the American Board of Internal Medicine. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Joseph Mario Matturri, MD, Pelham Manor. Died December 13, 1988; age 77. Dr Matturri was a 1937 graduate of the Facolta di Medicina e Chirurgia dell'Universita di Roma, Rome, Italy. Dr Matturri was a Fellow of the American College of Chest Physicians. His memberships included the American Thoracic Society, the American Academy of Family Physicians, the American Geriatric Society, the Bronx County Medical Society, and the Medical Society of the State of New York.

Thomas McGarry, MD, Schenectady. Died August 27, 1988; age 68. Dr McGarry was a 1951 graduate of the University of Vermont College of Medicine, Burlington, Vermont. He was a Fellow of the American College of Anesthesiologists and a Diplomate of the American Board of Anesthesiology. His memberships included the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the Medical Society of the County of Schenectady, and the Medical Society of the State of New York.

Philip Parillo, MD, Schenectady. Died September 1, 1988; age 81. Dr Parillo was a 1935 graduate of the Albany Medical College of Union University, Albany. Dr Parillo was a Fellow of the American College of Gastroenterology. He was a member of the Medical Society of the County of Schenectady and the Medical Society of the State of New York.

Arthur J. Reissig, MD, Buffalo. Died November 14, 1988; age 92. Dr Reissig was a 1921 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. He was a Diplomate of the American Board of Internal Medicine. His memberships included the New York Academy of Medicine, the American Geriatrics Society, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Henry Steven Rubinstein, MD, Boca Raton, Florida. Died November 24, 1988; age 77. Dr Rubinstein was a 1939 graduate of the Deutsche Univerzita Medizinische Fakulta, Praha, Czechoslovakia. He was a Fellow of the American College of Angiology. His memberships included the American Academy of Family Physicians, the American Geriatrics Society, the Onondaga County Medical Society, and the Medical Society of the State of New York.

Peter K. Sabey, MD, Rochester. Died November 30, 1988; age 89. Dr Sabey was a 1925 graduate of the Universitaet Leipzig Medizinische Fakultaet, Saxony, Germany. His memberships included the American Ophthalmological Society, the American Academy of Ophthalmology and Otolaryngology, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

John J. Sauer, MD, New York. Died December 4, 1988; age 77. Dr Sauer was a 1937 graduate of the State University of New York Health Science Center at Brooklyn. He was a Fellow of the American College of Surgeons, a Qualified Fellow of the International College of Surgeons, and a Diplomate of the American Board of Ophthalmology. His memberships included the New York Academy of Medicine, the New York Pathological Society, the New York Ophthalmology Society, the American Academy of Ophthalmology and Otolaryngology, the Pan American Association of Ophthalmology, the American Geriatrics Society, the New York County Medical Society, and the Medical Society of the State of New York.

Wallace M. Sheridan, MD, New York. Died December 20, 1988; age 80. Dr Sheridan was a 1935 graduate of Harvard Medical School, Boston. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. He was a member of the New York Academy of Medicine, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Charles Timothy Sullivan, MD, Rochester. Died December 5, 1988; age 83. Dr Sullivan was a 1932 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Ophthalmology. His memberships included the American Academy of Ophthalmology and Otolaryngology, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Samuel Waldman, MD, Floral Park. Died June 6, 1988; age 79. Dr Waldman was a 1932 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the American College of Cardiology, the American College of Physicians, the American College of Chest Physicians, and the American College of Preventive Medicine, and a Diplomate of the American Board of Internal Medicine. His memberships included the New York State Society of Internal Medicine, the Clinical Society, New York Diabetes Association, the New York Cardiological Society, the International Society of Internal Medicine, the Pan American Medical Association, the Academy of Psychosomatic Medicine, the American Geriatrics Society, the American Society of Internal Medicine, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Rudolf Weil, MD, New York. Died September 26, 1988; age 91. Dr Weil was a 1922 graduate of the Medizinische Fakultaet, Albert Ludwigs Universitaet, Freiburg im Breisgau, Baden-Wurttemberg, Germany. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Lewis R. Wolberg, MD, New York. Died February 3, 1988; age 83. Dr Wolberg was a 1930 graduate of the Tufts University School of Medicine, Boston. He was a Fellow of the American Psychiatric Association and a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the Academy of Psychoanalysis, the Academy of Psychosomatic Medicine, the Association for Advancement of Psychotherapy, the New York County Medical Society, and the Medical Society of the State of New York.

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1988; 88:456–462). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith^2 reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al.* Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83:925–927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics*, ed 12. Philadelphia, WB Saunders Co, 1983, pp 337–338.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Pascal James Imperato, MD, Editor, *New York State Journal of Medicine*, PO Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

AUXILIARY TO THE MEDICAL SOCIETY OF THE STATE OF NEW YORK

Barbara Ramsdell
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Marie Rizzuto
President-Elect

The Auxiliary to the Medical Society of the State of New York
53rd Annual Convention

President—Barbara Ramsdell (John A.)
President-Elect—Marie Rizzuto (Donald)
Convention Chairman—Gail Murphy (Gerald)

April 6, 1989

1:00 pm–4:00 pm	Registration, *Ballroom Mezzanine*
2:30 pm–5:00 pm	AMSSNY Board of Directors Meeting, *Algonquin Room*

April 7, 1989

8:00 am–4:00 pm	Registration, *Ballroom Mezzanine*
8:00 am	Continental Breakfast *Boulevard Room*
8:30 am–5:00 pm	Opening session of the 53rd Annual Meeting of the House of Delegates, *Algonquin Room* Keynote Speaker: Mary Strauss (Albert J., Jr.) AMA Auxiliary President
12:30 pm–1:00 pm	Past Presidents' Reception, *Windsor Room*
1:00 pm–2:30 pm	President's Luncheon in honor of Barbara Ramsdell (John A.) *Windsor Room*
2:30 pm–5:00 pm	House of Delegates reconvenes, *Algonquin Room.* Voting for AMSSNY Nominating Committee Delegates to the 1989 AMAA Annual Convention.

April 7, 1989

5:00 pm–6:00 pm	Gavel Club, *Algonquin Room*
8:30 pm	AMA-ERF Theatre Benefit, sponsored by Monroe County Medical Auxiliary, GeVa Theatre: *The Italian-American Reconciliation*

April 8, 1989

8:00 am–10:00 am	Registration, *Ballroom Mezzanine*
8:00 am	Continental Breakfast, *Windsor Room*
8:30 am–11:00 am	53rd Annual Meeting of the House of Delegates reconvenes, *Windsor Room* Installation of the 1989–1990 Officers Inaugural Address: Marie Rizzuto (Donald H.)
11:00 am–1:00 pm	AMSSNY Board of Directors Meeting, *Algonquin Room*

All spouses of members of the Medical Society of the State of New York are welcome to participate in the activities of the 53rd Annual Convention of the Auxiliary to the Medical Society of the State of New York.

For further details regarding:
The President's Luncheon—Friday, April 7, 1989, 12:30 pm—and the Theatre Benefit—Friday, April 7, 1989, 8:30 pm. Please contact Gloria Cassidy, AMSSNY Staff Executive, (516) 488-6100 (ext. 255).

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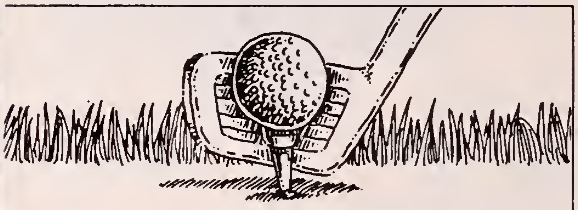

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PRECAUTIONS: PARAFON FORTE DSC chlorzoxazone should be used with caution in patients with known allergies or with a history of allergic reactions to drugs. If a sensitivity reaction occurs such as urticaria, redness, or itching of the skin, the drug should be stopped. If any symptoms suggestive of liver dysfunction are observed, the drug should be discontinued.
ADVERSE REACTIONS: After more than twenty-six years of extensive clinical use of chlorzoxazone-containing products in an estimated thirty-two million patients, it is apparent that the drug is well tolerated and seldom produces undesirable side effects. Occasional patients may develop gastrointestinal disturbances. It is possible in rare instances that chlorzoxazone may have been associated with gastrointestinal bleeding. Drowsiness, dizziness, lightheadedness, malaise, or overstimulation may be noted by an occasional patient. Rarely allergic-type skin rashes, petechiae, or ecchymoses may develop during treatment. Angioneurotic edema or anaphylactic reactions are extremely rare. There is no evidence that the drug will cause renal damage. Rarely, a patient may note discoloration of the urine resulting from a phenolic metabolite of chlorzoxazone. This finding is of no known clinical significance. Approximately thirty-six patients have been reported in whom the administration of chlorzoxazone-containing products was suspected as being the cause of liver damage. In one case, the jaundice was subsequently considered to be due to a carcinoma of the head of the pancreas rather than to the drug. In a second case, there was no jaundice but an elevated alkaline phosphatase and BSP retention. In this patient there was a malignancy with bony and liver metastases. The role of the drug was difficult to determine. A third and fourth case had cholelithiasis. Diagnosis in a fifth case was submassive hepatic necrosis possibly due to abusive use of the drug for approximately one year. The remaining cases had a clinical picture compatible with either a viral hepatitis or a drug-induced hepatitis. In all these latter cases, the drug was stopped, and with one exception, the patients recovered. It is not possible to state that the hepatitis in these patients was or was not drug-induced.
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References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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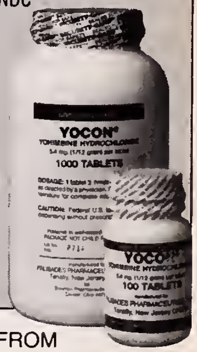

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Forty Seven Medical Equipment Corp. 22A
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Eli Lilly and Company 15A
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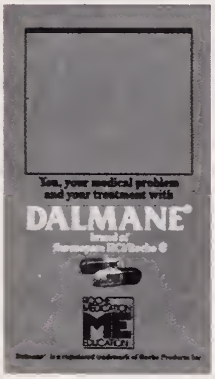

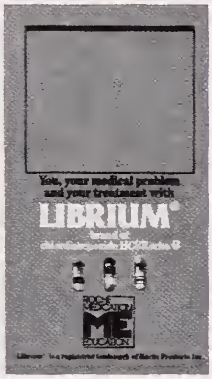

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APRIL 1989 • Volume 89, Number 4

Contents

COMMENTARIES

Mandatory Medicare assignment 195
DONALD F. FOY, GERARD L. CONWAY, JD

The tale of Ondine: A curse, a kiss, a clasp, and a comment 196
FRANK M. TAMARIN, MD; ROY J. GOLDBERG, MD; ROBERT D. BRANDSTETTER, MD

RESEARCH PAPERS

Coronary care in New York City hospitals 199
MARVIN BERGER, MD; HOWARD S. FRIEDMAN, MD; LINDA M. GERBER, PhD; MARK GREENBERG; MD; BARBARA LEVINE, PhD, RD; MICHAEL B. MIRSKY, MSE; CARL H. NACHT, MD; CHRISTINE SHEPPARD, RN

Diagnosis of stress-related hyperprolactinemia: Evaluation of the hyperprolactinemia rest test 205
OZGUL MUNEYYIRCI-DELALE, MD; DOV GOLDSTEIN, MD; FRANCISCO I. REYES, MD

SPECIAL ARTICLES

Periodic physician recredentialing proposed in New York State 209
ALFRED GELLHORN, MD, MARTIN CHERKASKY, MD

Quality education is essential for quality health care 213
CHARLES D. SHERMAN, JR, MD

Professional medical discipline: An overview 216
CHARLES J. VACANTI, MD

Concentrating on the problem physician: Perspectives in medical discipline 219
BRYANT L. GALUSHA, MD

POSITION PAPERS

Position paper of the Medical Society of the State of New York on physician recredentialing 222

Position statement of the Hospital Association of New York State on the Report of the New York State Advisory Committee on Physician Recredentialing 223

Position statement of The Federation of State Medical Boards of the United States on the assessment of licensed physicians 226

CASE REPORTS

Ondine's curse revisited 228
CHARLOTTE COLP, MD; ROSLYN SCHNEIDER, MD; DAVID ALTARAC, MD

Peptic ulcer disease presenting as life-threatening diarrhea following subtotal colectomy 231
JOHN B. RODGERS, MD

Endodermal sinus tumor and benign cystic teratoma of the ovary occurring simultaneously 233
ABDELMONEM ELHOSSEINY, MD; ORLANDO GONZALEZ, MD; MOHAMMED HEDJAZI, MD; VINCENT PILLARI, MD; RUDOLPH HOWARD, MD

Pulmonary hypertrophic osteoarthropathy as the initial manifestation of interstitial fibrosis 234
YIZHAK KUPFER, MD; JACOB E. GROOPMAN, MD; R. ASWINI LENORA, MD; SIDNEY TESSLER, MD

LETTERS TO THE EDITOR

Ben W. Aberman, MD 236
GERALD W. GRUMET, MD

Psychiatric teams and the psychiatrist's authority 236
FRED B. CHARATAN, MD

Poor legal representation in a malpractice lawsuit 237
SELMA J. SHERIDAN, MD

Doctors' penmanship 237
VICTOR H. FRANKEL, MD

LEADS FROM EPIDEMIOLOGY NOTES 238

BOOK REVIEWS 242

BOOKS RECEIVED 246

NEWS BRIEFS 247

OBITUARIES 249

MEDICAL MEETINGS AND LECTURES 8A

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Indianapolis, Indiana
46285

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[1] Data on file, Lilly Research Laboratories.

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AXID®

nizatidine capsules

Brief Summary

Consult the package literature for complete information.

Indications and Usage: Axid is indicated for up to eight weeks for the treatment of active duodenal ulcer. In most patients, the ulcer will heal within four weeks.

Axid is indicated for maintenance therapy for duodenal ulcer patients at a reduced dosage of 150 mg h.s. after healing of an active duodenal ulcer. The consequences of continuous therapy with Axid for longer than one year are not known.

Contraindication: Axid is contraindicated in patients with known hypersensitivity to the drug and should be used with caution in patients with hypersensitivity to other H_2-receptor antagonists

Precautions: *General* – 1 Symptomatic response to nizatidine therapy does not preclude the presence of gastric malignancy

2. Because nizatidine is excreted primarily by the kidney, dosage should be reduced in patients with moderate to severe renal insufficiency

3. Pharmacokinetic studies in patients with hepatorenal syndrome have not been done Part of the dose of nizatidine is metabolized in the liver In patients with normal renal function and uncomplicated hepatic dysfunction, the disposition of nizatidine is similar to that in normal subjects

Laboratory Tests – False-positive tests for urobilinogen with Multistix® may occur during therapy with nizatidine

Drug Interactions – No interactions have been observed between Axid and theophylline, chlordiazepoxide, lorazepam, lidocaine, phenytoin, and warfarin. Axid does not inhibit the cytochrome P-450-linked drug-metabolizing enzyme system, therefore, drug interactions mediated by inhibition of hepatic metabolism are not expected to occur. In patients given very high doses (3,900 mg) of aspirin daily, increases in serum salicylate levels were seen when nizatidine, 150 mg b.i.d., was administered concurrently

Carcinogenesis, Mutagenesis, Impairment of Fertility – A two-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa In a two-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations) The occurrence of a marginal finding at high dose only in animals given an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, mouse lymphoma assay, chromosome aberration tests, and a micronucleus test.

In a two-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny

Pregnancy – Teratogenic Effects – Pregnancy Category C – Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in one fetus and at 50 mg/kg it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in one fetus There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers – Studies conducted in lactating women have shown that <0.1% of the administered oral dose of nizatidine is secreted in human milk in proportion to plasma concentrations Caution should be exercised when administering nizatidine to a nursing mother

Pediatric Use – Safety and effectiveness in children have not been established.

Use in Elderly Patients – Ulcer healing rates in elderly patients are similar to those in younger age groups. The incidence rates of adverse events and laboratory test abnormalities are also similar to those seen in other age groups Age alone may not be an important factor in the disposition of nizatidine Elderly patients may have reduced renal function

Adverse Reactions: Clinical trials of nizatidine included almost 5,000 patients given nizatidine in studies of varying durations Domestic placebo-controlled trials included over 1,900 patients given nizatidine and over 1,300 given placebo Among reported adverse events in the domestic placebo-controlled trials, sweating (1% vs 0.2%), urticaria (0.5% vs < 0.01%), and somnolence (2.4% vs 1.3%) were significantly more common in the nizatidine group A variety of less common events was also reported; it was not possible to determine whether these were caused by nizatidine

Hepatic – Hepatocellular injury, evidenced by elevated liver enzyme tests (SGOT [AST], SGPT [ALT], or alkaline phosphatase), occurred in some patients and was possibly or probably related to nizatidine In some cases, there was marked elevation of SGOT, SGPT enzymes (greater than 500 IU/L) and, in a single instance, SGPT was greater than 2,000 IU/L. The overall rate of occurrences of elevated liver enzymes and elevations to three times the upper limit of normal, however, did not significantly differ from the rate of liver enzyme abnormalities in placebo-treated patients All abnormalities were reversible after discontinuation of Axid.

Cardiovascular – In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in two individuals administered Axid and in three untreated subjects.

CNS – Rare cases of reversible mental confusion have been reported

Endocrine – Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to Axid Impotence and decreased libido were reported with equal frequency by patients who received Axid and by those given placebo Rare reports of gynecomastia occurred.

Hematologic – Fatal thrombocytopenia was reported in a patient who was treated with Axid and another H_2-receptor antagonist. On previous occasions, this patient had experienced thrombocytopenia while taking other drugs Rare cases of thrombocytopenic purpura have been reported

Integumental – Sweating and urticaria were reported significantly more frequently in nizatidine- than in placebo-treated patients Rash and exfoliative dermatitis were also reported.

Hypersensitivity – As with other H_2-receptor antagonists, rare cases of anaphylaxis following administration of nizatidine have been reported Because cross-sensitivity in this class of compounds has been observed, H_2-receptor antagonists should not be administered to individuals with a history of previous hypersensitivity to these agents. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported

Other – Hyperuricemia unassociated with gout or nephrolithiasis was reported Eosinophilia, fever, and nausea related to nizatidine administration have been reported

Overdosage: Overdoses of Axid have been reported rarely The following is provided to serve as a guide should such an overdose be encountered

Signs and Symptoms – There is little clinical experience with overdosage of Axid in humans Test animals that received large doses of nizatidine have exhibited cholinergic-type effects, including lacrimation, salivation, emesis, miosis, and diarrhea. Single oral doses of 800 mg/kg in dogs and of 1,200 mg/kg in monkeys were not lethal Intravenous median lethal doses in the rat and mouse were 301 mg/kg and 232 mg/kg respectively

Treatment – To obtain up-to-date information about the treatment of overdose, a good resource is your certified regional Poison Control Center Telephone numbers of certified poison control centers are listed in the *Physicians' Desk Reference (PDR)*. In managing overdosage, consider the possibility of multiple drug overdoses, interaction among drugs, and unusual drug kinetics in your patient.

If overdosage occurs, use of activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy Renal dialysis for four to six hours increased plasma clearance

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Please see brief summary of prescribing information on next page.

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*Dosage must be adjusted to each patient's needs, starting with 60 to 120 mg twice daily.

BRIEF SUMMARY
CARDIZEM® SR
(diltiazem hydrochloride)
Sustained Release Capsules

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, (3) patients with hypotension (less than 90 mm Hg systolic), (4) patients who have demonstrated hypersensitivity to the drug, and (5) patients with acute myocardial infarction and pulmonary congestion documented by x-ray on admission.

WARNINGS

1. **Cardiac Conduction**. CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (nine of 2,111 patients or 0 43%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure**. Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt) An acute study of oral diltiazem in patients with impaired ventricular function (ejection fraction 24% ± 6%) showed improvement in indices of ventricular function without significant decrease in contractile function (dp/dt) Experience with the use of CARDIZEM (diltiazem hydrochloride) in combination with beta-blockers in patients with impaired ventricular function is limited Caution should be exercised when using this combination.
3. **Hypotension**. Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury** Mild elevations of transaminases with and without concomitant elevation in alkaline phosphatase and bilirubin have been observed in clinical studies. Such elevations were usually transient and frequently resolved even with continued diltiazem treatment In rare instances, significant elevations in enzymes such as alkaline phosphatase. LDH, SGOT, SGPT, and other phenomena consistent with acute hepatic injury have been noted These reactions tended to occur early after therapy initiation (1 to 8 weeks) and have been reversible upon discontinuation of drug therapy The relationship to CARDIZEM is uncertain in some cases, but probable in some (See PRECAUTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile As with any drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes, however, these changes were reversible with continued dosing

Dermatological events (see ADVERSE REACTIONS section) may be transient and may disappear despite continued use of CARDIZEM However, skin eruptions progressing to erythema multiforme and/or exfoliative dermatitis have also been infrequently reported. Should a dermatologic reaction persist, the drug should be discontinued

Drug Interaction. Due to the potential for additive effects, caution and careful titration are warranted in patients receiving CARDIZEM concomitantly with any agents known to affect cardiac contractility and/or conduction. (See WARNINGS.) Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM (See WARNINGS.)

As with all drugs, care should be exercised when treating patients with multiple medications CARDIZEM undergoes biotransformation by cytochrome P-450 mixed function oxidase. Coadministration of CARDIZEM with other agents which follow the same route of biotransformation may result in the competitive inhibition of metabolism. Dosages of similarly metabolized drugs, particularly those of low therapeutic ratio or in patients with renal and/or hepatic impairment, may require adjustment when starting or stopping concomitantly administered CARDIZEM to maintain optimum therapeutic blood levels.

Beta-blockers: Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated, but available data are not sufficient to predict the effects of concomitant treatment in patients with left ventricular dysfunction or cardiac conduction abnormalities.

Administration of CARDIZEM (diltiazem hydrochloride) concomitantly with propranolol in five normal volunteers resulted in increased propranolol levels in all subjects and bioavailability of propranolol was increased approximately 50%. If combination therapy is initiated or withdrawn in conjunction with propranolol, an adjustment in the propranolol dose may be warranted (See WARNINGS.)

Cimetidine: A study in six healthy volunteers has shown a significant increase in peak diltiazem plasma levels (58%) and area-under-the-curve (53%) after a 1-week course of cimetidine at 1,200 mg per day and diltiazem 60 mg per day Ranitidine produced smaller, nonsignificant increases. The effect may be mediated by cimetidine's known inhibition of hepatic cytochrome P-450, the enzyme system probably responsible for the first-pass metabolism of diltiazem. Patients currently receiving diltiazem therapy should be carefully monitored for a change in pharmacological effect when initiating and discontinuing therapy with cimetidine An adjustment in the diltiazem dose may be warranted

Digitalis: Administration of CARDIZEM with digoxin in 24 healthy male subjects increased plasma digoxin concentrations approximately 20%. Another investigator found no increase in digoxin levels in 12 patients with coronary artery disease Since there have been conflicting results regarding the effect of digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing CARDIZEM therapy to avoid possible over- or under-digitalization. (See WARNINGS.)

Anesthetics: The depression of cardiac contractility, conductivity, and automaticity as well as the vascular dilation associated with anesthetics may be potentiated by calcium channel blockers When used concomitantly, anesthetics and calcium blockers should be titrated carefully

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity There was also no mutagenic response in in vitro bacterial tests No intrinsic effect on fertility was observed in rats.

Pregnancy Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates There was an increased incidence of stillbirths at doses of 20 times the human dose or greater

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. Diltiazem is excreted in human milk One report suggests that concentrations in breast milk may approximate serum levels. If use of CARDIZEM is deemed essential, an alternative method of infant feeding should be instituted

Pediatric Use. Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded from these studies.

The adverse events described below represent events observed in clinical studies of hypertensive patients receiving either CARDIZEM Tablets or CARDIZEM SR Capsules as well as experiences observed in studies of angina and during marketing The most common events in hypertension studies are shown in a table with rates in placebo patients shown for comparison. Less common events are listed by body system; these include any adverse reactions seen in angina studies that were not observed in hypertension studies In all hypertensive patients studied (over 900), the most common adverse events were edema (9%), headache (8%), dizziness (6%), asthenia (5%), sinus bradycardia (3%), flushing (3%), and 1° AV block (3%). Only edema and perhaps bradycardia and dizziness were dose related The most common events observed in clinical studies (over 2,100 patients) of angina patients and hypertensive patients receiving CARDIZEM Tablets or CARDIZEM SR Capsules were (ie, greater than 1%) edema (5 4%), headache (4 5%), dizziness (3.4%), asthenia (2 8%), first-degree AV block (1.8%), flushing (1.7%), nausea (1.6%), bradycardia (1 5%), and rash (1.5%).

DOUBLE BLIND PLACEBO CONTROLLED HYPERTENSION TRIALS

Adverse	Diltiazem N=315 # pts (%)	Placebo N=211 # pts (%)
headache	38 (12%)	17 (8%)
AV block first degree	24 (7.6%)	4 (1.9%)
dizziness	22 (7%)	6 (2.8%)
edema	19 (6%)	2 (0.9%)
bradycardia	19 (6%)	3 (1 4%)
ECG abnormality	13 (4 1%)	3 (1.4%)
asthenia	10 (3.2%)	1 (0.5%)
constipation	5 (1.6%)	2 (0.9%)
dyspepsia	4 (1.3%)	1 (0.5%)
nausea	4 (1 3%)	2 (0 9%)
palpitations	4 (1 3%)	2 (0.9%)
polyuria	4 (1 3%)	2 (0.9%)
somnolence	4 (1 3%)	—
alk phos increase	3 (1%)	1 (0.5%)
hypotension	3 (1%)	1 (0.5%)
insomnia	3 (1%)	1 (0.5%)
rash	3 (1%)	1 (0.5%)
AV block second degree	2 (0 6%)	—

In addition, the following events were reported infrequently (less than 1%) or have been observed in angina trials. In many cases, the relation to drug is uncertain.

Cardiovascular:	Angina, arrhythmia, bundle branch block, tachycardia, ventricular extrasystoles, congestive heart failure, syncope
Nervous System:	Amnesia, depression, gait abnormality, hallucinations, nervousness, paresthesia, personality change, tinnitus, tremor, abnormal dreams.
Gastrointestinal:	Anorexia, diarrhea, dysgeusia, mild elevations of SGOT, SGPT, and LDH (see hepatic warnings), vomiting, weight increase, thirst.
Dermatological:	Petechiae, pruritus, photosensitivity, urticaria.
Other:	Amblyopia, CPK increase, dyspnea, epistaxis, eye irritation, hyperglycemia, sexual difficulties, nasal congestion, nocturia, osteoarticular pain, impotence, dry mouth.

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM alopecia, gingival hyperplasia, erythema multiforme, and leukopenia Definitive cause and effect relationship between these events and CARDIZEM therapy cannot yet be established.

Issued 1/89

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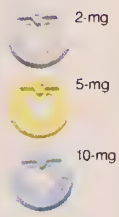

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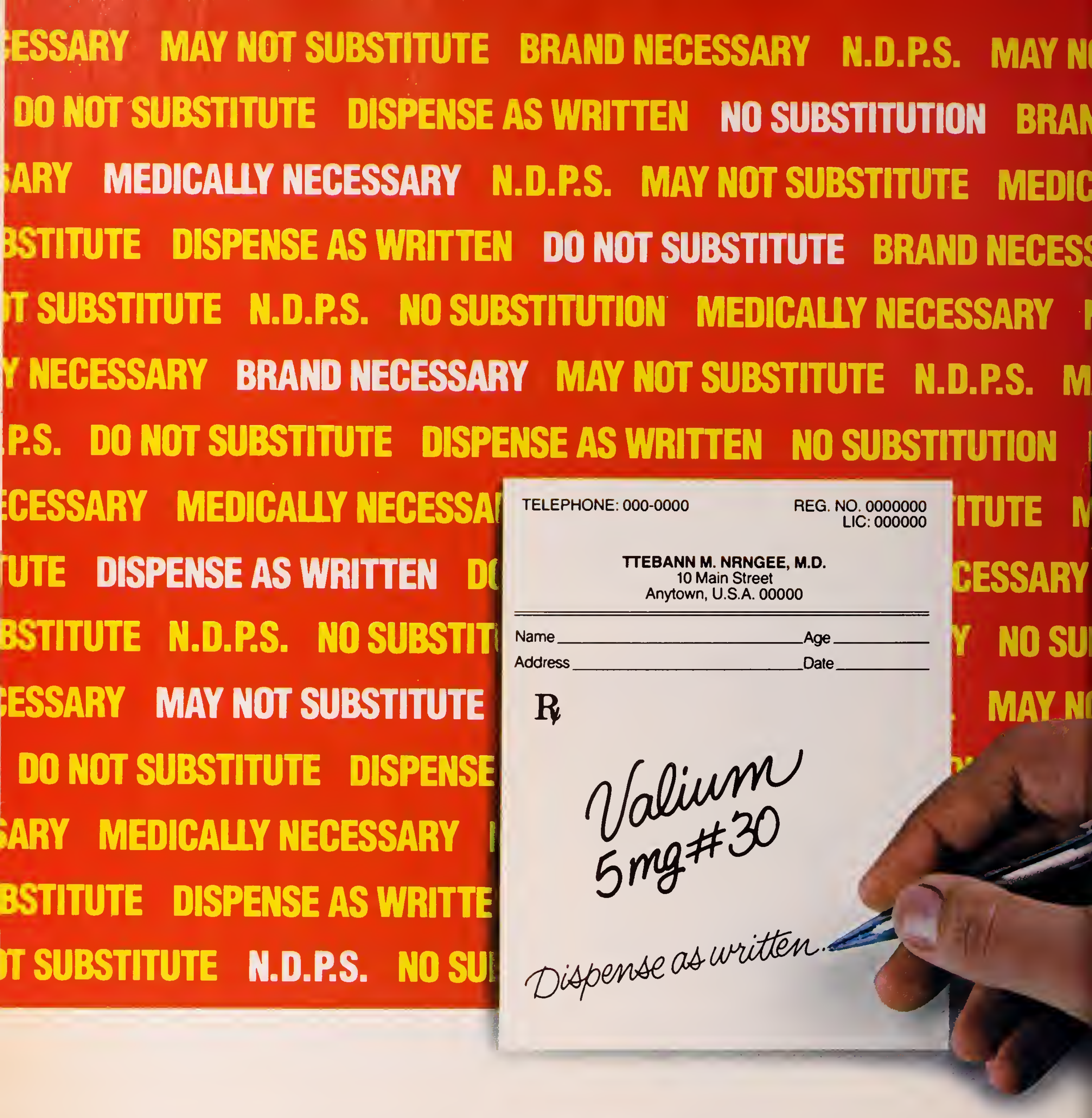
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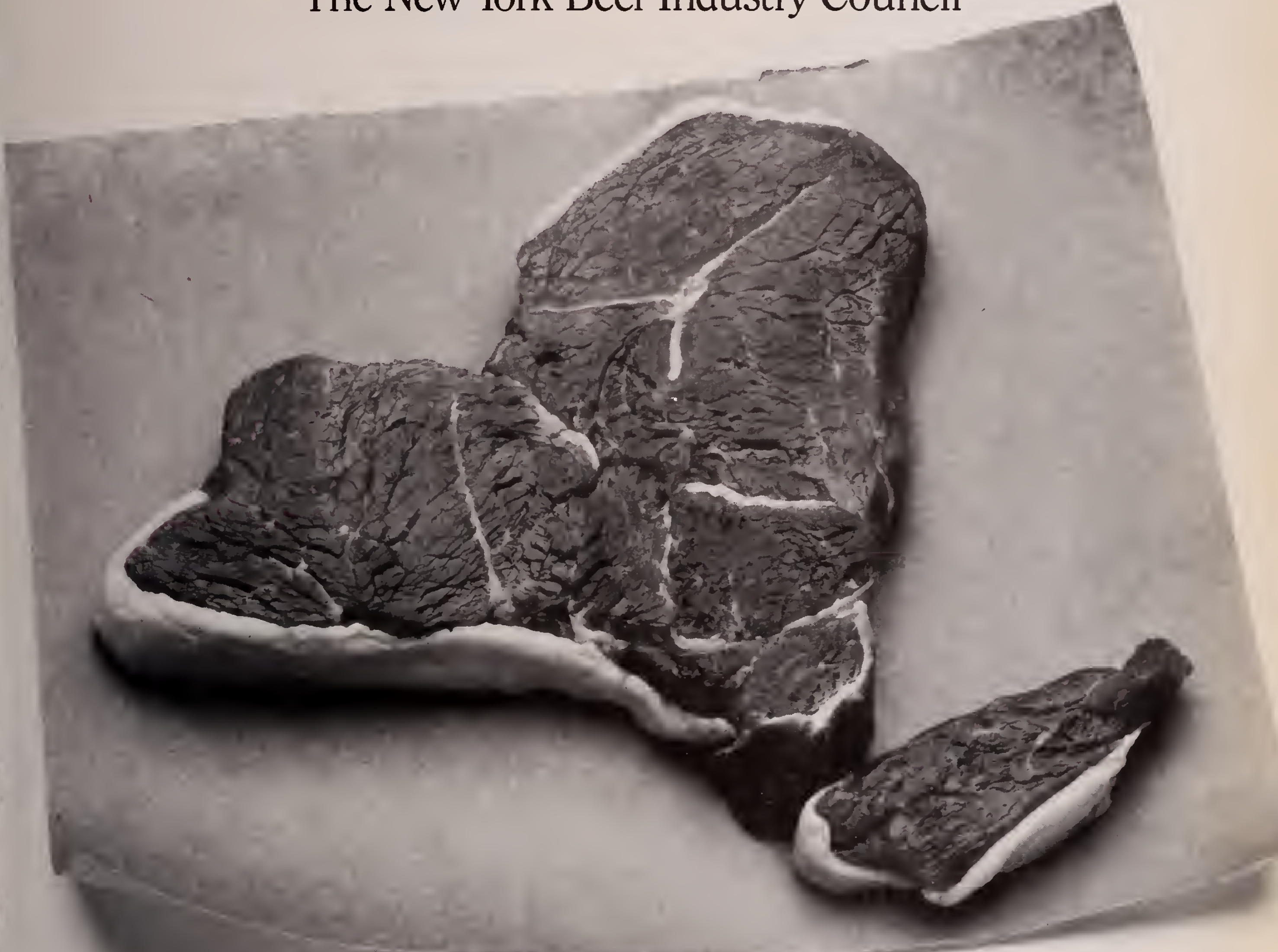

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The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices. Information must be submitted at least three months prior to the event.*

MAY 1989

AROUND THE STATE

ALBANY

May 4–5. **The Use of the Saphenous Vein In-Situ as an Arterial Bypass.** Albany Medical College & Desmond Americana Inn. *May 5.* **AIDS Conference.** Marriott Hotel. *May 18.* **Dermatology Teaching Day.** Contact: Diane M. Martin, Office of CME, Albany Medical College, 47 New Scotland Ave, Albany, NY 12208. Tel: (518) 445-5828.

BRONX

May 24. **The History of Drug Abuse and Narcotic Control.** Contact: Mark L. Taff, MD, President, New York Society of Forensic Sciences, 130 Gold Pl, Malverne, NY 11565. Tel: (516) 887-4691.

MANHATTAN

May 1–5. **Surgical Neuroangiography.** 36 Cat I Credits. NYU Medical Center. Contact: NYU Medical Center Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

May 5. **Rehabilitation of the Elderly.** Beth Israel Medical Center. Contact: Beverley R. Baptiste, Medical Education, Beth Israel Medical Center, First Ave at E 16th St, New York, NY 10003. Tel: (212) 420-2849.

May 12. **The Impact of Transplantation on Nursing Practice.** NYU Medical Center. Contact: NYU Medical Center Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

May 18–20. **Fourth International Symposium. Vascular Surgery 1989.** 24 Cat I Credits. New York Marriott Marquis. Contact: Ann J. Boehme, CMP, Asso-

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Contraindications: VASOTEC® (Enalapril Maleate, MSD) is contraindicated in patients who are hypersensitive to this product and in patients with a history of angioedema related to previous treatment with an ACE inhibitor.

Warnings: *Angioedema.* Angioedema of the face, extremities, lips, tongue, glottis, and/or larynx has been reported in patients treated with ACE inhibitors, including VASOTEC. In such cases, VASOTEC should be promptly discontinued and the patient carefully observed until the swelling disappears. In instances where swelling has been confined to the face and lips, the condition has generally resolved without treatment, although antihistamines have been useful in relieving symptoms. Angioedema associated with laryngeal edema may be fatal **Where there is involvement of the tongue, glottis, or larynx likely to cause airway obstruction, appropriate therapy, e.g., subcutaneous epinephrine solution 1:1000 (0.3 mL to 0.5 mL), should be promptly administered.** (See ADVERSE REACTIONS.)

Hypotension: Excessive hypotension is rare in uncomplicated hypertensive patients treated with VASOTEC alone. Heart failure patients given VASOTEC commonly have some reduction in blood pressure, especially with the first dose, but discontinuation of therapy for continuing symptomatic hypotension usually is not necessary when dosing instructions are followed; caution should be observed when initiating therapy (See DOSAGE AND ADMINISTRATION.) Patients at risk for excessive hypotension, sometimes associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death, include those with the following conditions or characteristics: heart failure, hyponatremia, high-dose diuretic therapy, recent intensive diuresis or increase in diuretic dose, renal dialysis, or severe volume and/or salt depletion of any etiology. It may be advisable to eliminate the diuretic (except in heart failure patients), reduce the diuretic dose, or increase salt intake cautiously before initiating therapy with VASOTEC in patients at risk for excessive hypotension who are able to tolerate such adjustments (See PRECAUTIONS, *Drug Interactions* and ADVERSE REACTIONS.) In patients at risk for excessive hypotension, therapy should be started under very close medical supervision and such patients should be followed closely for the first two weeks of treatment and whenever the dose of enalapril and/or diuretic is increased. Similar considerations may apply to patients with ischemic heart disease or cardiovascular disease in whom an excessive fall in blood pressure could result in a myocardial infarction or cerebrovascular accident.

If excessive hypotension occurs, the patient should be placed in supine position and, if necessary, receive an intravenous infusion of normal saline. A transient hypotensive response is not a contraindication to further doses of VASOTEC, which usually can be given without difficulty once the blood pressure has stabilized If symptomatic hypotension develops, a dose reduction or discontinuation of VASOTEC or concomitant diuretic may be necessary

Neutropenia/Agranulocytosis: Another ACE inhibitor, captopril, has been shown to cause agranulocytosis and bone marrow depression, rarely in uncomplicated patients but more frequently in patients with renal impairment, especially if they also have a collagen vascular disease. Available data from clinical trials of enalapril are insufficient to show that enalapril does not cause agranulocytosis at similar rates. Foreign marketing experience has revealed several cases of neutropenia or agranulocytosis in which a causal relationship to enalapril cannot be excluded. Periodic monitoring of white blood cell counts in patients with collagen vascular disease and renal disease should be considered.

Precautions: *General: Impaired Renal Function.* As a consequence of inhibiting the renin-angiotensin-aldosterone system, changes in renal function may be anticipated in susceptible individuals In patients with severe heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with ACE inhibitors, including VASOTEC, may be associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death.

In clinical studies in hypertensive patients with unilateral or bilateral renal artery stenosis, increases in blood urea nitrogen and serum creatinine were observed in 20% of patients These increases were almost always reversible upon discontinuation of enalapril and/or diuretic therapy In such patients, renal function should be monitored during the first few weeks of therapy

Some patients with hypertension or heart failure with no apparent preexisting renal vascular disease have developed increases in blood urea and serum creatinine, usually minor and transient, especially when VASOTEC has been given concomitantly with a diuretic This is more likely to occur in patients with preexisting renal impairment Dosage reduction and/or discontinuation of the diuretic and/or VASOTEC may be required.

Evaluation of patients with hypertension or heart failure should always include assessment of renal function. (See DOSAGE AND ADMINISTRATION.)

Hyperkalemia: Elevated serum potassium (> 5.7 mEq/L) was observed in approximately 1% of hypertensive patients in clinical trials. In most cases these were isolated values which resolved despite continued therapy. Hyperkalemia was a cause of discontinuation of therapy in 0.28% of hypertensive patients. In clinical trials in heart failure, hyperkalemia was observed in 3.8% of patients, but was not a cause for discontinuation.

Risk factors for the development of hyperkalemia include renal insufficiency, diabetes mellitus, and the concomitant use of potassium-sparing diuretics, potassium supplements, and/or potassium-containing salt substitutes, which should be used cautiously, if at all, with VASOTEC. (See *Drug Interactions.*)

Surgery/Anesthesia: In patients undergoing major surgery or during anesthesia with agents that produce hypotension, enalapril may block angiotensin II formation secondary to compensatory renin release If hypotension occurs and is considered to be due to this mechanism, it can be corrected by volume expansion

Information for Patients:

Angioedema: Angioedema, including laryngeal edema, may occur especially following the first dose of enalapril. Patients should be so advised and told to report immediately any signs or symptoms suggesting angioedema (swelling of face, extremities, eyes, lips, tongue, difficulty in swallowing or breathing) and to take no more drug until they have consulted with the prescribing physician.

Hypotension. Patients should be cautioned to report lightheadedness especially during the first few days of therapy If actual syncope occurs, the patients should be told to discontinue the drug until they have consulted with the prescribing physician.

All patients should be cautioned that excessive perspiration and dehydration may lead to an excessive fall in blood pressure because of reduction in fluid volume. Other causes of volume depletion such as vomiting or diarrhea may also lead to a fall in blood pressure; patients should be advised to consult with the physician.

Hyperkalemia Patients should be told not to use salt substitutes containing potassium without consulting their physician

Neutropenia: Patients should be told to report promptly any indication of infection (e.g., sore throat, fever) which may be a sign of neutropenia

NOTE: As with many other drugs, certain advice to patients being treated with enalapril is warranted. This information is intended to aid in the safe and effective use of this medication. It is not a disclosure of all possible adverse or intended effects

Drug Interactions.

Hypotension: Patients on Diuretic Therapy Patients on diuretics and especially those in whom diuretic therapy was recently instituted may occasionally experience an excessive reduction of blood pressure after initiation of therapy with enalapril. The possibility of hypotensive effects with enalapril can be minimized by either discontinuing the diuretic or increasing the salt intake prior to initiation of treatment with enalapril. If it is necessary to continue the diuretic, provide close medical supervision after the initial dose for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and DOSAGE AND ADMINISTRATION.)

Agents Causing Renin Release: The antihypertensive effect of VASOTEC is augmented by antihypertensive agents that cause renin release (e.g., diuretics).

Other Cardiovascular Agents: VASOTEC has been used concomitantly with beta-adrenergic-blocking agents, methyldopa, nitrates, calcium-blocking agents, hydralazine, prazosin, and digoxin without evidence of clinically significant adverse interactions.

Agents Increasing Serum Potassium. VASOTEC attenuates potassium loss caused by thiazide-type diuretics. Potassium-sparing diuretics (e.g., spironolactone, triamterene, or amiloride), potassium supplements, or potassium-containing salt substitutes may lead to significant increases in serum potassium. Therefore, if concomitant use of these agents is indicated because of demonstrated hypokalemia, they should be used with caution and with frequent monitoring of serum potassium. Potassium-sparing agents should generally not be used in patients with heart failure receiving VASOTEC.

Lithium. A few cases of lithium toxicity have been reported in patients receiving concomitant VASOTEC and lithium and were reversible upon discontinuation of both drugs. Although a causal relationship has not been established, it is recommended that caution be exercised when lithium is used concomitantly with VASOTEC and serum lithium levels should be monitored frequently

Pregnancy—Category C: There was no fetotoxicity or teratogenicity in rats treated with up to 200 mg/kg/day of enalapril (333 times the maximum human dose). Fetotoxicity, expressed as a decrease in average fetal weight, occurred in rats given 1200 mg/kg/day of enalapril but did not occur when these animals were supplemented with saline Enalapril was not teratogenic in rabbits. However, maternal and fetal toxicity occurred in some rabbits at doses of 1 mg/kg/day or more. Saline supplementation prevented the maternal and fetal toxicity seen at doses of 3 and 10 mg/kg/day, but not at 30 mg/kg/day (50 times the maximum human dose).

Radioactivity was found to cross the placenta following administration of labeled enalapril to pregnant hamsters.

There are no adequate and well-controlled studies in pregnant women. VASOTEC® (Enalapril Maleate, MSD) should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers. Milk in lactating rats contains radioactivity following administration of ^{14}C enalapril maleate. It is not known whether this drug is secreted in human milk Because many drugs are secreted in human milk, caution should be exercised when VASOTEC is given to a nursing mother.

Pediatric Use: Safety and effectiveness in children have not been established

Adverse Reactions: VASOTEC has been evaluated for safety in more than 10,000 patients, including over 1000 patients treated for one year or more. VASOTEC has been found to be generally well tolerated in controlled clinical trials involving 2987 patients.

Hypertension: The most frequent clinical adverse experiences in controlled trials were: headache (5.2%), dizziness (4.3%), and fatigue (3%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in controlled clinical trials were: diarrhea (1.4%), nausea (1.4%), rash (1.4%), cough (1.3%), orthostatic effects (1.2%), and asthenia (1.1%).

Heart Failure: The most frequent clinical adverse experiences in both controlled and uncontrolled trials were: dizziness (7.9%), hypotension (6.7%), orthostatic effects (2.2%), syncope (2.2%), cough (2.2%), chest pain (2.1%), and diarrhea (2.1%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in both controlled and uncontrolled clinical trials were: fatigue (1.8%), headache (1.8%), abdominal pain (1.6%), asthenia (1.6%), orthostatic hypotension (1.6%), vertigo (1.6%), angina pectoris (1.5%), nausea (1.3%), vomiting (1.3%), bronchitis (1.3%), dyspnea (1.3%), urinary tract infection (1.3%), rash (1.3%), and myocardial infarction (1.2%)

Other serious clinical adverse experiences occurring since the drug was marketed or adverse experiences occurring in 0.5% to 1% of patients with hypertension or heart failure in clinical trials in order of decreasing severity within each category.

Cardiovascular: Myocardial infarction or cerebrovascular accident, possibly secondary to excessive hypotension in high-risk patients (see WARNINGS, *Hypotension*); cardiac arrest; pulmonary embolism and infarction, rhythm disturbances, atrial fibrillation; palpitation.

Digestive: Ileus, pancreatitis, hepatitis or cholestatic jaundice, melena, anorexia, dyspepsia, constipation, glossitis.

Nervous/Psychiatric: Depression, confusion, ataxia, somnolence, insomnia, nervousness, paresthesia

Urogenital: Renal failure, oliguria, renal dysfunction (see PRECAUTIONS and DOSAGE AND ADMINISTRATION), prostate hypertrophy.

Respiratory: Bronchospasm, rhinorrhea, asthma, upper respiratory infection.

Skin: Herpes zoster, pruritus, alopecia, flushing, photosensitivity.

Other. Muscle cramps, hyperhidrosis, impotence, blurred vision, taste alteration, tinnitus.

A symptom complex has been reported which may include fever, myalgia, and arthralgia, an elevated erythrocyte sedimentation rate may be present. Rash or other dermatologic manifestations may occur. These symptoms have disappeared after discontinuation of therapy.

Angioedema. Angioedema has been reported in patients receiving VASOTEC (0.2%). Angioedema associated with laryngeal edema may be fatal. If angioedema of the face, extremities, lips, tongue, glottis, and/or larynx occurs, treatment with VASOTEC should be discontinued and appropriate therapy instituted immediately. (See WARNINGS.)

Hypotension: In the hypertensive patients, hypotension occurred in 0.9% and syncope occurred in 0.5% of patients following the initial dose or during extended therapy Hypotension or syncope was a cause for discontinuation of therapy in 0.1% of hypertensive patients. In heart failure patients, hypotension occurred in 6.7% and syncope occurred in 2.2% of patients Hypotension or syncope was a cause for discontinuation of therapy in 1.9% of patients with heart failure. (See WARNINGS.)

Clinical Laboratory Test Findings:

Serum Electrolytes. Hyperkalemia (see PRECAUTIONS), hyponatremia.

Creatinine, Blood Urea Nitrogen. In controlled clinical trials, minor increases in blood urea nitrogen and serum creatinine, reversible upon discontinuation of therapy, were observed in about 0.2% of patients with essential hypertension treated with VASOTEC alone. Increases are more likely to occur in patients receiving concomitant diuretics or in patients with renal artery stenosis. (See PRECAUTIONS.) In patients with heart failure who were also receiving diuretics with or without digitalis, increases in blood urea nitrogen or serum creatinine, usually reversible upon discontinuation of VASOTEC and/or other concomitant diuretic therapy, were observed in about 11% of patients. Increases in blood urea nitrogen or creatinine were a cause for discontinuation in 1.2% of patients.

Hemoglobin and Hematocrit: Small decreases in hemoglobin and hematocrit (mean decreases of approximately 0.3 g % and 1.0 vol %, respectively) occur frequently in either hypertension or heart failure patients treated with VASOTEC but are rarely of clinical importance unless another cause of anemia coexists In clinical trials, less than 0.1% of patients discontinued therapy due to anemia.

Other (Causal Relationship Unknown): In marketing experience, rare cases of neutropenia, thrombocytopenia, and bone marrow depression have been reported

Liver Function Tests: Elevations of liver enzymes and/or serum bilirubin have occurred

Dosage and Administration: *Hypertension:* In patients who are currently being treated with a diuretic, symptomatic hypotension occasionally may occur following the initial dose of VASOTEC. The diuretic should, if possible, be discontinued for two to three days before beginning therapy with VASOTEC to reduce the likelihood of hypotension. (See WARNINGS.) If the patient's blood pressure is not controlled with VASOTEC alone, diuretic therapy may be resumed.

If the diuretic cannot be discontinued, an initial dose of 2.5 mg should be used under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, *Drug Interactions.*)

The recommended initial dose in patients not on diuretics is 5 mg once a day. Dosage should be adjusted according to blood pressure response. The usual dosage range is 10 to 40 mg per day administered in a single dose or in two divided doses. In some patients treated once daily, the antihypertensive effect may diminish toward the end of the dosing interval In such patients, an increase in dosage or twice-daily administration should be considered. If blood pressure is not controlled with VASOTEC alone, a diuretic may be added

Concomitant administration of VASOTEC with potassium supplements, potassium salt substitutes, or potassium-sparing diuretics may lead to increases of serum potassium (see PRECAUTIONS).

Dosage Adjustment in Hypertensive Patients with Renal Impairment: The usual dose of enalapril is recommended for patients with a creatinine clearance >30 mL/min (serum creatinine of up to approximately 3 mg/dL) For patients with creatinine clearance ≤30 mL/min (serum creatinine ≥3 mg/dL), the first dose is 2.5 mg once daily The dosage may be titrated upward until blood pressure is controlled or to a maximum of 40 mg daily

Heart Failure: VASOTEC is indicated as adjunctive therapy with diuretics and digitalis. The recommended starting dose is 2.5 mg once or twice daily. After the initial dose of VASOTEC, the patient should be observed under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, *Drug Interactions.*) If possible, the dose of the diuretic should be reduced, which may diminish the likelihood of hypotension. The appearance of hypotension after the initial dose of VASOTEC does not preclude subsequent careful dose titration with the drug, following effective management of the hypotension. The usual therapeutic dosing range for the treatment of heart failure is 5 to 20 mg daily given in two divided doses. The maximum daily dose is 40 mg. Once-daily dosing has been effective in a controlled study, but nearly all patients in this study were given 40 mg, the maximum recommended daily dose, and there has been much more experience with twice-daily dosing. In addition, in a placebo-controlled study which demonstrated reduced mortality in patients with severe heart failure (NYHA Class IV), patients were treated with 2.5 to 40 mg per day of VASOTEC, almost always administered in two divided doses. (See CLINICAL PHARMACOLOGY, *Pharmacodynamics* and *Clinical Effects.*) Dosage may be adjusted depending upon clinical or hemodynamic response. (See WARNINGS.)

Dosage Adjustment in Heart Failure Patients with Renal Impairment or Hyponatremia: In heart failure patients with hyponatremia (serum sodium <130 mEq/L) or with serum creatinine >1.6 mg/dL, therapy should be initiated at 2.5 mg daily under close medical supervision. (See DOSAGE AND ADMINISTRATION, *Heart Failure*, WARNINGS, and PRECAUTIONS, *Drug Interactions.*) The dose may be increased to 2.5 mg b.i.d., then 5 mg b.i.d. and higher as needed, usually at intervals of four days or more, if at the time of dosage adjustment there is not excessive hypotension or significant deterioration of renal function. The maximum daily dose is 40 mg

For more detailed information, consult your MSD representative or see Prescribing Information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, PA 19486. J6VS18R(815)

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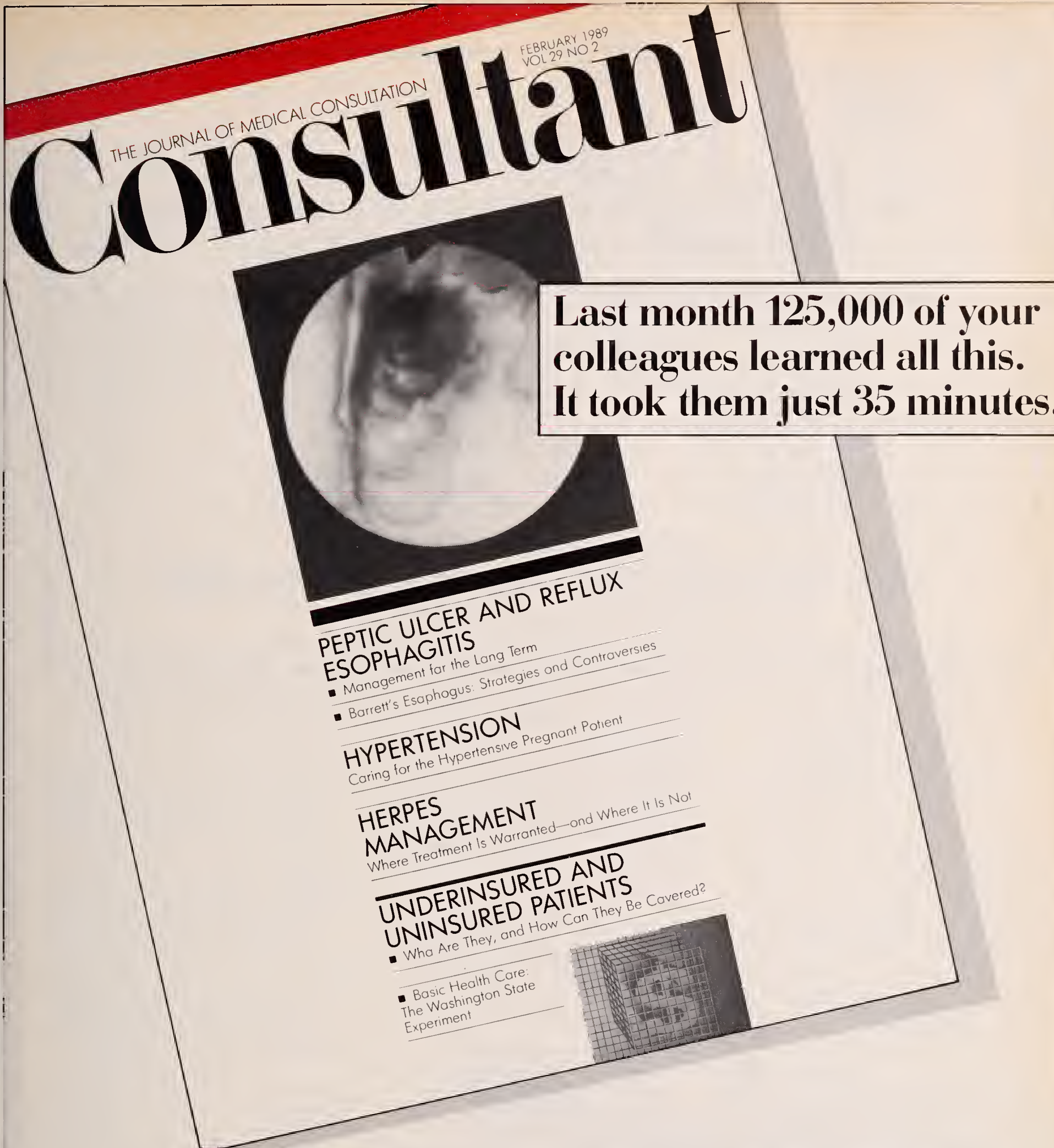

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VOL 29 NO 2

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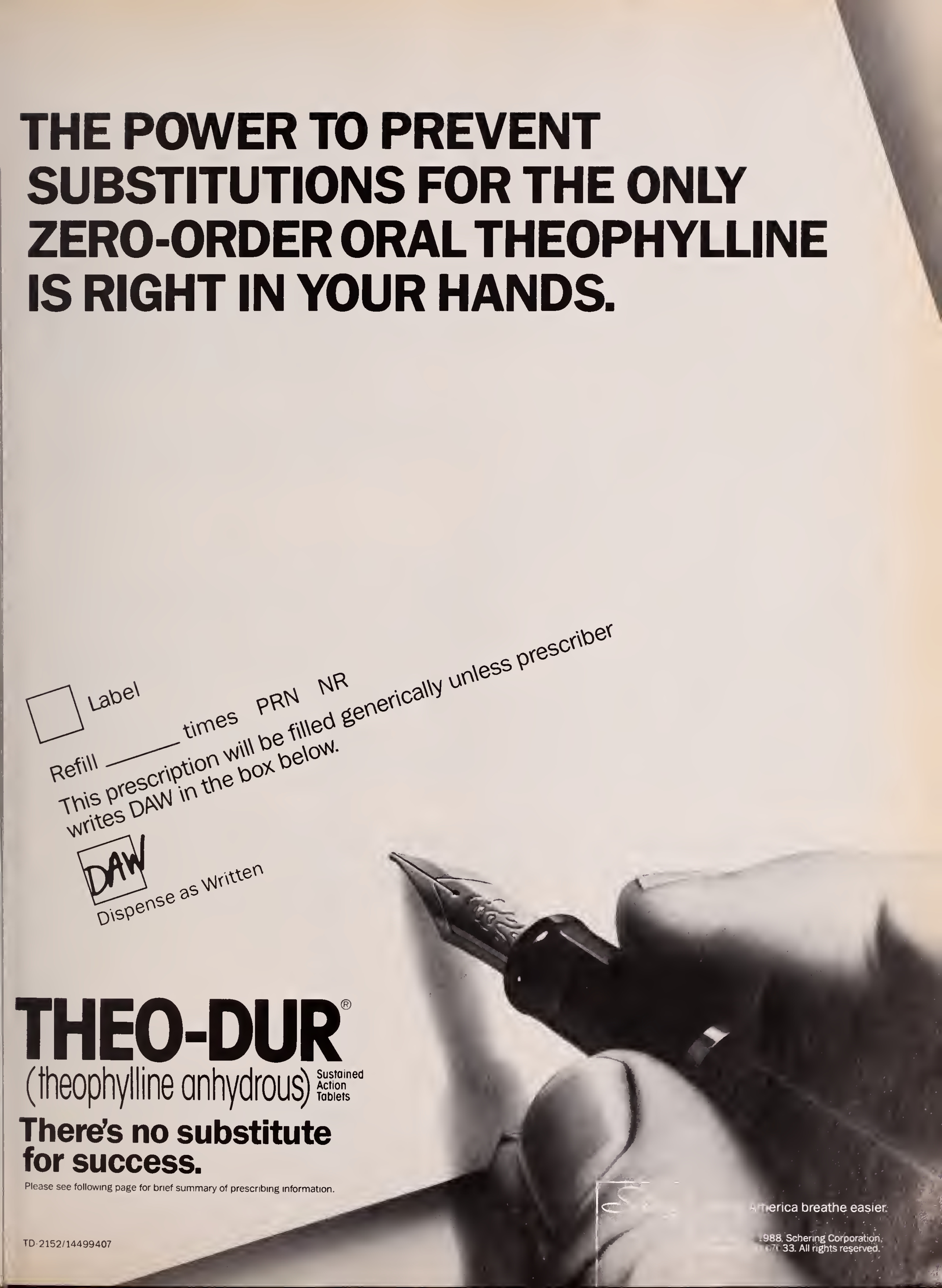

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INDICATIONS THEO-DUR is indicated for relief and/or prevention of symptoms of asthma and for reversible bronchospasm associated with chronic bronchitis and emphysema
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WARNINGS. Status asthmaticus should be considered a medical emergency and is defined as that degree of bronchospasm which is not rapidly responsive to usual doses of conventional bronchodilators Optimal therapy for such patients frequently requires both *additional medication*, parenterally administered, and close *monitoring*, preferably in an intensive care setting
Although increasing the dose of theophylline may bring about relief such treatment may be associated with toxicity The likelihood of such toxicity developing increases significantly when the serum theophylline concentration exceeds 20 mcg/ml Therefore, determination of serum theophylline levels is recommended to assure maximal benefit without excessive risk
Serum levels above 20 mcg/ml are rarely found after appropriate administration of recommended doses However, in individuals in whom theophylline plasma clearance is reduced *for any reason* even conventional doses may result in increased serum levels and potential toxicity Reduced theophylline clearance has been documented in the following readily identifiable groups 1) patients with impaired renal or liver function, 2) patients over 55 years of age, particularly males and those with chronic lung disease, 3) those with cardiac failure from any cause, 4) neonates, and 5) those patients taking certain drugs (macrolide antibiotics and cimetidine) Decreased clearance of theophylline may be associated with either influenza immunization or active infection with influenza
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Many patients who require theophylline may exhibit tachycardia due to their underlying disease process so that the cause/effect relationship to elevated serum theophylline concentrations may not be appreciated
Theophylline products may cause dysrhythmia and/or worsen pre-existing arrhythmias and any significant change in rate and/or rhythm warrants monitoring and further investigation
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PRECAUTIONS: THEO-DUR TABLETS SHOULD NOT BE CHEWED OR CRUSHED
General: Theophylline half-life is shorter in smokers than in non-smokers Therefore, smokers may require larger or more frequent doses Morphine and curare should be used with caution in patients with airway obstruction as they may suppress respiration and stimulate histamine release Alternative drugs should be used when possible Theophylline should not be administered concurrently with other xanthine medications. Use with caution in patients with severe cardiac disease, severe hypoxemia, hypertension, hyperthyroidism, acute myocardial injury, cor pulmonale, congestive heart failure, liver disease, in the elderly (especially males) and in neonates In particular, great caution should be used in giving theophylline to patients with congestive heart failure Frequently, such patients have markedly prolonged theophylline serum levels with theophylline persisting in serum for long periods following discontinuation of the drug Individuals who are rapid metabolizers of theophylline, such as the young, smokers, and some non-smoking adults, may not be suitable candidates for once-daily dosing These individuals will generally need to be dosed at 12 hour or sometimes 8 hour intervals Such patients may exhibit symptoms of bronchospasm near the end of a dosing interval, or may have wider peak-to-trough differences than desired
Use theophylline cautiously in patients with history of peptic ulcer Theophylline may occasionally act as a local irritant to the G I tract although gastrointestinal symptoms are more commonly centrally mediated and associated with serum drug concentrations over 20 mcg/ml
Information for Patients. The physician should reinforce the importance of taking only the prescribed dose and time interval between doses THEO-DUR tablets should not be chewed or crushed When dosing THEO-DUR on a once daily (q24h) basis, tablets should be taken whole and not split As with any controlled-release theophylline product, the patient should alert the physician if symptoms occur repeatedly, especially near the end of the dosing interval
DRUG INTERACTIONS Drug-Drug: Toxic synergism with ephedrine has been documented and may occur with some other sympathomimetic bronchodilators In addition the following drug interactions have been demonstrated

Drug	Effect
Theophylline with lithium carbonate	Increased excretion of lithium carbonate
Theophylline with propranolol	Antagonism of propranolol effect
Theophylline with cimetidine	Increased theophylline blood levels
Theophylline with troleandomycin, erythromycin	Increased theophylline blood levels

Drug-Food: *THEO-DUR 100 mg Sustained Action Tablets* have not been adequately studied to determine whether their bioavailability is altered when given with food Available data suggest that drug administration at the time of food ingestion may influence the absorption characteristics of theophylline controlled-release products resulting in serum values different from those found after administration in the fasting state
A drug-food effect, if any, would likely have its greatest clinical significance when high theophylline serum levels are being maintained and/or when large single doses (greater than 13 mg/kg or 900 mg) of a controlled-release theophylline product are given
THEO-DUR (200, 300, and 450 mg) Sustained Action Tablets The rate and extent of absorption of theophylline from THEO-DUR 200 mg, 300 mg, and 450 mg tablets when administered fasting or immediately after a moderately high fat content breakfast is similar
Drug-Laboratory Test Interactions: When plasma levels of theophylline are measured by spectrophotometric methods, coffee, tea, cola beverages, chocolate, and acetaminophen contribute falsely high values
Carcinogenesis, Mutagenesis, and Impairment of Fertility: Long-term animal studies have not been performed to evaluate the carcinogenic potential, mutagenic potential, or the effect on fertility of xanthine compounds
Pregnancy: Category C—Animal reproduction studies have not been conducted with theophylline It is not known whether theophylline can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity Xanthines should be given to a pregnant woman only if clearly needed
Nursing Mothers. It has been reported that theophylline distributes readily into breast milk and may cause adverse effects in the infant Caution must be used if prescribing xanthine to a mother who is nursing, taking into account the risk-benefit of this therapy
Pediatric Use: Safety and effectiveness of THEO-DUR administered
1 Every 24 hours in children under 12 years of age, have not been established
2 Every 12 hours in children under 6 years of age, have not been established
ADVERSE REACTIONS: The most consistent adverse reactions are usually due to overdose and are
1 *Gastrointestinal* nausea, vomiting, epigastric pain, hematemesis diarrhea
2 *Central nervous system* headaches, irritability, restlessness, insomnia, reflex hyperexcitability, muscle twitching clonic and tonic generalized convulsions
3 *Cardiovascular* palpitation, tachycardia, extrasystoles, flushing, hypotension, circulatory failure, ventricular arrhythmias
4 *Respiratory* tachypnea
5 *Renal* albuminuria, increased excretion of renal tubular and red blood cells, potentiation of diuresis
6 *Other* rash hyperglycemia and inappropriate ADH syndrome
OVERDOSAGE. Management: If potential oral overdose is established and seizure has not occurred
A Induce vomiting
B Administer a cathartic (this is particularly important if sustained-release preparations have been taken)
C Administer activated charcoal
If patient is having a seizure
A Establish an airway
B Administer oxygen
C Treat the seizure with intravenous diazepam 0 1 to 0 3 mg/kg up to 10 mg
D Monitor vital signs maintain blood pressure and provide adequate hydration
Post Seizure Coma.
A Maintain airway and oxygenation
B If a result of oral medication follow above recommendations to prevent absorption of the drug but intubation and lavage will have to be performed instead of inducing emesis, and the cathartic and charcoal will need to be introduced via a large bore gastric lavage tube
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"CASE MANAGEMENT AND CLINICAL CARE DILEMMAS"

FRIDAY, SEPTEMBER 15

8:30 a.m.–9:30 a.m.	Medical Technology, *Nassau A*
9:30 a.m.–12:30 p.m.	Gastroenterology/Colon & Rectal Surgery, *Sutton Center*
	Computers in Medicine, *Nassau A*
	Occupational & Environmental Health/Family Practice, *Murray Hill B*
	Physical Medicine & Rehabilitation, *Murray Hill A*
	Medical Liability Mutual Insurance Company, *Sutton South*
2:00 p.m.–5:00 p.m.	Chest Diseases/Radiology, *Sutton Center*
	Cardiovascular Diseases, *Sutton South*
	American Medical Women's Association/Orthopaedic Surgery Society, *Murray Hill A*
	Neurology, *Nassau A*
	Neurosurgery, *Murray Hill B*
	Emergency Medicine/Critical Care, *Nassau B*

SATURDAY, SEPTEMBER 16

8:30 a.m.–9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m.–12:30 p.m.	Substance Abuse, *Sutton North*
	Internal Medicine (All Day), *Sutton Center*
	Neurology/Ophthalmology (All Day), *Beekman Parlor*
	Psychiatry (All Day), *Nassau B*
	Obstetrics & Gynecology/Urology, *Murray Hill A*
	General Surgery, *Sutton South*
	Dermatology/Plastic Reconstructive Surgery, *Nassau A*
	Allergy & Immunology, *Sutton North*
2:00 p.m.–5:00 p.m.	Internal Medicine (All Day), *Sutton Center*
	Neurology/Ophthalmology (All Day), *Beekman Parlor*
	Psychiatry (All Day), *Nassau B*
	Pediatrics/Forensic Medicine, *Sutton North*
	Otolaryngology/Neurosurgery/Plastic Reconstructive Surgery, *Sutton South*
	Plastic Surgery, *Regent Parlor*

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Technical Exhibits

NEW YORK STATE
JOURNAL OF MEDICINE

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COMMENTARIES

Mandatory Medicare assignment

The Medical Society of the State of New York (MSSNY) prevailed in 1987 and 1988 in its efforts to prevent the imposition of mandatory Medicare assignment. However, the struggle is far from over. The New York Statewide Senior Council has made mandatory Medicare assignment a high legislative objective for 1989.

If Massachusetts Governor Michael Dukakis had been elected to the presidency, the issue may very well have been settled at the national level, even though Congress has already rejected mandatory assignment. Now, it is likely that each state will have to deal with the issue.

Physicians in New York State and the Medical Society of the State of New York have mounted a very strong campaign against this legislation. Our opposition involves two levels. First, we have attacked the bill itself as inequitable to physicians, nonresponsive to legitimate senior citizen needs, and possibly extremely counterproductive to the extent that it will exacerbate system access rather than resolve it. We have steadfastly opposed any linkage to licensure.

We have also pointed out the already high rate of assignment acceptance (80%) by New York State physicians. We have actively pursued a voluntary assignment acceptance program: our "VAAP" program, for which more than 16,000 physicians have signed up. We have reiterated our commitment to assuring that no senior is denied care because of financial difficulty. We have offered to work with seniors to address the real problems with Medicare coverage which clearly include the areas of catastrophic illness and long term institutional coverage.

We have tried to point out the rigorous statutory and regulatory physician fee controls already present in Medicare at the federal level. We have pointed out that this is a complex federal program and that modifying it on a state by state basis is bound to aggravate an already confusing bureaucratic process.

We have repeatedly asked seniors, who justify assignment at least partially on the basis of inadequate financial means, why they adamantly refuse to consider a means test. We have pointed out that the new catastrophic program under Medicare contains a means test, albeit one based on premium payments.

Whatever the merit of the argument that Medicare is not adequately responsive to senior needs (and we agree much must be done to improve the system), we will continue to resist the attempt represented by mandatory assignment to simply shift the seniors' problems with Medicare to physicians. We at the MSSNY are regularly meeting with all of the involved parties. These include not only representatives of senior groups and involved government officials, but other interested groups as well. Recently several meetings were held with representatives of Governor Cuomo. We continue to try to resolve this problem.

The danger of mandatory assignment looms great in 1989. New York State is in very serious financial difficulty. Major state and federal tax reform has produced a potential budget shortfall as high as $2 billion. Obviously there is not going to be a great deal of public money for Governor Cuomo to put into public programs in 1989. He is up for re-election in 1990. Mandatory assignment legislation, if he can produce it for the seniors, gives a huge voting bloc its number one priority without costing the state one nickel! This is truly a politician's dream. Furthermore, the major leadership changes in the state senate, with the retirement of Senate Majority Leader Warren Anderson, raise many questions about the role that we can expect that body to take in blocking mandatory assignment. However, the change in senate leadership provides us with a great opportunity for developing new and stronger relationships with the senate.

In 1988 the senate chose not to take up mandatory assignment. However, the senate did prepare a bill that might have been introduced if the assembly had passed its bill. The draft senate bill contained an elaborate "means test" which in all likelihood would have been rejected by seniors.

The 1989 session of the legislature convened during the first week of January. Before it concludes, more than 10,000 legislative proposals will have been introduced. A very large number of these, perhaps 10% or more, will involve health issues. Medical liability, mandatory assignment, Medicaid, workers' compensation, private insurance coverages, the allied health professions, infectious waste, AIDS, relicensure of physicians, and quality assurance initiatives are but some of the many issues that will be discussed in the senate and in the assembly and in the governor's office. Additionally, the state departments of social services, insurance, and, of course, health will be acting every day on issues that affect the professional lives of physicians.

Physicians must become involved both organizationally and individually. Basic changes in the profession are occurring within the arena of government. Moreover, the great majority of legislative and regulatory issues are not specialty-specific. They affect the entire profession.

At the direction of the MSSNY's leadership, the Division of Governmental Affairs has organized a group of specialty society lobbyists to work together in Albany on the issues that confront all of us. We are truly encouraged by this new joint undertaking. This group will continue to meet on a regular basis throughout the session to enhance communication and orchestrate strategy. Mandatory Medicare assignment is among the most important issues being dealt with by this new group.

We must not let those who do not share our commitment to an enhanced health care system divide us, pick us apart, and, having done so, overwhelm us with their own agendas. We must remain united in these difficult times. It is well to remember that "tough times do not last, but tough people do!"

DONALD F. FOY
Executive Vice-President

GERARD L. CONWAY, JD
Director
Division of Governmental Affairs
Medical Society of the State of New York
Lake Success, NY 11042

The tale of Ondine: A curse, a kiss, a clasp, and a comment

Ancient as well as modern fables and myths have enchanted readers, each story finding a way to offer a moral, or a touch of humor as the narrative draws to a close. Amidst the fantasy of such tales, however, a shimmer of reality sometimes comes through. A common and intriguing example of physiologic reality is found in the descriptions of altered patterns of both breathing and sleep.

The giant in "Jack in the Beanstalk" has been well described as the prototype of the sufferer of an obstructive breathing disorder with obesity-hypoventilation.[1] Between his shouts of "Fee-Fi-Fo-Fum," the grouchy and grossly obese giant would spend his time sleeping, snoring, and rocking in a somnolent state. In contrast, Snow White, who suffered from a more prolonged somnolence, was tricked into eating an apple laced with poison. She awoke only when a prince kissed her as she coughed out a piece of retained apple.[2] While a part of the picture would seem to be a reversal of airway obstruction, the malignant narcotic effects of the fruit are clearly attributed to the occult poison. A barbiturate with resultant coma would resemble death and could possibly be the offending drug.

Sleeping Beauty drifted into a similar comatose state after pricking her finger on a spindle and slept for 100 years, until the curse expired as a rescuing prince appeared to awaken her.[3] Rip Van Winkle, a "thirsty soul," also had difficulty with prolonged sleep. He quaffed an excessive amount from a keg of liquor and slept for 20 years in an alcoholic stupor, albeit of prodigious duration.[4] And, more recently, Joe, the wonderfully fat boy of *The Pickwick Papers*, so typified the obese patient with sleep apnea and hypoventilation that he gave rise to the term "Pickwickian" to describe that special type of sleep disorder.[5,6]

Of all the charming stories relevant to sleep and breathing, the most intriguing may still be the story of the water-nymph, Ondine, and the vivid curse that bears her name. The legend of Ondine has been traced back as far as Paracelsus, where a female water spirit, the Undine, acquired a soul through marriage. (Some versions also required the Undine to bear a child to obtain a soul.) The tale was picked up and modified by de la Motte-Fouque into a story, "Undine," which was so popular that it was turned into an opera, a ballet, and subsequent prose works by other authors. These tell of the mermaid, or Undine, daughter of a water prince, who marries a mortal, the questing knight, Count Huldbrand. The count brings her to his castle but then rekindles a relationship with his former lover, Bertalda, and rejects Undine. While Bertalda is sailing on the Danube, a water spirit snatches a necklace from her which had been a gift from the count. Undine also surfaces from the river and offers a more beautiful necklace to the count. Huldbrand, in anger, curses Undine, commands her to return to the water, and then marries Bertalda, believing himself rid of Undine. At their wedding ceremony, when Bertalda opens a water fountain, Undine comes forth and Huldrand is reminded of his faithlessness. He dies with Undine's "kiss" on his lips. The play version of Giraudoux portrays Ondine's father, the king of the water spirits, as requiring the death of Hans (Huldbrand) for his faithlessness. The "curse" of Hans means that during one moment of inattention, he would forget to see, move, hear, or breathe. He "died because breathing bored him."[7] A final version of the tale of Ondine includes her embrace of this mortal man, fatally squeezing his last breath away.[8,9]

Several authors of articles on Ondine's curse have erred as to her sex, occupation, subject of the curse, or details of the myth. Poor Ondine was even blamed for having uttered the curse or committing the infidelity.[7] It is no wonder, with all the confusion, that one commentator has, in jest, recommended that the eponym be applied most correctly to "death from vigorous kissing and clasping, preferably by a nymph."[10]

Physicians, too, have found another use for this tale of the sudden cessation of breathing. The first application of the eponym "Ondine's curse" in the medical literature was in 1962, when Severinghaus and Mitchell[11] described three patients with prolonged periods of apnea even when awake, who nevertheless retained the capability of hyperventilating on command. In each of these cases, the onset of the disorder involved surgery to the high cervical spinal cord or brain stem. Artificial respiration was needed, as sleep apnea occurred in all. One patient ultimately died in apnea and two survived. Damage to the medullary carbon dioxide chemoreceptors was suspected.[11]

Normal autonomic and voluntary control of respiration is mediated by two separate neural mechanisms. These interconnected impulses are regulated by a series of positive and negative feedback loops which innervate the inspiratory muscles of respiration while inhibiting the expiratory muscles, and vice versa.

Autonomic respiration is stimulated by a rhythmic discharge of neurons in the medullary pattern generator (the respiratory center). This discharge is spontaneous, but modified by chemical stimuli such as hypercapnea via increased hydrogen ion concentration in the cerebral spinal fluid, and by hypoxemia or acidemia via the carotid and aortic bodies. Non-chemical stimuli may also affect breathing via afferents from inflation and deflation lung receptors, via afferents for sneezing, coughing, swallowing, and yawning, and from arterial, atrial, ventricular, and pulmonary baro-receptors.

The respiratory center comprises at least two groups of inspiratory neurons, both located in the medulla. Dorsomedially in the nucleus tractus solitarius (NTS) is the set which supplies the rhythmic drive to the contralateral phrenic neurons; and ventrally and laterally is the second group comprising two divisions. One division seems to be mainly concerned with laryngeal and bronchial skeletal muscle activity as well as bronchomotor control. The second division is near the nucleus retroambiguous (NRA) and drives the intercostal and expiratory muscles. It appears as if the NTS sets the respiratory pattern as well as drives the diaphragms, as these neurons send axons to the NRA; but no axons connect the NRA to the NTS. Neurons in the nucleus parabrachialis medialis (NPBM) of the pons also have a regulatory effect on neuron thresholds and are involved with the characteristic prolonged inspiratory pauses of apneustic breathing. This effect is overcome with time, suggesting that the NPBM is not essential for the genesis of phasic respiratory activity.

Voluntary breathing, especially necessary in those patients afflicted with Ondine's curse, is regulated by neurons located in the cerebral cortex, sending impulses directly to the respiratory motor neurons via the corticospinal tracts.[12,13]

Although the initial descriptions of Ondine's curse included mostly adults with a form of alveolar hypoventilation after birth, the term was soon expanded to include the pediatric age group with a report of alveolar hypoventilation from a congenital defect in the central control of automatic respiration.[14] Despite the alveolar hypoventilation while asleep, these patients' arterial blood gas levels were normal while they were awake. With reduced responsiveness to inhaled carbon dioxide, retention of carbon dioxide ensued.

Idiopathic hypoventilation has also been associated with aganglionosis of the colon (Hirschsprung disease). A developmental deficit of stem serotonergic nerve cells in these regions of the gastrointestinal plexus and the lower brain stem has been proposed as the causative factor.[15,16]

An additional case of brainstem dysfunction manifesting as Ondine's curse followed a near drowning episode. Postmortem neuropathologic findings one year after near drowning demonstrated only brain stem defects in the nucleus ambiguous, nucleus retroambiguous, and the nucleus of the solitary tract.[17] Finally, in addition to bilateral medullary damage to the respiratory neurons, a unilateral medullary infarct may acutely compromise automatic respiration as well.[18] Accordingly, it would seem that Ondine's curse may arise from such disparate causes as brain stem trauma, surgery, hypoxia-ischemia, infarction, or, rarely, as a congenital phenomenon.

Although breathing is normally an automatic process with impulses from the brainstem, the cortex has the capacity to override these respiratory centers when voluntary control is wanted. Breath holding, so common in infants below the age of two years, or hyperventilation are examples of such voluntary control. A form of forced hyperventilation before submergence in swimming is also well known and may lead to apneic episodes with sometimes tragic consequences. The body depletes carbon dioxide by rapid respiration and while the swimmer is underwater for a period of time, hypoxia may cause loss of consciousness before the respiratory center is able to intervene to trigger a breath. Ondine's curse, in contrast, is a case of a loss of automatic breathing. Although some authors have classified Ondine's curse as a primary or idiopathic hypoventilation syndrome,[19,20] this is not so clearly the case.

Alveolar hypoventilation encompasses a number of different disorders which result in an elevation of the arterial carbon dioxide blood gas tension. Decreased peripheral chemoreceptor drive, peripheral motor neuron disease, muscular diseases, chest wall deformities, and obstructive airway disease are principal causes of alveolar hypoventilation. A decreased central drive may also cause alveolar hypoventilation and may be associated with drugs (sedatives, barbiturates), metabolic alkalosis, myxedema, and neurologic disease (encephalitis).[21] Over the past decade, descriptions of patients with obesity-hypoventilation and sleep apnea have been added to the list of disturbances responsible for hypercapnea. When the etiology of alveolar hypoventilation is not clear, and pulmonary function studies are normal, the term primary alveolar hypoventilation is commonly used.

In this issue of the *Journal*, Colp and associates[20] rekindle our thoughts concerning Ondine's curse. A 69-year-old patient is thought to have a defect in central respiratory drive following abdominal surgery, recurrent pneumonias, and prolonged ventilator care. Although the causative mechanism for this breathing disturbance is not clear, hypoxia, transient ischemia, or hypotension may have resulted in limited compromise to the central medullary center responsible for automatic breathing. The patient demonstrated hypoventilation and hypercapnea, although the presence of obstructive sleep apnea afterwards may have led to his carbon dioxide retention. In their discussion, the authors allude to the fact that Ondine's curse may be synonymous with idiopathic hypoventilation. We believe, however, that the two terms, Ondine's curse and idiopathic pulmonary hypoventilation, are most likely separate entities.

It would seem that Ondine's curse, with decreased central drive, is a subset of the larger disorder of central alveolar hypoventilation. In Ondine's curse, respiratory drive and arterial blood gases are essentially normal while the patient is awake, and the patient can hyperventilate on command if periods of apnea are present. During sleep, hypoventilation and apnea occur from impaired automatic brainstem respiratory control, mechanisms which otherwise allow for spontaneous breathing during sleep. Patients with primary alveolar hypoventilation, in contrast to those with Ondine's curse, manifest chronic alveolar hypoventilation during the day, which is exaggerated at night. By the time this disorder is detected, the clinical manifestations of chronic hypoxemia and even polycythemia are already evident.

We applaud Dr Colp and coworkers for treading into the wilderness of confusion surrounding the use of the eponym of Ondine's curse. We should try, however, to encourage appropriate restriction of the reference to this somewhat maligned and misused nymph in so many different conditions.

Whether Ondine kissed or clasped her husband to death depends on the version of the tale, and one can never know who cursed whom. Any good myth or fable has its point or moral, and Ondine's story certainly revolves around the concept of faithfulness. We, as physicians, should try to assure fidelity to the myth, Ondine's good name and reputation, and apply the eponym of Ondine's curse only to those features with which it was initially described, namely as a failure of automatic respiration during sleep. Let us not hold our breath waiting for exactitude and constancy to be applied to this condition.

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Coronary care in New York City hospitals

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ABSTRACT. New York City, with six medical schools and an elaborate emergency medical system, would appear to be a locale with optimal resources for coronary care. To assess coronary care facilities and practices, a comprehensive questionnaire was prepared by a multidisciplinary committee and submitted to 72 hospitals. Seventy-nine percent (57/72) responded, including all medical school affiliated hospitals and 41 of the 45 with more than 300 beds. Twenty-nine percent (16/55) did not have a coronary care unit separate from a general intensive care unit. Thirty-three percent (18/55) of coronary care units were designed before 1970. Although 85% (46/54) of the coronary care units had a 1:2 registered nurse per patient ratio during days, only 43% (23/54) maintained this ratio at night. Central monitoring was available in all hospitals, but only 40% (21/52) indicated 24-hour continuous monitor observation. Forty-five percent (25/55) had cardiac catheterization facilities, and 43% (24/56) had an intra-aortic balloon available. Forty-six percent (26/57) of coronary care units transferred post-myocardial infarction patients to non-monitored general beds. Fifty-eight percent (31/53) offered dietary counseling to patients, and 33% (18/54) had an inpatient cardiac rehabilitation program. Thus, New York City hospitals demonstrate a marked variability in coronary care unit facilities and practices and a substantial number of hospitals deviate from published recommendations for coronary care units.

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The development of closed-chest techniques for cardiopulmonary resuscitation heralded the contemporary era of coronary care.[1] Because the outcome of resuscitative efforts is exquisitely time dependent, mobile life support systems, heart care stations, and intensive care units have been developed for the expeditious diagnosis, treatment, and prevention of catastrophic coronary events. Although the original goals of such medical care systems were conceptually simple, monitoring patients and treating arrhythmias,[2] advances in medical technology and science have led to a substantial broadening of their purview. Hemodynamic measurements, coronary thrombolysis, and attempts at limiting myocardial injury now fall within the scope of the coronary care unit.[3] Computerized monitoring systems, highly specialized nurse practitioners and technologists, and comprehensive health care teams are also available in many hospitals for the treatment and rehabilitation of patients with coronary disease. These changes have not, however, been made without criticism. Overuse of expensive technology, unnecessary procedures and testing, the use of still unproven treatments, as well as the impersonal and insensitive care that a technology-oriented environment might engender are concerns that have been raised.[4] Despite differences in perceptions of the value of these changes, consensus recommendations for optimal care have been developed.

New York City, with six medical schools and an elaborate emergency medical system, would appear to be a locale with optimal resources for coronary care. To determine whether coronary care facilities and practices in this city are indeed consistent with consensus recommendations, a multidisciplinary committee was established by the American Heart Association, New York City Affiliate (AHA NYCA). The committee prepared a comprehensive questionnaire, based in part on the recommendations of the Joint Commission on Accreditation of Hospitals (JCAH)[5] and those of the Coronary Heart Disease Study Group of the Intersociety Commission for Heart Disease Resources (ICHDR),[6] and submitted it to 72 hospitals. This article reports the major findings of the survey. (Further details may be obtained from the American Heart Association, New York City Affiliate.)

METHODS

The AHA NYCA, as part of a long range goal "to assess current standards for treatment of coronary heart disease," established a coronary care committee. This multidisciplinary group comprised board-certified cardiologists, a physician with expertise in preventive and rehabilitative cardiology, a coronary care unit nurse, a nutritionist, a bioengineer, a health care administrator, and an epidemiologist. To accomplish its task of determining the state of coronary care facilities and practices in New York City, the committee designed a comprehensive questionnaire.

Questionnaire. The questionnaire was divided into five parts: bioengineering, medical policies and practices, nursing, nutrition, and cardiac rehabilitation. Members of the committee prepared questions in their area of expertise using the recommenda-

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This paper is based on a survey by the Coronary Care Committee of the American Heart Association, New York City Affiliate.

tions of the JCAH and those of the ICHDR as a guide. All questions were reviewed by the entire committee. A medical sociologist with expertise in the questionnaire format and an epidemiologist were consulted. The questionnaire was trial-tested in committee members' hospitals. The questionnaire in its final form was separated into five parts, each having its own colored paper, and consisting of 1,048 questions.

Distribution. The questionnaire was sent to the 72 New York City hospitals listed in *Health Facilities in Southern New York.*[7] Of the 72 hospitals, 46 had been designated as Emergency Heart Care Stations (EHCS). Hospitals having this designation had voluntarily met the standards of the AHA NYCA for emergency cardiac care and had received site visits by members to certify that those standards were being met.

The questionnaires were mailed to the coronary care unit director of each hospital. An accompanying letter requested that each section be completed by those persons in the hospital best able to do so. The chief of cardiology was advised of the questionnaire and was asked to ensure that it was completed. To acquaint hospitals with the purpose of the questionnaire and to assist them in completing it, a conference was held, which was attended by representatives of 31 New York City hospitals.

Response. The questionnaires were sent to the hospitals in October 1983; January 1, 1985, was set as the deadline for return of completed questionnaires. Twenty-three hospitals completed the questionnaire in response to the original mailing. To obtain a larger sample, letters and telephone calls by members of the committee were made to the coronary care unit directors requesting return of the completed questionnaire. At the cut-off date, 58 of the 72 hospitals had returned the questionnaire. However, one questionnaire was deemed "unusable," and one, although completed, did not identify the hospital. The responses of this questionnaire were included in the tabulation of data, but not in the comparative analyses. Thus, 79% (57 of 72) of the hospitals completed the questionnaire. Table I shows the distribution of the responding hospitals by bed number, hospital type, and EHCS certification status.

RESULTS

Biomedical Engineering. Seventy-one percent (39/55) of the coronary care units (CCUs) were freestanding units, that is, not part of a larger intensive care unit (ICU). Fifteen percent (8/55) were located in a surgical ICU and 16% (9/55) in a medical ICU. Sixty-eight percent (37/55) of the CCUs were designed between 1966 and 1980, with 33% (18/55) having been designed between 1966 and 1970. Twenty percent (11/55) of the CCUs had undergone renovations that required changes in building structure since their original construction, while 40% (22/55) of responding hospitals planned to renovate their units. The number of beds in units that served only as a CCU is shown in Table II. In 53% (29/55) of the CCUs the nurse could view all patients directly from a nurses' station. Closed circuit television was used for patient observation in 35% (19/55), in lieu of direct patient observation.

TABLE I. Hospitals Responding to the Survey

	No. of Hospitals	Percent
Size		
Number of beds		
<300	15	27
≥300 and <600	20	36
≥600	21	37
Total	56	100
Type		
Municipal*	11	20
University†	7	12
Non-university voluntary	32	57
Proprietary‡	4	7
Veterans‡	2	4
Total	56	100
Emergency heart care station certification		
Emergency heart care station	42	75
Nonemergency heart care station	14	25
Total	56	100

* Member of New York City Health and Hospital Corporation.

† A major teaching hospital of one of the six New York City medical schools.

‡ Not used in comparative analysis because of insufficient numbers.

TABLE II. Coronary Care Unit Beds and Services

Coronary Care Unit Beds

1-3 beds	4-6 beds	7-9 beds	10-12 beds	>12 beds
2% (1/41)	32% (13/41)	34% (14/41)	17% (7/41)	15% (6/41)

Cardiovascular Services for Coronary Care Unit

Services	Available	Available 24 hours a day
Fluoroscopy	76% (42/55)	58% (32/55)
Cardiac catheterization	45% (25/55)	29% (16/55)
Echocardiography	82% (45/55)	36% (20/55)
Nuclear medicine	80% (44/55)	18% (10/55)
Cardiac surgery	24% (13/55)	22% (12/55)

The cardiovascular services available to CCU patients are shown in Table II. Sixty percent (33/55) of the respondents had a room for invasive procedures in or near the CCU. Fluoroscopy could be performed in the CCU itself in 53% (29/55) of the facilities, x-ray in 85% (47/55), echocardiographic examinations in 62% (34/55), and nuclear medicine scans in 25% (14/55). In 87% (48/55) of the units there was a monitor permanently assigned to each bed. Of these monitors, 58% (28/55) were equipped with strip chart recorders. Central station monitoring was present in 98% (54/56) of the units.

Medical Practices and Policies. ***Admission Policies.*** Ninety-six percent (55/57) of hospitals listed specific criteria for admission to the CCU. The criteria are shown in Table III. As is also shown in Table III, the medical house staff was responsible for approving ad-

TABLE III. Admissions by Diagnostic Criteria, Approval, and Killip Classification

Admission Diagnostic Criteria	No. of Responding Hospitals	Percent Yes	Percent No
Documented or suspected acute MI	55	100	0
Unstable angina pectoris	54	100	0
Cardiac arrhythmias	55	98	2
Pacemaker malfunction	53	96	6
Patients requiring hemodynamic monitoring	50	94	6
Pulmonary edema	51	84	16
Elective cardioversions	54	80	20
Post-cardiac arrest	54	94	6
Patients on respirators	49	39	61

Approval of Admissions	Approves Routine Admissions*	Ultimate Authorization for Admission
Intern	0	0
Resident	41	7
Cardiac fellow	18	6
Service attending	10	9
Private attending	8	3
Director	15	39

Distribution of Admissions to CCU by Killip Classification

Killip Class	No. of Hospitals Responding†	% Patients 0–25%	26–50%	51–75%	76–100%
I	54	6	26	20	2
II	56	18	34	4	0
III	55	48	7	0	0
IV	54	53	1	0	0

* In some hospitals approval for admission could be made by more than one individual.

† Two hospitals did not indicate numbers for Killip I and II and one did not for Killip III.

missions to the cardiac care unit in most hospitals; however, only 14% of responders (8/57) indicated that the private attendings could approve routine admission on their own, and 5% (3/57) listed the private doctor as having ultimate authority for admission to the unit. Table III also shows the distribution of patients with acute myocardial infarction by Killip classification. Of admissions to the unit, the majority were either Killip Class I or Killip Class II.

Fifty-one percent (29/57) of the hospitals indicated that when CCU beds were full, patients with acute myocardial infarction could be sent to a medical intensive care unit, 42% (24/57) to a combined medical/surgical intensive care unit, 16% (9/57) to a progressive or intermediate cardiac care unit, 12% (7/57) to a respiratory care unit, 24% (14/57) to a monitored bed on the general medical floor, and 42% (24/57) to a holding area. Only one hospital admitted patients with acute myocardial infarctions to non-monitored beds on a general medical ward (2%). Twenty-eight percent (16/57) of hospitals transferred patients with acute myocardial infarction to other hospitals, whereas 70% (40/57) did not.

Duration of stay and transfers from CCU. Lengths of stay in the CCU for patients admitted with an acute or suspected myocardial infarction are shown in Table IV. Forty percent (23/57) of hospitals reported that on discharge from the CCU, patients were transferred to a progressive or intermediate care unit; 14% (8/57) to a monitored bed on a general medical floor; and 46% (26/57) to non-monitored beds on a general medical floor.

Standing orders. Sixty-eight percent (39/57) of hospitals had standing orders for patients admitted to the CCU. Table V presents activities permitted in the CCU and the diagnostic tests that were part of the standing orders in these 39 hospitals. The following were the routine medications ordered: 85% (33/39) stool softeners; 59% (23/29) tranquilizers such as diazepam; 56% (22/39) sleep medications; and 87% (34/39) nasal oxygen. Only 10% (4/39) of the hospitals administered intravenous (IV) heparin on their standing orders, while an additional 41% (16/39) had standing orders for subcutaneous heparin. Five percent (2/39) of hospitals had standing orders for sodium warfarin.

Medications. Eighty-four percent (48/57) of the hospitals used prophylactic lidocaine. This survey, however, did not discriminate whether the lidocaine was given only to high-risk patients or to all patients with suspected myocardial infarction.

For an uncomplicated myocardial infarction, 56% (32/57) of the hospitals routinely administered heparin, whereas 44% did not. Most of the hospitals that administered heparin gave it subcutaneously; 51% (29/57) gave routine subcutaneous heparin. Of the 25 hospitals that did not routinely administer heparin, 52% (13/25) gave heparin to patients with either large anterior wall myocardial infarctions or myocardial infarctions complicated by congestive heart failure.

Availability of Laboratory Tests. *Isoenzymes.* Creatine kinase isoenzyme determinations were available in all institutions responding to this question (56/56), while lactic dehydrogenase isoenzymes were available in 84% (48/56). Isoenzymes were performed on a routine basis in 36% (20/56) hospitals, only if these enzymes were elevated in 55% (31/56), and only if specifically requested in 9% (5/56).

TABLE IV. Duration of Stay

Type of Patient	No. of Hospitals Responding	Median Length of Stay (Days)	Actual Length of Stay (Days)			
			3	4	5	5+
Uncomplicated non Q-wave myocardial infarction	57	3	34	11	11	1
Uncomplicated Q-wave myocardial infarction	57	4	17	20	18	2
Stable patient with chest pain, but without myocardial infarction	57	4	12	22	20	3

TABLE V. Standing Orders

	No. of Hospitals Responding	Percent Yes	Percent No
Activities			
Chair rest when stable	39	74	26
Feed self	39	77	23
Wash self	39	54	46
Bedside commode	39	74	26
Stand to void	37	43	57
Antiembolic stockings	38	39	61
Limitation on visitors	39	90	10
Diagnostic tests			
Complete blood count	39	100	0
Erythrocyte sedimentation rate	39	54	46
Urinalysis	39	97	3
SMA-18 chemistry screen	39	100	0
Cardiac isoenzymes	39	100	0
Prothrombin time	39	85	15
Partial thromboplastin time	39	71	29
Arterial blood gases	38	39	61
Chest x-ray	38	100	0
Electrocardiogram	38	100	0

Serum drug concentrations. Serum drug concentrations were available for lidocaine in 70% (38/54), for quinidine in 96% (54/56), for procainamide in 96% (54/56), and for digoxin in 100% (56/56) of respondents.

Waiting time for results. Waiting times for obtaining laboratory results are summarized in Table VI. Waiting times for results of enzymes and isoenzymes tended to be considerably longer on weekends and holidays.

Special Procedures. Primary responsibility for invasive procedures is shown in Table VII. Temporary pacemakers and Swan-Ganz catheters were inserted mainly by cardiology fellows, arterial lines mainly by medical residents, and intra-aortic balloon pumps by cardiology attendings.

Temporary pacemakers. Forty percent (23/50) of hospitals responding routinely used fluoroscopy for temporary pacemaker insertion. The subclavian vein was the most commonly used route in 58% (32/55) of hospitals, the internal jugular in 15% (8/55), the femoral vein in 7% (4/55), and the antecubital vein in 2% (1/55). In 18% (10/55) of hospitals two or more routes were used with equal frequency.

In patients with an acute anterior wall myocardial infarction, the presence of a new right bundle branch block pattern (RBBB) and either left anterior or posterior fascicular block pattern was considered an indication for prophylactic pacing by 98% (54/55) of those hospitals responding. Pacing was indicated in the presence of a new RBBB in 59% (29/49) of hospitals, of a new left bundle branch block pattern (LBBB) in 71% (37/52) of hospitals, and of a new bundle branch block pattern and first degree atrioventricular (A-V) block in 92% (49/53) of hospitals.

In stable patients with inferior wall myocardial infarction none of the 54 hospitals responding considered Mobitz I A-V block an indication for prophylactic pacing. In contrast, 58% (32/55) of hospitals considered Mobitz II A-V block an indication for prophylactic pacing, and 51% (28/55) of hospitals considered third degree A-V block an indication for prophylactic pacing in stable patients with acute inferior myocardial infarction.

Hemodynamic monitoring. Sixty-five percent (36/55) of hospitals responding indicated there was sufficient equipment for every patient to undergo hemodynamic monitoring with a Swan-Ganz catheter. The main indications for insertion of Swan-Ganz catheters in patients with acute myocardial infarction included hypotension or

TABLE VI. Waiting Time for Results of Serum Enzyme Determinations and Drug Concentrations for Blood Drawn on Weekdays vs Weekends and Holidays

Serum Enzymes/ Drug Concentrations	No. of Hospitals Responding	Median Waiting Time (hr)	Waiting Time (hr)				
			0–6	7–12	13–24	25–48	48+
Weekdays							
Creatine kinase (total)	56	9.5	21	14	18	3	—
Creatine kinase (isoenzymes)	56	14.8	10	15	18	10	3
Serum glutamic oxaloacetic transaminase	56	9.8	19	16	19	2	—
Lactic dehydrogenase (total)	55	9.8	19	15	19	2	—
Lactic dehydrogenase (isoenzymes)	47	19.3	7	9	13	13	5
Lidocaine	37	31.2	4	4	7	13	9
Procainamide	53	34.6	5	6	8	18	16
Quinidine	54	33.8	5	6	8	21	14
Digoxin	56	26.2	7	7	13	20	9
Weekends/Holidays							
Creatine kinase (total)	56	15.4	13	13	9	9	12
Creatine kinase (isoenzymes)	52	26.4	3	8	14	16	21
Serum glutamic oxaloacetic transaminase	55	19.2	10	13	8	12	12
Lactic dehydrogenase (total)	55	19.2	2	13	8	11	13
Lactic dehydrogenase (isoenzymes)	49	48+		5	1	16	25

cardiogenic shock, 95% (53/56); acute mitral regurgitation or ventricular septal defect, 91% (51/56); severe congestive heart failure, 88% (49/56); and suspected right ventricular infarction 77% (43/56). Mild heart failure (Killip II) was an indication in 16% (9/56), uncomplicated large anterior wall in 4% (4/56), most anterior wall MIs in 2%, and most MIs in 2%.

The maximum number of days that a Swan-Ganz catheter was permitted to remain in a patient before being removed or replaced varied. Of the 53 hospitals responding, two allowed a maximum of two days, 17 allowed three days, 15 allowed four days, and 19 permitted five or more days.

Intra-aortic balloon pump. Forty-three percent (24/56) of hospitals responding had an intra-aortic balloon pump in their CCU. Ninety-two percent (22/24) used the percutaneous approach, and 8% (2/24) used a surgical approach as the preferred method for insertion of the intra-aortic balloon pump. Ninety-two percent (22/24) of hospitals considered ischemic pain unresponsive to medical therapy, acute mitral regurgitation, or ventricular septal defect complicating acute myocardial infarction to be indications for intra-aortic balloon insertion. Eight-eight percent (21/24) considered cardiogenic shock an indication for its insertion.

Miscellaneous Data. Therapy with intracoronary streptokinase was available in 67% (18/27) of hospitals responding, and percutaneous transluminal coronary angioplasty in 48% (13/27). (Intravenous thrombolytic therapy had not yet been approved for use in acute myocardial infarction at the time the survey was completed.)

Record Keeping. Seventy-six percent (41/54) of hospitals had a method for recording statistics in their CCUs. Eight percent (4/50) used a computer to store data.

Nursing. ***Monitoring.*** Although 100% (53/53) of the respondent CCUs and 90% (18/20) of the post coronary care units (PCUs) had 24-hour central monitoring capabilities, only 40% (21/53) of the CCUs and 33% (6/18) of the PCUs had someone physically present at the central monitor 24 hours a day. In 93% (41/44) of the CCUs and 94% (15/16) of the PCUs, monitor observers were assigned other responsibilities including answering telephones, charting, and supervision.

Staffing. Most of the CCUs met the standard of a 1:2 or a 1:3 nurse/patient ratio. However, the ratio appeared to be adjusted by shift hours rather than by patient needs (Table VIII). Of the respondent institutions, only 51% (26/51) of the CCUs met the standard of 12 direct registered nurse (RN) care hours per patient day (Table IX).

On shifts when the RN-to-patient ratio decreased, the expectation

TABLE VII. Primary Responsibility for Invasive Procedures

Procedure	Total No. Responding	Cardiology Fellow	Medical Resident	Full-time Cardiologist	Voluntary Attending	Cardiac Surgeon
Temporary pacemaker	56	31 (55%)	1 (2%)	16 (29%)	8 (14%)	
Swan-Ganz catheter	53	28 (53%)	7 (13%)	13 (25%)	5 (9%)	
Arterial line	53	12 (23%)	29 (55%)	8 (15%)	4 (7%)	
Intra-aortic balloon pump	27	—	—	19 (70%)	1 (4%)	7 (26%)

TABLE VIII. Staffing Patterns on Coronary Care Units: Registered Nurse/Patient Ratios

	Registered Nurse/Patient Ratio			
Shift	1:1	1:2	1:3	1:4
Days	0% (0/52)	85% (44/52)	13% (7/52)	2% (1/52)
Evenings	0% (0/52)	61% (32/52)	37% (19/52)	2% (1/52)
Nights	0% (0/49)	47% (23/49)	45% (22/49)	8% (4/49)

would be an increase in allied personnel to assist with patient care. In actuality, the number of allied personnel also decreased on the evening and night shifts (Table X). Thus, as shown in Tables VIII, IX, and X, CCUs had adequate staffing on the day and evening shift, but on the night shift there was a reduction in RN services and also a reduction in support services.

Organization and Operation. The responsibility for operations of CCU and PCU was multidisciplinary, with input from the director of cardiology, 66% (36/54); the director of CCU, 83% (45/54); nursing administration, 88% (48/54); and the head nurse, 72% (39/54). Ninety-six percent (51/53) of the CCUs had a quality assurance program. Participation was multidisciplinary as follows: 94% (48/51), head nurse; 88% (45/51), nursing administration; 73% (37/51), director of CCU; 53% (27/51), director of cardiology; 63% (32/51), RN staff; and 12% (6/51), house staff.

Standing Orders. RNs assigned to CCU and PCU must be responsible for the initiation of treatment in cardiovascular emergencies. Ninety-one percent (48/54) of the CCUs and 74% (14/19) of the PCUs had standing orders to permit RNs to defibrillate patients in the absence of a physician. In 88% (48/54) of the CCUs and 84% (16/19) of the PCUs, RNs could administer intravenous (IV) medications according to a predetermined protocol. Seventy-seven percent (42/54) of RNs in CCUs and 48% (11/23) in PCUs were allowed to start IVs. Even though New York State laws do not permit licensed practical nurses (LPNs) to add medications to IV solutions, to initiate IV medicated fluids, or to change bottles/bags to different medicated fluids, 5% (3/54) of the CCUs allowed LPNs to perform such activities in the absence of a physician.

Nutrition. The information provided by the dietary staff of the hospitals surveyed was often incomplete. It would appear, however, that very little nutrition education or standardization of nutrition care is incorporated into the recovery program of the patient with MI. Fifty-eight percent (31/53) of the hospitals surveyed had one registered dietitian permanently assigned to the CCU at least on a part-time basis. Eleven percent (6/53) utilized students or dietary interns for nutrition support in their CCU. The chief dietitian or director of food service in most hospitals was responsible for writing dietary procedures. One hospital responded that a medical board/nutrition committee was responsible for establishing dietary protocols. Fifty-eight percent (31/53) of the hospitals indicated that each patient in the CCU was counseled by a registered dietitian. In those hospitals in which patients were not regularly counseled by a registered dietitian, 6% (3/53) were counseled by dietitians who were registry eligible, 47% (25/53) by a nurse, and 21% (11/53) by a physician. Hospitals varied in their post-myocardial infarction diet protocols. For example, a sodium-restricted diet ranged from 500 mg in one hospital to 2 g in another. In other cases, hospitals responded that cholesterol and fat were modified without giving specific values.

TABLE IX. Coronary Care Unit—Post-Coronary Care Unit Patient Care Hours

Total hours of direct nursing care/day by a registered nurse				
	4 hours	4–6 hours	7–10 hours	12 hours
CCU	0% (0/51)	22% (11/51)	27% (14/51)	51% (26/51)
PCU	5% (1/20)	55% (11/20)	20% (4/20)	20% (4/20)
Total nursing care hours*				
	4 hours	4–6 hours	7–10 hours	11–13 hours
CCU	0% (0/50)	6% (3/50)	30% (15/50)	64% (32/50)
PCU	10% (2/20)	45% (9/20)	15% (3/20)	30% (6/20)

* In calculating total nursing care hours the following staff are included:

	CCU	PCU
Head nurse	52% (28/54)	25% (5/20)
Assistant head nurse	41% (22/54)	35% (7/20)
Licensed practical nurse	15% (8/54)	20% (4/20)
Nurses' aide	30% (16/54)	25% (5/20)
Unit clerk	4% (2/54)	10% (2/20)

TABLE X. Utilization of Allied Health Personnel: Percent of Units Utilizing Nurses' Aides and Unit Clerks by Shift

	Coronary Care Unit		Post-Coronary Care Unit	
	Nurses' Aide	Unit Clerk	Nurses' Aide	Unit Clerk
Days	69% (34/69)	87% (46/53)	65% (15/23)	70% (16/23)
Evenings	51% (25/49)	62% (33/53)	57% (13/23)	39% (9/23)
Nights	53% (26/49)	27% (14/52)	52% (12/23)	0% (0/23)

In 55% (29/50) of the hospitals, patients were counseled routinely by a registered dietitian while they were in PCU. Ninety-two percent of the hospitals stated that educational materials were available and distributed to patients and their families. The source of these materials varied from excerpts from hospital diet manuals, information from the American Heart Association, and materials prepared by private corporations.

Twenty-three percent (12/50) of the hospitals responded that patients were seen by a registered dietitian after discharge from the hospital. The majority of hospitals providing follow-up care saw patients every other or every third month. In most instances, patients were seen individually rather than in groups, and only five hospitals had a formal outpatient nutrition education program.

Cardiac Rehabilitation. Thirty-three percent (18/54) of hospitals that responded had a formal cardiac rehabilitation program. Seventeen of 18 programs had a salaried director. Twelve of the 18 were directed by a physician, five by a nurse, and one by a social worker. Nine of the physician directors were cardiologists, and three were psychiatrists. The person responsible for patient education was the CCU nurse in ten hospitals, a nurse practitioner in four, a medical nurse in two, and a health educator in two. In 13 programs, cardiac rehabilitation began in the CCU, whereas in five it began in the PCU or in the general medical ward. All 18 programs educated persons individually. Thirteen programs also had family discussions, and 14 had group classes. Audiovisual aids were used in all programs. Fifteen programs had a written protocol of progressive ambulatory exercise after discharge from the CCU. Fourteen of the programs systematically treated smoking, hypertension, elevated cholesterol, and obesity. Twelve programs treated Type A behavior. Twelve programs had a specific dietitian assigned to their cardiac rehabilitation programs. Twelve programs had a physical therapist. Discussions were held in 17 of the programs, either as group, individual, or family sessions. Four programs made electrocardiograms available to their patients at the time of discharge from the hospital, and four programs taught cardiopulmonary resuscitation to family members. Eight programs referred patients to outpatient exercise programs.

Comparative Analyses. As expected, the larger hospitals and the university hospitals tended to have more high technology related services. Of interest, being designated an EHCS did not ensure that the CCUs of those hospitals would be more likely to comply with JCAH or ICHDR recommendations. Moreover, hospital size, type, location or relationship to a medical school did not appear to influence compliance.

DISCUSSION

The results of a questionnaire distributed to 72 hospitals in New York City by the AHA NYCA demonstrated a substantial variability in the facilities and practices of coronary care for those hospitals that responded. Differences do not appear to be explained by hospital size, type, location, or relationship to one of the six medical schools in the city. Overall, hospitals appear to be most closely compliant with the recommendations of the JCAH[5] and ICHDR[6] in the areas of biotechnology and medical care, and least likely to satisfy those recommendations for patient education and rehabilitative services. Moreover, there exists a substantial departure from the ideal that hospitals provide optimal nursing care and laboratory services 24 hours a day.

While CCUs appeared to be strongest in their biotechnical resources, even here there was a substantial departure from the optimum. Twenty percent of hospitals did not have a freestanding CCU. Moreover, one third of units had been designed before 1970. Use of the computer is still limited. Forty-five percent of hospitals indicated that they had computer-assisted arrhythmia detection systems; however, only 15% had computer software for a "patient management system." Moreover, only 8% used a computer for record keeping.

Because it is not feasible, or even desirable, for all hospitals to have a full range of cardiology services, hospitals that do not offer cardiac surgery must be able safely to transport critically ill patients. For some such patients, an intra-aortic balloon support may be required. Yet, only 42% of hospitals indicated that they had this device. Moreover, those hospitals that had intra-aortic balloons tended to be those that offered cardiac surgery. Thus, the ability to stabilize severely compromised patients before transfer to cardiac surgery centers within New York City is limited.

The major role of the CCU remains the recognition and treatment of cardiac arrhythmias.[2] However, only 40% of CCUs had someone continuously observing the central monitor 24 hours a day. Since computer back-up for arrhythmia detection was present in only 45% of CCUs and electrocardiograph recall in 65%, there exists a substantial departure from the ideal that all patients be continuously monitored for arrhythmia detection.

The registered nurse with expertise in coronary care remains the paramount health care professional in the CCU. She must be trained to recognize and treat cardiac emergencies. But, also, she must provide the human support for patients confronted with a catastrophic illness in a high technology, and frequently isolated, environment. To meet these needs, a ratio of one nurse to every two patients and 12 hours of direct nurse-patient care is recommended. This survey indicates that 85% of CCUs indeed have a one-to-two ratio on the day shift. However, the percentage satisfying this recommendation decreased to 61% on the evening shift and was only 47% on the night shift. Moreover, only 51% of CCUs indicated that 12 hours of direct registered nursing care were provided. Not only was the number of registered nurses likely to fall off after the day shift, but the availability of experienced registered nurses and the assistance of allied health professionals was less likely on evening shifts and least likely at night.

Post coronary care follow-up was the area of greatest deficiency identified by the questionnaire. Despite continued risk of an untoward event following myocardial infarction after the coronary care unit period, 46% of hospitals transferred patients directly from the CCU to unmonitored medical wards. Nutritional counseling was available in only 58% of hospitals. In fact, only 38% had a special diet for post-myocardial infarction patients. Also, only 38% offered any formal inpatient cardiac rehabilitative program.

The reliability of data derived from a questionnaire is dependent on the number responding and the accuracy of the responses. Four out of five hospitals that received the questionnaire responded. The distribution of the responding hospitals was representative of those that received the questionnaire as to size, location, and type of hospital. A review by the committee failed to disclose a pattern that might suggest that the nonresponding hospitals were substantially different from those responding. Also, even though one might expect that the care and time given to completing the questionnaire varied among hospitals, similar responses to identical questions in different sections, which were completed by different respondents at the hospital, tended to show a consistency of answers, suggesting that the data reported at least reflected the beliefs of the respondents. Thus, within the inherent limitations of the questionnaire methodology, this report would appear to reflect the state of coronary care in New York City hospitals during the period of the review.

The findings of this questionnaire must also be viewed in the context of the economic conditions of the hospitals surveyed. Many New York City hospitals were having financial difficulties at the time of the study. The expense of medical high technology, the marked increase in labor costs during the past decade, the prevalence of a relatively large body of nonpaying patients in New York City, the delays in third party reimbursement with its attendant effects on "cash flow," and the cost of administering programs that must respond to a marked increase in governmental regulation are factors that have contributed to the economic plight in which some hospitals found themselves. Moreover, this report was completed before the diagnosis-related grouping of payments to hospitals for Medicare patients was introduced in New York State or the emphasis on outpatient treatment had begun. These recently imposed economic factors are likely further to increase the fiscal difficulties of many hospitals. Thus, while the recommendations of JCAH and ICHDR for optimal medical care are clearly desirable, these expectations may not be realistic in the setting of the medical economics prevalent in New York City at this time.

In summary, these results suggest that, overall, there is a sharp departure from optimum care as defined by the recommendation of ICHDR and JCAH for coronary care in the hospitals of New York City. While most facilities generally meet the physical plant requirements, this survey suggests that many are antiquated and lack state-of-the-art equipment; in particular, use of computer technology is limited. Although almost all CCUs satisfy the basic requirements for continuous electrocardiographic monitoring systems, most CCUs do not have someone available 24 hours each day to identify arrhythmias. Basic nursing care generally meets recommendations on the day shift. This, however, falls off substantially during evening and night shifts. Also, relatively few hospitals have adequate programs directed at risk factor modification, nutritional counseling, and cardiac rehabilitation. Not surprisingly, this survey would support the notion that optimal coronary care is most likely in the daytime of weekdays and least likely on holiday nights.

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Diagnosis of stress-related hyperprolactinemia

Evaluation of the hyperprolactinemia rest test

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ABSTRACT. **It remains controversial whether two or three random estimations of prolactin (PRL) concentration obviate the need for special testing to rule out stress-related hyperprolactinemia. In order to clarify this issue, we measured PRL, cortisol, and growth hormone (GH) in serial blood samples obtained under resting conditions (HPR test) in 70 women who had had high PRL levels in two or more random blood samples. Twenty out of 70 women were found to have stress-related hyperprolactinemia, and 11 of the 20 would have been misdiagnosed by using only three random samples. Cortisol levels from the HPR test indicated stress-related pituitary adrenal reactivity in all groups, including the idiopathic (N = 30) and prolactinoma (N = 20) groups. However, PRL secretion in response to stress—a downward trend as a function of time—was evident only in the stress-related hyperprolactinemia group. These results suggest that a limited HPR test (ie, serial PRL determinations at 0, 30, and 60 min) is a valuable and simple measure to identify stress-related hyperprolactinemia in order to avoid diagnostic pitfalls and unnecessary treatment.**

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Man, like other vertebrates, reacts to physical and psychological stressors by releasing prolactin (PRL), as well as adrenocorticotropin (ACTH) and growth hormone (GH) from the anterior pituitary gland.[1] The secretory response of these hormones to acute stressful events is similar: a prompt but transient rise in circulating hormone levels. However, the increase in cortisol, secondary to ACTH release, is the most consistent reaction to stress. Transient hyperprolactinemia appears to be a common finding in women who attend infertility or general gynecologic clinics.[2,3] Koninckx[2] found high PRL levels in 19% and 24% of infertile patients following the interview or the gynecologic examination, respectively. We and others have also noted a frequent occurrence of apparently stress-related hyperprolactinemia following clinical examination in patients with reproductive disorders.[4,5] To avoid misdiagnosis in women with initial moderate hyperprolactinemia, Koninckx[2] suggested that measuring PRL in serial blood samples obtained during a 45-minute period would identify transient hyperprolactinemia due to stress. On the other hand, Moult et al,[5] who studied a large and diverse group of patients of both sexes, concluded that random sampling PRL determinations were sufficient to rule out stress-related hyperprolactinemia.

We sought to discern which method (PRL determinations in blood samples collected either randomly or serially under resting conditions) is more sensitive for diagnosis of stress-related hyperprolactinemia. We studied a group of patients with oligo-ovulation or anovulation who showed high serum PRL levels (>20 ng/mL) at the initial visit. We also simultaneously measured other stress-related hormones, such as cortisol and GH, for a more complete evaluation of stress reactivity in these patients.

PATIENTS AND METHODS

The total population in this study included 70 women with oligo-ovulation or anovulation. They ranged in age from 17 to 44 years (mean ± SE, 30 ± 4 years) and had had serum PRL levels greater than 20 ng/mL in two consecutive blood samples taken one to two weeks apart ("random sampling"). Their clinical presentations included oligo- or amenorrhea, galactorrhea, infertility, and/or hyperandrogenism. Pituitary and hypothalamic lesions were ruled out through hypocycloidal polytomography or computed tomography (CT) scanning. There were no patients with hypothyroidism or renal insufficiency, nor any with drug-related hyperprolactinemia, and no postmenopausal patients were included.

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Serial blood sampling (hyperprolactinemia rest (HPR) test) was conducted with the patient in supine or sitting position isolated in a quiet room. Patients were allowed a light, low-protein meal more than two hours prior to testing. An indwelling catheter with heparin lock was inserted into a brachial vein, and three to six blood samples were taken at 15 to 30 minute intervals. Serum samples were frozen at −20°C until assayed for hormone determinations. PRL and GH serum concentrations were analyzed by radioimmunoassay (RIA) using commercial kits (Diagnostic Products Corp, Los Angeles, Calif). In our hands, the intraassay and interassay variabilities were 5.0% and 6.6% for PRL, and 7.0% and 7.4% for GH, respectively. Cortisol was also measured by RIA according to a previously described method[6]; intraassay and interassay variation coefficients were both 5.3%. All individual patient samples, except for the pretest ones, were measured in the same assay run.

Comparison of age and parity, as well as pretest PRL and initial HPR test values ("random samples") among the three groups of patients was carried out by one-way analysis of variance; differences were assessed by a multiple range test (Student-Newman Keuls procedure). Differences in PRL, GH, and cortisol levels at different times during the HPR test within each patient group were analyzed by multiple analysis of variance (MANOVA) and the Student's paired t-test with Bonferroni adjustment utilizing SPSS software. Correlations among PRL, GH, and cortisol value trends within and between groups were evaluated by regression analysis, setting confidence limits to the r value using the Z-transformation.[7] All results were expressed as the mean ± standard error unless noted otherwise.

RESULTS

Patients were categorized into three groups according to a priori determined criteria: radiologic evidence of sellar mass and/or serum PRL concentrations >200 ng/mL = prolactinoma; negative radiologic findings and PRL levels <200 ng/mL = idiopathic or stress-related hyperprolactinemia; persistent high PRL levels during the HPR test = idiopathic hyperprolactinemia; and downward trend with normalization of PRL levels (<20 ng/mL) with time during the HPR test = stress-related hyperprolactinemia.

The prolactinoma group included 20 patients (age, 31.5 ± 1.9 years; range, 19–44 years; parity, 1.0 ± 0.5; range, 0–5) with radiologic evidence of a pituitary tumor (15 microadenomas and five macroadenomas). Two patients underwent surgery, and a microprolactinoma was found in both. This group showed the highest serum PRL levels (213 ± 29 ng/mL; range, 25–1,196 ng/mL).

The idiopathic hyperprolactinemia group included 30 patients (30.5 ± 1.5 years; range, 17–46 years; parity, 0.7 ± 0.3; range, 0–3) without radiologic abnormality. Their initial circulating PRL levels (61 ± 9 ng/mL; range, 23–156 ng/mL) were persistently elevated during the HPR test.

The stress-related hyperprolactinemia group consisted of 20 patients who showed negative radiologic findings (28.0 ± 0.9 years; range, 21–33 years; parity, 0.5 ± 0.1; range, 0–4). Their pretest hyperprolactinemia (32 ± 2 ng/mL; range, 26–69 ng/mL) disappeared at or during the HPR test. There were significant differences among the three groups in regard to age and parity.

Mean serum PRL levels (Fig 1) were significantly different ($p < 0.01$) among the three groups before (pretest I and II) and throughout testing. In the stress-related hyperprolactinemia group, pretest mean PRL concentrations were higher ($p < 0.01$) than mean HPR test values at all times. Also, in the same group, the mean PRL level at time zero was significantly higher than at 60–90 minutes. Mean circulating PRL concentrations during testing were 20 ng/mL or lower, except for time zero. Indeed, nine out of 20 patients with stress-related hyperprolactinemia showed normal PRL levels at the initiation of testing, and by 60 minutes all patients were euprolactinemic. In contrast, mean serum PRL concentrations in the prolactinoma and idiopathic hyperprolactinemia groups showed neither a downward trend during the HPR test nor a difference between pretest and test values. All serum PRL values from each patient in these two groups remained elevated throughout testing.

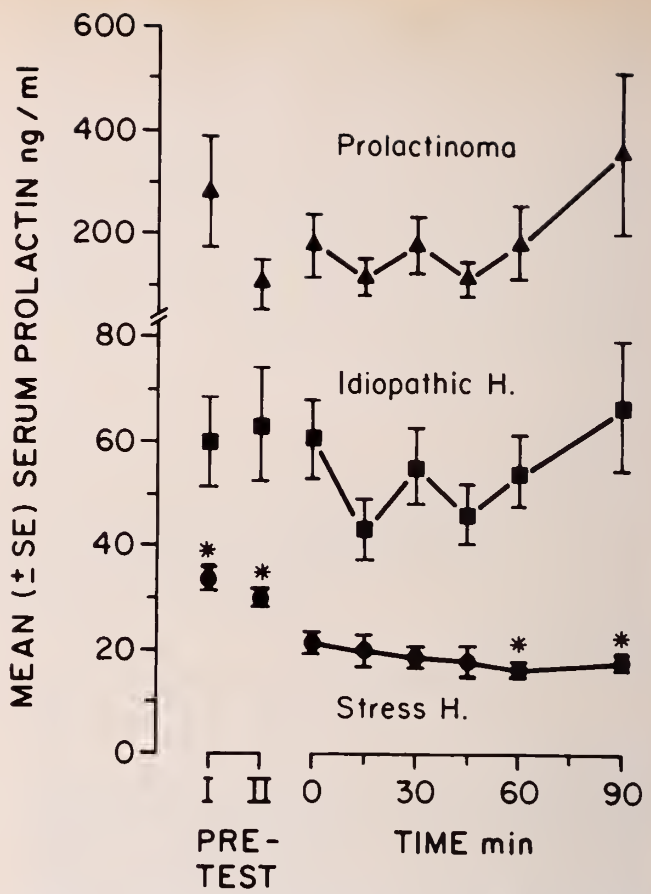

FIGURE 1. Mean ± SE serum PRL levels in women with prolactinoma (N = 20), and idiopathic (N = 30) and stress-related (N = 20) hyperprolactinemia before (pretest I and II) and during the HPR test. Differences (*$p < 0.01$) are in relation to time zero values. (Mean PRL values among groups were also significantly different—$p < 0.01$—at all times.)

The HPR test serum cortisol levels of all three groups are shown in Fig 2. Cortisol, like GH, was not measured in pretest serum samples. There was no significant difference in mean cortisol levels among the three groups throughout testing. The cortisol pattern showed a downward trend to reach a final value significantly lower than the zero value in all groups. At zero time, high serum cortisol concentrations (greater than 20 μg/dL) were found in four patients with prolactinoma, and in three patients from each of the other two groups. In all these cases, cortisol values fell within normal levels with time.

Mean serum GH levels showed no significant differences among the three groups during the HPR test (Fig 3). Following a small peak at 15 min, GH levels declined slightly thereafter, however, no significant differences were found in relation to initial values in any group. High initial GH concentrations (greater than 5 ng/mL) were found in three patients with prolactinoma, in six with idiopathic hyperprolactinemia, and in two with stress-related hyperprolactinemia. As in the case of cortisol and PRL levels, these high GH concentrations normalized with time during the HPR test.

A significant positive correlation ($p < 0.01$) was found only between PRL and cortisol values during the HPR test in the stress-related hyperprolactinemia group, but not in the other two groups. The PRL-cortisol trend correlation in the stress group was found to be significantly different ($p < 0.001$) in relation to that of the idiopathic group, but not to that of the prolactinoma group. There were no significant correlations between GH and other hormone values.

DISCUSSION

These results show that stress-related hyperprolactinemia is common among other hyperprolactinemic condi-

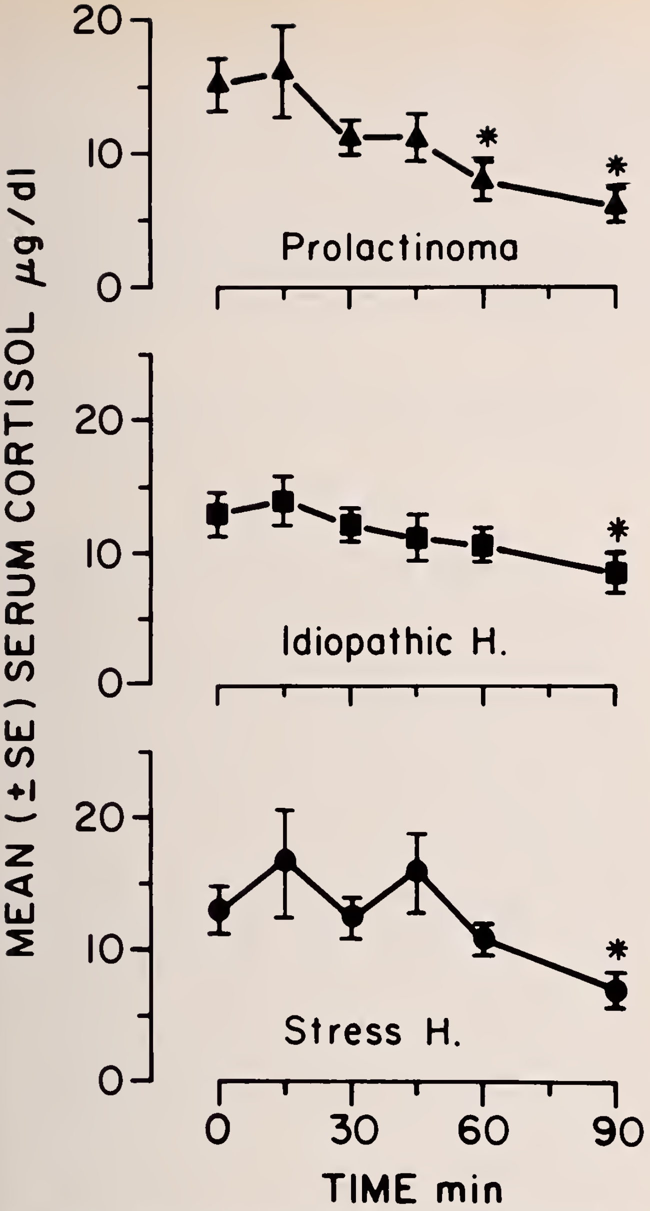

FIGURE 2. Mean ± SE serum cortisol levels in women with prolactinoma, and idiopathic and stress-related hyperprolactinemia during the HPR test. Differences (*$p < 0.01$) are in relation to time zero values.

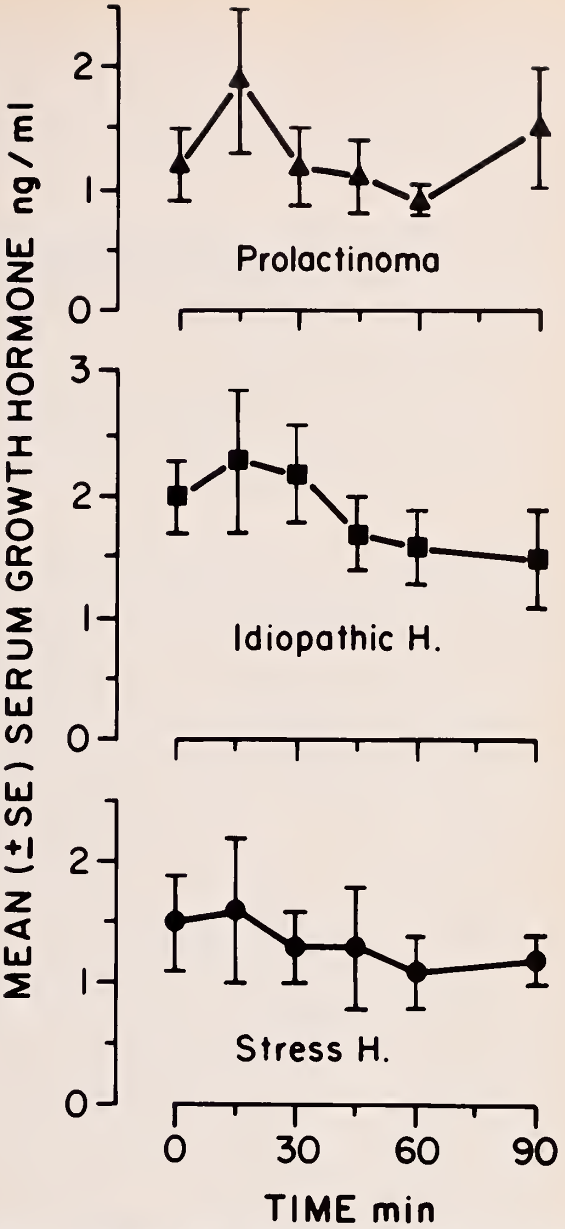

FIGURE 3. Mean ± SE serum GH levels in women with prolactinoma, and idiopathic and stress-related hyperprolactinemia during the HPR test.

tions in oligo-ovulatory or anovulatory women with or without galactorrhea. These findings also suggest that the HPR test may be an effective measure for the diagnosis of this transient, nonpathologic form of hyperprolactinemia, as proposed by Koninckx.[2] Twenty out of 70 patients (28%) who had shown initially high serum PRL levels in two random consecutive blood samples were found to be euprolactinemic by the HPR test. Nine of these patients showed normal circulating PRL concentrations at the initiation of the test. In other words, a third random PRL determination would still have misdiagnosed 11 patients whose initial hyperprolactinemia was stress-related. Thus, contrary to the suggestion expressed by others,[5] two or three consecutive random serum PRL level determinations diminish but do not obviate the need for an HPR test to rule out stress-related hyperprolactinemia.

As expected,[8,9] patients with prolactinoma showed the highest circulating PRL concentrations, and they were followed by the idiopathic hyperprolactinemia group. There were no significant changes in mean PRL levels during the HPR test in both groups. Moreover, individual serum PRL levels did not reach normal concentrations in any patient with idiopathic or tumoral hyperprolactinemia.

Cortisol release is the most consistent endocrine response to stressful events.[1] Indeed, in all three hyperprolactinemic groups, the mean serum cortisol levels declined significantly during the HPR test. The finding of lower cortisol, as well as PRL levels, with time during the HPR test most likely represents the dissipation of an initial stress-related pituitary reaction. The significant correlation between PRL and cortisol values during the HPR test in the stress-related but not in the other hyperprolactinemic groups reinforces this contention. A similar stress effect on GH secretion, that is, initial high values followed by declining levels, was evident only in a few patients. This dissociation in endocrine response to physical and psychological stressors has been found before in both animals and man.[10,11]

Increase in release of PRL, and also GH and cortisol, can result from diverse kinds of physical and psychological stressors.[1,11] The major determinant for the stress-related increased PRL release in these hyperprolactinemic patients was probably psychological in nature. A transient rise in serum PRL levels has been found in women with no apparent anxiety in anticipation of a major event like surgery.[11] Likewise, experimental mental distress produced an increment in PRL secretion in young women.[12] A similar response occurred following interview in infertile patients.[2,3]

Aside from emotional distress, physical stressors might also have contributed to the stress-related hyperprolactinemia in some cases. Pelvic and breast examinations may inconsistently cause a discrete rise in circulating PRL levels.[2,10] Similarly, in some individuals, venipuncture, particularly if painful, also affects PRL, as well as GH and cortisol levels.[13-15] The individual endocrine reactivity appears to be related to a behavioral trait as well as to experience—"anxious" and "anxious—not engaged" patients showed dissimilar endocrine responses to cardiac catheterization, but no reaction occurred among "depressed and calm" patients.[16] Decreased or absent endocrine responses have been found to develop with repeated catheterization or other stimuli, suggesting adaptation.[15,17] Indeed, the decline in circulating PRL levels in these patients from the initial visit to the time of testing could have been the result of adaptation. It is noteworthy that chronic anxiety does not effect increased PRL release.[18] The mechanism of acute stress-related PRL release is not clear. Recent experimental studies suggest that stress triggers PRL release mainly through activation of PRL-releasing substances, such as vasoactive intestinal peptide and polypeptide histidine isoleucine-27, and secondarily by attenuation of dopamine secretory activity.[19]

Transient hyperprolactinemia can be secondary to factors other than physical or psychological stressors. Ingestion of a protein-rich meal stimulates PRL release, which occasionally results in mild, short-lasting hyperprolactinemia.[21,22] Postprandial elevation in cortisol and GH levels has also been described.[20,21] Sometimes, the normal episodic PRL secretory bursts reach beyond the upper normal limit of circulating PRL levels.[5] Thus, sampling at the time of a PRL pulse may also be misleading.

It is unlikely that a sporadic, mild elevation in circulating PRL levels, as seen in stress-related hyperprolactinemia, disturbs reproductive function. For example, in partial lactators, the large magnitude, suckling-dependent PRL release bursts fail to maintain anovulation, frequently leading to conception.[23] Further, pregnancy has been reported to occur with relative frequency in hyperprolactinemic patients.[24] On the other hand, it has been claimed that intermittent, mild rises of serum PRL levels negatively affect fertility.[25] Other findings, however, contradict this claim.[26]

Stress-related hyperprolactinemia is a common event which, in the majority of cases, can be identified by two or three random serum PRL determinations.[5] An HPR test is, however, needed to further identify an important group of patients with stress-related hyperprolactinemia who show mild to moderately elevated PRL levels (>20–<100 ng/mL) in two to three consecutive random samples.

In summary, an HPR test limited to three blood samplings for only PRL determination at 0, 30, and 60 min should suffice to diagnose stress-related hyperprolactinemia and to help avoid diagnostic pitfalls and unnecessary treatment.

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Periodic physician recredentialing proposed in New York State

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Usually reliable authorities have attributed to Adam the following comment while pausing under an apple tree in the Garden of Eden, "Eve, we are living in a period of transition." It was true for Adam and Eve and it is abundantly true for us in the health care professions. There are many manifestations of this. In a recent editorial in the *New England Journal of Medicine*, Relman characterized the last 50 years of American medicine as comprising three revolutionary periods.[1] These included the Era of Expansion, in which biomedical knowledge increased at an exponential rate, medical specialties proliferated, and the production of physicians was doubled by increasing medical school output and licensing foreign medical graduates. This period from the end of World War II to the mid-1970s was followed, Relman proposes, by the Revolt of the Payers, or the Era of Cost Containment. This period is familiar to all deliverers of health care and represents a struggle which is likely to continue for the foreseeable future. Relman, however, suggests that a new revolution is now in the making, the Era of Assessment and Accountability. The editorial was stimulated by a special article in the same issue of the *New England Journal of Medicine* by Roper and his colleagues[2] from the Health Care Financing Administration entitled "Effectiveness in health care: An initiative to evaluate and improve medical practice." This important policy statement begins a cooperative program between the Department of Health and Human Services, health care providers, researchers, medical educators, private purchasers, and consumer groups to assess the effectiveness of health care. Among its many possibilities are improved guidelines for medical practice.

INITIATIVE FOR PHYSICIAN RECREDENTIALING

Of particular pertinence to the above was a public pronouncement in 1986 by Governor Mario Cuomo of New York that there would be periodic recredentialing of physicians for the license to practice medicine in the state.[3] This proposal is a part of an overall program to maintain a high quality of medical care in the state and was a logical follow-up of the administration's medical malpractice law which was approved by the legislature in 1985.[4] This latter law emphasizes quality of care for hospitalized patients through mandatory incident reporting of adverse events which are unexpected in the natural history of an illness, increased monitoring by the quality assurance committees of adverse patient outcomes, and ultimate responsibility of the governing boards of hospitals for all aspects of hospital care.[5] In considering the unique step of physician recredentialing by a state, there are other considerations which indicate that this is a timely move with national implications.

HISTORY OF PHYSICIAN LICENSURE

Physician recredentialing may not be accepted with enthusiasm by the medical profession. It is to be noted, however, that medical licensure as a mechanism to assure the public that it will be well served by a physician also had a stormy course in America. First attempted in Massachusetts in 1649, it failed to provide assurance of uniformity of knowledge and skills in physicians and was abandoned. Other attempts were made by other states but it was not until 1846 that the Medical Society of the State of New York (MSSNY) called for a national convention for the purpose of elevating the standard of medical care in the United States.[6] The American Medical Association (AMA) was created as a result of that meeting and in 1883 the AMA recommended that state medical boards develop licensing examinations.[7] By the turn of the century, almost all states had passed legislation requiring the use of such examinations. Subsequently, the Federation of State Medical Boards (1912) and the National Board of Medical Examiners (1915) were established to provide a voluntary national certification process for the initial license to practice medicine. Medical specialty certification adopted by a few specialties in the early 1900s multiplied rapidly following the explosion of fundamental and applied knowledge after World War II so that now there are 23 specialties which require examinations for designation as a board-certified specialist.

PROGRESS IN MEDICINE AND MEDICAL LICENSURE

The rapidity and magnitude of advances in medical science with attendant clinical applications challenges the initial assumption that the primary medical license or spe-

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cialty board certification provides lifetime assurance of continuing knowledge and skills. In order to maintain the specialty board certification, for example, 17 of the 23 medical specialty boards have already initiated or have planned a requirement for recertification at intervals of seven to ten years. Another recognition of the attrition in medical knowledge is the expectation that all physicians would participate in continuing medical education either through individual effort or organized conferences and courses, and for some states, this is mandated for maintenance of the medical license. Continuing medical education is undoubtedly valuable and necessary, but in the absence of validation of the putative beneficial effect, it fails to provide the necessary objective assurance that it prevents professional obsolescence and that appropriate standards of medical care are being maintained.

The medical care system in the United States can with good justification claim that it is of high quality. Despite this positive view, there are indications that not all is well. The malpractice crisis saps the trust in the patient-physician relationship with unnecessary and even hazardous defensive medical practices, with escalating malpractice insurance premiums which lead to incremental health care costs, and with inadequate understanding by patients of the limitations of medicine. There is also clear evidence of inadequacies among physicians as manifested by significant variation in the utilization of operations and procedures among comparable populations which are not explicable by variations in the health needs of the patients. An example documented recently in New York was the variable frequency of surgical procedures in the elderly from one county to another which could not reasonably be accounted for by variation in medical indications.[8]

These facts, together with public sophistication and the recognition that physicians, like airline pilots who also make critical decisions affecting life and limb, have a responsibility to provide periodic assurance that their knowledge base is current. It was to this end that the governor requested the commissioner of health and the chancellor of the board of regents to appoint an advisory committee on physician recredentialing.

The New York State Advisory Committee on Physician Recredentialing

The committee,[*] appointed in the spring of 1987, had broad representation of medical practitioners, medical educators, public representation, members of the state medical society, national organizations involved in medical certification, and state government. At the outset, it was recognized that the charge to develop recredentialing for appropriate standards of medical practice is not synonymous with certification of medical competence. The latter concept describes a comprehensive quality which includes knowledge, skills, psychosocial relationships to patients, and mature judgment and there are no tests of competence which have been validated. The recommendations[9] for recredentialing in New York are a first approximation to an additional quality assurance mechanism in medicine and include many of the identified components of competence.[10]

There are implicit and explicit guidelines which were adopted by the advisory committee:

- The standards of medical practice to be used in recredentialing would be defined and publicized widely and the evaluation of the individual physician's compliance would be examined prospectively, not retrospectively.
- The process of recredentialing is to be educational, not punitive.
- The evaluations are to be done by peers.
- The outcome of the evaluations is, if necessary, to be remediation, not the loss of the license to practice medicine.
- There are to be multiple options for the recredentialing process.
- Before formal implementation of recredentialing, there would be validation of the proposed processes.

The criteria for recredentialing are necessarily different from those which must be met at the time of initial medical licensure. For the latter it is appropriate to determine the cognitive knowledge of the applicant in the broad fields of biomedical science and clinical medicine. Subsequently, physicians differentiate into a variety of clinical disciplines and modes of activity to meet the health needs of the public. The recredentialing is not designed to determine the physician's knowledge of all aspects of medicine, but rather to determine whether his/her current practice meets appropriate standards. This will be achieved by providing ample educational material about which it is reasonable to expect doctors to be knowledgeable and to validate that by testing; by peer review of performance through precise protocols; and by utilizing the specialty board recertification process where appropriate.

At the present time, all physicians in New York are required to register at three-year intervals for a revalidation of the medical license. This is a formality and merely requires the completion of a form and payment of a registration fee. It is proposed that the recredentialing process be temporally related to registration at nine-year intervals rather than three years. This conveniently falls within the intervals of recertification by the specialty boards (seven to ten years).

The general mechanisms for recredentialing which have been proposed include specialty board certification or recertification, cognitive and clinical problem solving testing, and peer review according to specified protocols.

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[*] Members of the New York State Advisory Committee on Physician Recredentialing: Arthur Aufses, MD, Mt Sinai Medical Center; Bertrand M. Bell, MD, Albert Einstein College of Medicine; Robert Biblo, Health Insurance Plan; Valerie Burke, MD, Queens Hospital/South Jamaica Community Health Center; Martin Cherkasky, MD, Board of Regents; John A. Finkbeiner, MD, Medical Society of the State of New York; Julius W. Garvey, MD, Queens Clinical Society; Alfred Gellhorn, MD, New York State Department of Health; Stanley L. Grossman, MD, Mid-Hudson Surgical; Nathaniel Hurst, MD, Park Hope Nursing Home; Donald Langsley, MD, American Board of Medical Specialties; Marvin Lieberman, PHD, New York Academy of Medicine; David Mesches, MD, New York Medical College; John P. Naughton, MD, State University of New York at Buffalo; Lloyd F. Novick, MD, New York State Department of Health; Richard N. Pierson, MD, St Luke's Roosevelt; Joseph T. Post, MD, New York Academy of Medicine; John T. Prior, MD, Empire State Peer Review Organization; Linda Randolph, MD, New York State Department of Health; Lawrence Scherr, MD, American College of Physicians; Judith Simmons, MD, New York State Department of Health; William A. Stewart, MD, Board for Professional Medical Conduct; Anthony P. Tartaglia, MD, Albany Medical Center Hospital; Robert Volle, PHD, National Board of Medical Examiners.

Every physician will have the option of selecting one of the three methods.

Specialty board certification and recertification is standard for those who meet the eligibility requirements. Table I indicates the current status of specialty board recertification.

Cognitive and clinical problem solving testing, another suggested mechanism for recredentialing, is now in an early stage of development. Cooperation of the Federation of State Medical Boards Licensing Examination (FLEX) Board and the National Board of Medical Examiners (NBME) is an essential and much desired component of this option. These two organizations have been approached to develop a unique examination for those practicing general adult or pediatric medicine. The examination, it has been proposed, will be four to six hours in length and will be designed to indicate areas of knowledge or clinical problem solving deficiencies that require remediation. A syllabus and/or suggested reading will be available for refresher study prior to the examination. This, as well as other proposed recredentialing means, will be validated by pre-trial.

In addition to specialty board recertification, or a cognitive problem solving practice oriented test, there is a third option which will be developed. This is a peer review of a physician's patient record according to a precise protocol. The objective criteria for hospital-based and office-based practitioners are being developed. The review of physicians' hospital or office records permits assessment of a broad range of the elements that determine the quality of the practice of medicine. Although a precise protocol for the evaluation of office records is being developed, the committee identified the requirements for such an instrument:

- The protocol must be sensitive to variation in the practice profiles of the physician population,
- The model developed must be credible to physicians and nonphysicians, and
- The record evaluation must incorporate peer review by appropriate professionals.

Systems have been developed for office record review such as that used by the American Board of Family Practice in its recredentialing process or the Ontario College of Physicians and Surgeons. A possible approach to the review of patients' office records is outlined in Table II.

The general purposes of the review of office-based physicians' records would be to evaluate the following:

- Information gathering skills
  —interview
  —physical examination
  —laboratory and other diagnostic procedures
- Ability to use information to:
  —formulate hypotheses

TABLE I. Current Status of Specialty Board Recertification

Specialty	Current Requirement*	Future Requirement	No Requirement
Allergy and immunology		1989	
Anesthesiology			x
Colon and rectal surgery			x
Dermatology		1991	
Emergency medicine	x		
Family practice	x		
Internal medicine		1990	
Neurological surgery			x
Nuclear medicine		1992	
Obstetrics and gynecology	x		
Ophthalmology		1992	
Orthopaedic surgery	x		
Otolaryngology		1989	
Pathology			x
Pediatrics		1988	
Physical medicine and rehabilitation		1993	
Plastic surgery	x		
Preventive medicine			x
Psychiatry and neurology			x
Radiology			x
Surgery	x		
Thoracic surgery	x		
Urology	x		

* The requirement for recertification does not apply to those who have already received a no-time-limited board certificate. For such persons, voluntary recertification is available.

TABLE II. Review of Office Records of Adult Patients

I. **Legibility**

II. **History**
  The reason the patient seeks medical advice
  The history should embody the facts of medical significance to the present complaints
  The past history, medications, family history, social and occupational history, and system review should be recorded when relevant to the present illness or in an initial general work-up

III. **Physical Examination**
  Physical signs are the objective and verifiable marks of pathology whose significance is enhanced when they confirm a functional or structural change evidenced by the history
  Although attention is directed by the history to the organ or system or part of the body, the examination should extend to all parts of the body, in a systematic procedure, except in a specialty work-up
  All investigations of the body are a part of the physical examination such as with the ophthalmoscope, sphygmomanometer, percussion hammer, etc.

IV. **Provisional Diagnosis**

V. **Instrumental and Laboratory Examinations**
  The selection of laboratory and other diagnostic procedures should be clearly justified by the findings to date. Every procedure that does not have a specific purpose contributory to the management of the patient's illness is wasteful of time, money, and/or safety
  Appropriate screening or preventive measures (Pap smear, immunizations, etc.)

VI. **Diagnosis and Formulation of a Management Plan**

VII. **Action Taken**
  Therapy, consultations, etc.

VIII. **Follow-Up**
  Including telephone responses

  —solve problems
- Knowledge base and ability to use knowledge
- Management
  —diagnosis
  —treatment
  —patient education
  —follow-up plan
  —follow up

By selecting particular diagnostic categories, evidence of physicians' recognition of important preventive medicine procedures can be ascertained, such as, for example, Pap exfoliative cytology in women or rectal examination in men or immunization information in children (Table III). Physicians' approaches to difficult diagnostic problems can be evaluated and the pertinence of follow-up visits noted. Peer review would be carried out. The recording in the chart of telephone consultations with patients could be another measure of continuity of care.

Communication with the major specialty societies indicates that many have or are in the process of developing standards of medical practice. It is possible that such standards of practice can be used for the peer review of the records of hospital-based physicians. Discussions of this approach are in progress with the Council of Medical Specialty Societies and with New York State specialty groups. An alternative to this preferred methodology is described in the advisory committee report.

PURPOSES OF RECREDENTIALING

The public interest will be best served by improvements in available medical services, and recredentialing is directed to this purpose. The aim of the process described is educational, not punitive. To this end, full information on the protocol for hospital recredentialing, on the examinations which may be selected for recredentialing, and on the methods for office medical record review will be widely publicized to the medical profession. It is expected that the methods selected for evaluation will provide an accurate indication of particular deficiencies. It was also proposed by the Committee that approved programs for correction of deficiencies be developed and made readily available to those judged to be in need of remediation. State and national groups, expert in this area, have been consulted.

The Advisory Committee on Physician Recredentialing recognized that this action by New York State is unique and without guiding precedents. The committee recommended that before implementation, appropriate pilot experiments be performed to validate the various instruments proposed for recredentialing. This will be a time consuming process which may require two years for completion.

TABLE III. Basic Components of a Pediatric Record

Summary of major diagnoses and problems
Family configuration and health history
Social history
Past history including birth, illnesses, review of systems
Nutritional history
Developmental history and/or school adjustment
Physical examination
Treatment plan (anticipatory guidance)
Progress notes
Immunization record
Vision, hearing, tuberculin, and laboratory screening tests
Growth charts
Consent for treatment

The Report of the New York State Advisory Committee on Physician Recredentialing: Phase One: General Principles, Proposed Process, Recommendations was transmitted to the chancellor of the board of regents and the commissioner of health in January 1988. Since its submission, an expert committee, consisting of active practitioners in the major specialties and a representative of the American Osteopathic Association, has met with representatives of the National Board of Medical Examiners and the FLEX Board to discuss possible test instruments. A similar committee has begun the development and review of the peer review protocols. After the test instruments have been developed, there will be a validation process with a randomly selected group of volunteer physicians who will take the tests and provide a critical assessment. Before final implementation, the recredentialing process must be approved by the board of regents of the state education department and the state legislature as well.

DISCUSSION

Despite the probable interval of two years or more between the Phase One report on physician recredentialing and the possible time of implementation, the reactions of physicians, organized medicine, national organizations and the public have been instructive and revealing. The formal responses of individual physicians both in-state and out-of-state as well as the American Medical Association, the Medical Society of the State of New York, and the New York County Medical Society have been largely negative. The reasons have included reservations with regard to the need for such recredentialing for the vast majority of physicians, questions regarding the pertinence of cognitive tests as a measure of competence, views that this activity represents uncalled for interference of government in the practice of medicine, and charges that the proposal to recredential physicians threatens the public confidence in doctors. Since the criticisms expressed in no way reflect the objectives of the advisory committee, it is important to respond to them.

The statement by physicians that recredentialing is unnecessary because most physicians keep their knowledge and skills current is a reasonable presumption and therefore there should be no anxiety about demonstrating that this is a fact. Physicians have a responsibility to their patients and to the public at large to provide objective reassurance of their current knowledge and skills. The financing of medical care is now accomplished largely through taxation. Medicare and Medicaid constitute a significant source of physicians' income. The Subcommittee on Health of the House Ways and Means Committee of the US Congress is drafting legislation to be introduced in the next congress which would require recertification of all physicians for Medicare reimbursement eligibility. Each time a physician utilizes a hospital, the likelihood is great that capital expenditures have been made possible by tax dollars. The public accountability of the medical profession is an obligation which should be accepted without demur.

It has been argued by physicians that they would not object to recredentialing if it were also required of other health professionals, and lawyers and other skilled groups. Although recredentialing of other professions may be a good idea, it is not relevant to the issue of physician recredentialing which stands by itself as an appropriate mechanism of public reassurance.

In discussions of recredentialing with physicians, it has been argued that cognitive testing is not a mechanism for measuring competence. As was pointed out in the report and earlier in this communication, competence is a many faceted characterization which includes knowledge and skills, judgment developed through experience, psychosocial skills, and an understanding of variations in human physiology and behavior. There are no objective tests for competence although it is possible that, with time, correlations between the types of testing proposed in the recredentialing report, which include components of clinical competence, and the recognition of competent physicians will provide a closer approximation than now exists. In the meantime, it should be apparent to the medical profession that the sort of tests proposed are analogous to the medical licensure examinations which have always been accepted as valid for the practice of medicine or those that are used in the certification of a specialist. Is it not inconsistent to recognize the validity of these and question the appropriateness of similar measures for recredentialing?

The issue of undue intrusion of government into the practice of medicine does not appear to be substantial. The advisory committee making the recommendations consisted of largely physicians. The testing mechanisms are being developed by physicians, and the appeal process outlined in the report is to physicians. Government is representative of the public and it too has a responsibility to provide quality assurance of medical care. Thus the medical profession and government are of necessity joint partners in recredentialing.

It has been suggested by some that the recredentialing of physicians threatens public confidence in the profession. The response of the public would appear to allay that concern completely. Individual citizens, citizen organizations, and newspaper and radio editorials have been uniformly supportive.[11-16] Other states and the US Congress have also expressed interest. In fact, it would appear likely that if the medical profession endorsed recredentialing in principle pending the development of the specific methods, it would markedly increase the confidence and respect of the public and might win public support for reform of malpractice litigation. This action merits serious consideration.

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Quality education is essential for quality health care

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Quality health care is directly related to quality medical education.[1] For centuries, the medical profession has held this concept as one of medicine's most important credos. In this article I outline what the medical profession in America has done to amplify that concept, note our current position, and suggest future actions. I conclude with a short statement outlining why this whole mechanism of education for quality control should remain a voluntary one of the profession, and that imposition of an outside "recredentialing bureaucracy" by our State Health Department would be detrimental for the profession and the public.

EARLY EFFORTS IN MEDICAL EDUCATION IN THE UNITED STATES

In the frontier days of America very few trained physi-

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cians "braved the new world." Anyone could claim to be a doctor—and all sorts of people did. Slowly a small cadre of physicians trained in Europe arrived and took apprentices. Then "medical schools" developed—mostly proprietary, mostly didactic, supplementing the practical apprenticeships. In the 1840s New York physicians were instrumental in forming the American Medical Association (AMA) primarily because of a felt need for setting higher standards for education at the national level.[2] In the 1890s the founding of Johns Hopkins set the highest standards of all—one even had to have a college degree to be admitted. The great watershed in US medical education came with the Flexner report in 1910[3] which recommended high standards for all schools, including a four-year curriculum. Within a short time over half the schools closed.

One of the side effects of this upheaval in medical education was an unfortunate one for organized medicine. Up until that time all of the leaders of the medical profession at large were also leaders in the AMA. After the Flexner report, with the marked emphasis on upgrading the quality of medical school education, many of the leaders of American medicine concentrated upon improving their medical schools and did not participate nearly so actively in organized medicine. Indeed, it is not unfair to say that during the 1920s and 1930s academia tended to look down on those physicians who remained active in the American Medical Association.

CURRENT STANDARDS AND ACCREDITATION FOR US MEDICAL SCHOOLS

In addition to setting standards for medical school education, an increasingly sophisticated system of accreditation has been established to make certain that those standards are, in fact, met. The most recent revisions of these standards were made in 1985.[4] Today each school is surveyed periodically by a high powered team representing the AMA and the Association of American Medical Colleges (AAMC).

To prepare for the accreditation survey each medical school is required to do an extensive "self-study" which is then used as part of the survey team's evaluation after a four-day visit to the school critically reviewing every element that might have an impact on medical student education. The survey team reports are further reviewed by some 50–60 reviewers prior to final recommendations by the Liaison Committee for Medical Education. Even the best schools do not escape some recommendations for improvement.

RESIDENCY TRAINING AND BOARD CERTIFICATION

From the 1920s through the 1950s the development of the specialty societies and specialty boards was a very important factor in setting the standards for internship and residency training and for developing site visits by "residency review committees" to ensure that the standards that had been promulgated were followed. Both the AMA's Council on Medical Education and the American Medical College of Surgeons were instrumental in this development.[5]

Although the medical schools and the specialty societies and boards initially concentrated almost exclusively on educational and scientific matters, in the 1960s they belatedly began to recognize that socioeconomics and "the environment" in which medicine is practiced are just as important for patient care as is the strict attention to education itself, and both groups began to develop policies and actions in the socioeconomic field (although sometimes at odds with the concerns of organized medicine). Parenthetically, it is interesting to note that today 25–35% of the budgets of medical schools come from the professional fees earned by their faculties.[6] Because of this the schools have just as much concern for adequate fees for services rendered as does the practitioner. Also one should note that from the very beginning the concepts of learning through working with patients and of increasing responsibility under supervision have been unique features of American medical education.

We cannot allow our state government to damage this crucial element in the learning process because of its zeal to accommodate to a highly publicized unfortunate event in one of our hospitals.

CONTINUING MEDICAL EDUCATION

During the last half of the 20th century there has been such an explosion in information that knowledge has doubled every few years. Much of what one learned in medical school was out of date within a decade or so after leaving formal educational programs. While medicine had always been considered a profession where "lifetime learning was vital to the care of the patient," it has become even more important to try to ensure that physicians keep up to date as much as possible after leaving their residency training programs. Continuing medical education is personal and self-directed. Bill Ruhe, then an AMA vice-president, showed a few years ago that physicians feel their most important continuing medical education is derived from reading books and journals.[7] Nevertheless, more formalized continuing medical education (conferences, courses, and seminars) has been developed, and attention paid to attempts to improve the standards in continuing medical education just as has been accomplished in medical school education and residency training.[8] Although we now have a national Accreditation Council for Continuing Medical Education (ACCME), many of us feel that these more formalized programs at the national level are only a relatively small and less important part of all the things that the profession does to keep up to date. There are now several national organizations committed to a variety of activities in continuing medical education, and one of the most important of these is the Alliance for Continuing Medical Education (ACME). The ACME was organized by a New York physician, Dr William Felch, who has been its executive director since its inception, and it is now headed by another New York physician, Dr Richard Pierson. Thus New York continues to take a leading role in medical education. Many consider CME the most important part of medical education, since it lasts the several decades of a physician's practicing career.

HOSPITAL QUALITY CONTROL AND EDUCATIONAL METHODS

In today's world, the educational programs at the hospital level are one of the most important ways in which we guarantee quality care of the public. These include, but are not limited to: death and complications conferences to analyze problems that occur and devise means of preventing them in the future; chart reviews so that each hospital case is reviewed after discharge for inadequacies or for additional data needed to complete the chart (any problems identified are referred back for action); and tissue committees to make sure that the indications for surgery correspond to the pathological specimens resected. In addition, each specialty and almost every subspecialty in the hospital has frequent conferences covering special cases and subjects of interest in their field. Special lectures, symposia, courses, and other educational programs are organized. Hospital directors of continuing medical education have been appointed in many hospitals to help the educational programs of the entire institution. Quality assurance departments are now standard in most hospitals and use a variety of methods to ensure quality care.

Just as with medical schools and with residency training programs, the process of setting standards for hospitals and ensuring those standards are adhered to has been developed voluntarily by the medical profession which formed the Joint Commission on Accreditation of Healthcare Organizations (JCAHO) for this purpose. While this process remains important, the JCAHO today is trying to go one step further and evaluate outcomes, to make even more certain that all patients receive quality care.

OTHER QUALITY CONTROL MEASURES

In recent years we have developed professional peer review organizations (PROs) all over the country and have identified clinical care standards for patients with each kind of illness. Specialized physician reviewers audit cases that have been selected for review by less sophisticated personnel. The concept is to identify in more detail exactly what quality care means and to take a variety of actions to ensure that each patient gets such care. Sanctions of physicians are made in a few instances, although the general approach is an educational rather than a punitive one. Remedial action may be required. Some physicians may be referred to other agencies for further review. In New York State this PRO for Medicare patients is organized under the aegis of the Medical Society of the State of New York (MSSNY) and is one of its largest activities. It is important to note that this process includes review of office based physicians.

Physicians are required by law to report misconduct of their peers to New York State's Office of Professional Medical Conduct (OPMC). All reimbursement systems have increasingly sophisticated quality control mechanisms so that payments are not made for inappropriate care.

The AMA and the Federation of State Medical Boards have organized a nationwide reporting system for serious infractions, adverse licensing actions, etc.

The MSSNY has a Committee for Physicians' Health to identify and treat physicians with substance abuse or mental health problems. These activities are being extended into medical schools.

FACTORS BEYOND PHYSICIAN CONTROL THAT AFFECT PATIENT CARE

It is quite clear that our profession has set standards for education and training that translate into the best medical care in the world. And we continue to fine tune all of these things that are necessary for such care. But there are other factors that affect the quality of patient care and many of these relate to the role of government. Three of these are illustrative: First and foremost, appropriate changes in our life style, individually and collectively, would make a greater impact on improving our health than all other health activities combined. Yet we spend a fraction of 1% of the health budget on health education in our schools and health promotion in adults. We must demand that our state government do a better job. Problems related to drug, alcohol, and tobacco use take a huge toll on our people (especially the young) and use up a large portion of our health resources.

Second, the bureaucracy surrounding Medicaid and Medicare has become stifling. With each successive set of rules and regulations to close smaller and smaller loopholes physicians are more and more restricted in trying to deliver quality care. All of us—government, physicians, and patients—are spending so much time and effort on paperwork and process that patient care often takes second place. Major overhauls are desperately needed. The MSSNY is joining the AMA to fight for such changes.

Third, we need a large number of new nursing home beds and chronic care facilities at a cost we can afford. Large numbers of older people remain in acute care hospitals, sometimes for over a year, because there aren't any alternatives for them. Chronic care beds are simply not available. Rules and regulations are such that it costs almost as much to build and maintain a nursing home bed as it does for a hospital bed. The huge spectre of AIDS looming over us compounds the problem of ensuring an adequate number of beds and other resources. We will continue to urge state government to correct this huge deficit.

However, there are other important failures of government in relation to health, particularly with respect to the health needs of minorities: Many of our patients do not have enough education to understand how to utilize the medical care system available to them; many patients are sick because of inadequate nutrition; we treat victims of rat bites, lead poisoning, and crime, and then send them back to the ghetto for repeat performances; we develop high technology medical care but do not ensure that it is available to everyone—millions of Americans have no insurance at all.

In summary, adequate education, adequate nutrition, adequate housing in a low crime environment, adequate income for the necessities of life including health insurance are all essential for good health. The medical profession cannot ensure all these things. Government must take a leading role to set the stage so these problems are overcome.

In this context I would like to comment on the New York State Department of Health's plan for the "recredentialing" of physicians. As I have demonstrated, the profession itself has continuously developed and refined many mechanisms for quality control. The huge and vital difference is that now the Department of Health wants to have control of this process and, therefore, control of the profession. The Department of Health wants to develop a state bureaucracy to be able to search out and identify the smallest criticism (whether valid or not) for each individual physician and to use this information for its own purposes. To try to achieve 100% perfection by requiring increasingly stringent rules and regulations would strangulate the profession. The Medical Society of the State of New York and, indeed, all the physicians in New York will simply not tolerate such control. We already have many mechanisms to identify errant physicians, and they should be penalized appropriately; but to stifle the profession with an extremely expensive and time consuming bureaucratic morass makes no sense at all.

Shall we refuse to deal with the commissioner? Of course not! Our state government has a huge stake in the health of the public. We must continue to interact but we cannot become subservient to authoritarian bureaucratic control. Let us demand that the commissioner and state government turn from trying to dictate how doctors should practice medicine and expend more efforts in the other areas that have a huge impact on the health of our citizens.

SO WHAT OF THE FUTURE?

We must continue to fine tune our system of medical education in directions that further guarantee quality care. The report of the Council on Medical Education, "Future Directions for Medical Education," lists some 42 such recommendations.[5]

The MSSNY needs to work more closely with the specialty medical societies to give them support for their scientific meetings, other educational programs, and to develop pilot projects for quality care.

The MSSNY needs to collaborate with our 14 medical schools to strengthen continuing medical education projects in their regions, to collect information on educational needs, to identify system and individual failures, and to devise solutions to problems found, including remedial programs.

The MSSNY needs to combine forces with the Hospital Association of New York State to further strengthen educational and quality control mechanisms in our hospitals, which is probably the single most important focus we have—that is where the real action is.

Since costs considerations are forcing us more and more to practice outside the hospital, we need to devise better self-instructional and other educational methods for physicians in their own offices.

And finally, to do all of these things the MSSNY must strengthen its education division.

I believe we can do all these things. We must do all these things if we are to retain control of our profession.

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Professional medical discipline: An overview

CHARLES J. VACANTI, MD

This oft quoted line, "Physician heal thyself," from the gospel of Luke is used to remind the medical profession of its great responsibility to maintain a high degree of professionalism and discipline within its ranks to ensure that the public will receive high quality medical care. During the 19th century in New York State, medical societies granted, and at times revoked, licenses to practice medicine. Since the turn of the century in New York, the board of regents has been charged with the ultimate responsibility for regulating, disciplining, and otherwise insuring the competence of all who practice medicine and are licensed under Title 8 of the Official Compilation of Rules and Regulations of the State of New York.

In actual practice, the New York State Board of Regents regulates the practice of medicine by first approving all educational programs that lead to a degree in medicine. Next they issue licenses to applicants who have demonstrated their competence in a manner prescribed by the regents (ie, board examinations). Licenses are currently registered triennially, but as a general rule, once a person has been granted a license to practice medicine, that license is valid for life unless action is taken by the state for reasons of misconduct. Finally, the board of regents oversees all who practice medicine and disciplines those who are proved guilty of professional misconduct.

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This article represents the personal views of the author, and does not represent the official policy of the New York State Department of Health or the New York State Board for Professional Medical Conduct.

THE STATE BOARD FOR PROFESSIONAL MEDICAL CONDUCT

Title II-A of the Public Health Law, enacted in 1975, directed the creation of a State Board for Professional Medical Conduct which assumed from the board of regents the responsibility for investigating all complaints against physicians and conducting initial disciplinary proceedings. This same Public Health Law (§ 230), which has been amended several times by the legislature since 1975, created the Office of Professional Medical Conduct within the State Department of Health. In its current form, § 230 authorizes the investigation of any complaints of medical misconduct; describes the process whereby the charges of medical misconduct are heard; mandates the reporting of misconduct by licensed providers and administrators of licensed facilities; authorizes the Committee for Physicians' Health of the Medical Society of the State of New York; provides a mechanism for the temporary surrender of the license of an incapacitated physician; and authorizes the Commissioner of Health in cases of imminent danger to the public to summarily suspend a physician's license for a finite period of time during which a hearing will take place.

It is through the professional discipline process, as delineated in this law, that a sanction may be taken against a physician's license. And that is the only way a license to practice medicine in the State of New York may be limited or revoked.

The principal definitions of medical misconduct in the State of New York are:

- practicing the profession fraudulently, beyond its authorized scope, with gross negligence or with gross incompetence on a particular occasion, or negligence or incompetence on more than one occasion;
- practicing the profession while the ability to practice is impaired by alcohol, drugs, physical disability, or mental disability;
- being convicted of committing an act constituting a crime under New York State or federal law, or the law of another jurisdiction and which, if committed in this state, would have constituted a crime under New York State law;
- failing to make available to another physician, upon a patient's request, x-rays or records prepared for and paid for by the patient;
- willfully making or filing a false report, or failing to file a report, required by law, or inducing another person to do so;
- ordering excessive tests or treatment;
- abandoning or neglecting a patient in need of immediate care.

The Board for Professional Medical Conduct is a group of volunteer public servants, approximately 180 physician and lay members from all areas of the state, appointed by the commissioner of health with the approval of the governor. The physician members are appointed, by law, only upon "recommendations made by the Medical Society of the State of New York, the New York State Osteopathic Society, the New York Academy of Medicine, county medical societies, any organization of physicians licensed to practice in the state and the Hospital Association of New York State." Approximately three quarters of the members of the board are practicing physicians in various specialties from around the state. All board members fulfill several responsibilities. The physician members assist in the preliminary review of completed investigations to determine if sufficient evidence or information has been uncovered that would warrant a formal hearing. This is done by means of an investigative committee of the board which is convened approximately twice a month to review complaints/cases in order to determine the appropriate disposition.

THE PROCESS OF DETERMINING PROFESSIONAL MISCONDUCT

Once it is determined that allegations of misconduct warrant a hearing, the case is forwarded to the Office of Counsel where there is a separate Professional Medical Conduct Bureau which handles only professional medical conduct proceedings. An attorney is given the case to prepare charges which incorporate the state's evidence. The physician respondent is then given an opportunity to defend himself or herself before a second committee of the board (two physicians and one lay member) which is constituted to hear the case. This hearing committee of the board hears both the state's evidence and the physician's defense. The committee makes findings of fact, draws conclusions, and recommends a penalty if charges are sustained.

The hearing process varies and may last from one day to several days. Hearings are complex administrative and legal proceedings which are vigorously contested because often the physician's license is at stake. This process, because it must afford the respondent full protection and due process under the law, is often lengthy. It may be spread out over several months or even years due to scheduling difficulties and other delays.

At the conclusion of the hearing, the hearing committee issues a report incorporating its findings and its recommendations. The entire case, including transcripts of the hearing and all of the exhibits that have been accepted as evidence, are forwarded to the commissioner of health who, in turn, reviews the entire record and makes an independent recommendation. The case is then forwarded with the board hearing committee report, the commissioner's report, and all the transcripts and exhibits to the New York State Board of Regents, which, through its own procedures, reviews the case, affords the physician another opportunity to be heard, and ultimately makes the final decision on the guilt or innocence of the physician and the penalty to be imposed.

The process from the reception of a complaint to a final resolution by the regents is lengthy and cumbersome, involving five stags of review. This process is summarized in Table I. There are approximately 55,000 licensed physicians in the state of New York. However, not all currently practice or reside within the state. In 1987, 3,429 complaints were received by the Office of Professional Medical Conduct. These resulted in 297 final disciplinary actions (see Table II).

TABLE I. The Process of Professional Medical Discipline in New York State

A **Complaint** is reviewed to determine if there are issues that warrant an investigation. The investigation may consist of personal interviews, obtaining documents, including medical records, and consultation with medical experts.

The completed **Investigation** is reviewed by supervisors and medical staff. If there is no evidence of misconduct, the case is closed.

A **Committee of the Board for Professional Medical Conduct** reviews all cases where there is possible misconduct and recommends whether a hearing is warranted, or the case is closed.

A Department of Health attorney prepares a Notice of Hearing and **Statement of Charges** which describes the substance of the alleged misconduct.

The **Hearing** provides the physician, with his/her attorney, an opportunity to present witnesses and evidence on his or her own behalf.

The hearing is before a committee of two physicians and a lay member of the Board for Professional Medical Conduct and an administrative law judge which makes the **Findings** as to whether the evidence supports the charges and recommends a penalty, if appropriate.

The **Commissioner of Health** reviews the case and makes his recommendation.

The **New York State Board of Regents** makes the final decision on all physician misconduct cases. The Regents may order:

- Revocation;
- Suspension (wholly or in part);
- Restriction of a license;
- Censure and reprimand;
- Probation;
- 100 hours of public service;
- A fine of up to $10,000 per violation;
- A course of education or training; or
- Dismissal of the case.

TABLE II. Activities of the New York State Office of Professional Medical Conduct, 1987

Activity		Number
Complaints received		3,429
Completed investigations		3,380
Cases referred for hearing		254
Cases referred for warning		87
Final disciplinary actions		297
Surrender	20	
Revocation	79	
Suspension	150	
Censure and reprimand	29	
Censure, reprimand, and probation	4	
Community service	1	
No further action	2	
Dismiss	12	
Total	297	
Temporary/permanent surrenders of medical license	33	

SOURCES OF COMPLAINTS

Patients and their families are the most frequent source of complaints. However, reports from hospitals and physicians are the most fruitful. The New York State Public Health Law mandates that all persons licensed pursuant to the Education Law, and administrators of all Article 28 licensed institutions, report information which reasonably appears to show that a physician is guilty of professional misconduct. Hospitals must report the suspension, restriction, termination, or curtailment of training, employment, association, or professional privileges or denial of certification of completion of training of an individual for reasons related to alleged mental and physical impairment, incompetence, malpractice, or misconduct, or impairment of patient safety or welfare. Facilities must also report resignation by a physician to avoid imposition of disciplinary measures and the receipt of information which indicates that a licensee has been convicted of a crime.

IMPAIRED PHYSICIANS

The Impaired Physicians Program was established to divert physicians suffering from alcoholism and/or drug abuse to rehabilitation and treatment as opposed to the lengthy disciplinary process. The Office of Professional Medical Conduct works with the Medical Society of the State of New York which operates an intervention, referral, and monitoring program for impaired physicians. The law permits impaired physicians to temporarily surrender their medical license, undergo rehabilitation, and apply for restoration of the license once recovery has occurred. A board committee conducts a hearing at which the physician presents evidence or proof of rehabilitation, and the committee recommends to the commissioner of health whether the license should be restored. Once the license is restored, the physician is monitored for a minimum of two years. Monitoring consists of reports of random blood samples or urinalysis, and reports from a supervising physician who monitors the practice. Drug abuse accounts for the majority of identified impaired physicians. It appears that physician and hospital staff colleagues are much more reluctant to confront or report physicians who have problems with alcohol. It is the goal of the Office and the Board for Professional Medical Conduct and the Medical Society of the State of New York to raise the awareness and consciousness of hospital administrators, trustees, and physicians to the problem of the impaired physician and the options for treatment available.

SUMMARY

The new, current process of professional medical discipline in New York State is effective but cumbersome. The complexities associated with defining and proving professional misconduct together with the necessary due process provisions of the current law result in a time consuming process. There is room for improvement. It is clearly in the interests of the medical profession to have a strong, visible, viable, and effective program of professional medical discipline.

Concentrating on the problem physician: Perspectives in medical discipline

BRYANT L. GALUSHA, MD

The Federation of State Medical Boards is *the* national organization of medical licensing and disciplinary boards and is made up of the medical boards of all states, the District of Columbia, Guam, the Virgin Islands, and Puerto Rico. Eleven of the 16 independent osteopathic medical boards are also members. Additionally, the Canadian provincial medical licensing authorities are affiliate members. At the direction of the member boards and on behalf of the people they serve, the federation contributes significantly to the effectiveness and integrity of the medical licensing and disciplinary systems in the United States, systems which are essential components of medical quality and physician accountability.

It would be difficult to overemphasize the importance of the challenge the professional liability crisis presents American medicine. It may seem a profound irony that in these closing decades of the 20th century, in the face of the remarkable accomplishments and incredible potential of medicine, there is, simultaneously, a growing emphasis on questions of professional incompetence and impairment. Yet more surprising would be the absence of such concern in view of the ever-increasing complexity of medical science and its essential role in modern life, and in view of the fact that health care is recognized as one of the nation's biggest businesses by consumer groups, government, insurance firms, labor, the media, and others. The problems of liability in an increasingly litigious environment plague and often threaten to destroy not only physicians and hospitals—early victims—but virtually every element of the national community, from banks to the Boy Scouts and from architects to ice rinks. Defense costs are often obscene and threaten to destroy the very concept of justice. Insurance premiums are confiscatory and insurance itself is virtually unavailable for many. Adverse judgments are often financial death sentences for individuals and organizations. This crippling situation demands the attention we all give it. As Dr Samuel Johnson said in the 18th century, "When a man knows he is to be hanged . . . it concentrates his mind wonderfully." Heaven knows, medicine is concentrating today!

It would be easy to point out all that is good and fine in American medicine, to cite the hundreds of thousands of skilled, conscientious, and dedicated physicians who serve the American people so well, and to bewail the faults of a system which appears to have lost its sense of balance in pursuit of a strangely distorted and retributive form of justice. But the liability crisis, severe as it has become, cannot be allowed to obscure the painful reality that there are physicians whose practice of medicine is, for whatever reason, unprofessional, improper, and incompetent.

Physicians are all too human, they are a part of Everyman, as subject to his frailty as to his nobility. We would have them no other way. But the privilege of practicing medicine demands of physicians an obligation far beyond the ordinary, a fact so simple that it is too easily forgotten by some. Negligence, ignorance, deliberate disregard for professional responsibility, deficiencies in skill and knowledge, greed—these are the ingredients of true malpractice and they deny the physician's obligation. It is far too simplistic to say that the solution to the present malpractice problem is to end malpractice. Human error is inevitable—the poor result will occur despite the finest efforts of the brightest and best. And the law will see to it that redress is made. The end of the medical malpractice problem, legally, can only be achieved by ending medical practice itself.

Medical licensing and disciplinary boards are charged by the people, acting through their elected representatives, with the duty of preventing admission to the practice of medicine of those unfit under law to assume that privilege and of taking judicious and appropriate action against those whose practice fails to fulfill the fundamental requirements of the law. And while the language of the law may differ from one jurisdiction to another, the intent is always the same; to protect the public from the unprofessional, improper, and incompetent practice of medicine. In granting licenses and performing disciplinary functions, boards must be active partners with several voluntary agencies in a complex process of quality assurance, a process that includes, in addition to evaluation of candidates for licensure, the accreditation of educational institutions, certification procedures, and peer review.

The responsibility of medical licensing and disciplinary boards to protect the public must begin at the time of original licensure. Surely, it is easier and far cheaper to deny initial licensure to an unqualified or unfit physician than to revoke or limit a license once given. To short the licensing process betrays public trust and predictably will result

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in increased disciplinary problems and costs. Such an approach, one that emphasizes the remedy of discipline over the prevention of sound licensing, is patently illogical. There are those applicants who present false credentials—effective verification procedures are essential. There are medical school faculties and residency program directors who will rubber-stamp a candidate rather than point out even serious problems to a board—precise evaluations at all levels of the educational process are required and the boards, in an appropriate way, should have access to such information. In fact, the federation advocates a form of limited, educational license for residency training, in part to stimulate a more effective review and evaluation system prior to the application for full licensure. Indications of trouble may well be available in the public record as well—court records, etc. Candidates should also be reviewed against the records of other states—mobility can be an effective smokescreen. And licensing authorities should not neglect the value of the personal interview in triggering recognition of potential problems.

ENDORSEMENT

In this same vein, the federation is urging that medical boards require written clinical examinations, such as the federation's new Special Purpose Examination (SPEX), for those physicians who seek licensure by endorsement of another state's license and who are eight to ten years away from any other testing approved by the medical board to which the physician is making application. Some states now have this policy and the federation hopes all will adopt it to assure that physicians who leave their established practice and peer review environments to enter new jurisdictions have maintained their qualifications over time. The SPEX, a one-day examination, is also appropriate for re-examination of specific physicians for whom a licensing board determines the need for a current demonstration of medical knowledge. It should be stressed that SPEX is not designed or intended as a routine competency examination for all physicians for relicensure or periodic license reregistration.

Altogether, boards should do all in their power to license only those who are able and willing to practice medicine with reasonable skill and safety. Prevention is preferable to remedy.

The development of a strong state licensing examination in the FLEX, the growth of sophisticated information systems such as the federation's Physician Disciplinary Data Bank, the newly developed Special Purpose Examination, improved medical practice acts in many jurisdictions, better communication among boards, and more, have marked a steady progress in what was once a perfunctory licensing process. But, unfortunately, this is not the best of all possible worlds, and licensure, by examination or endorsement, is not always what we would have it be, while some physicians, once licensed, fail to maintain the professionalism they first bring to practice—a problem nothing short of clairvoyance may ever effectively resolve.

DISCIPLINARY ACTIVITIES OF BOARDS

The granting of a medical license is only the beginning of the medical board's responsibilities. Equally important is the board's obligation—its duty—to exercise the authority vested in it to protect the public from unqualified, unfit, and impaired physicians—to identify those physicians through the systematic review of meaningful markers and indicators of performance and fitness, to act against them in accord with law, and to prevent their undetected movement from one jurisdiction to another. This is a formidable task that must be pursued diligently while not interfering needlessly with the practice of medicine by the overwhelming majority of capable and fine physicians.

The need to identify possible problem physicians is fundamental to the work of the boards. Never easy, the task is almost impossible if a board must act in an information vacuum. The so-called conspiracy of silence among physicians has been no myth, but clearly it is dying away in the face of today's realities. It was never an option, in any case. It was only a self-destructive denial of professionalism. The most elemental understanding of self-interest dictates that the unfit practitioner should be identified and appropriately dealt with in order to protect the public as well as the integrity of the profession.

MANDATORY REPORTING

To answer the need of the boards for information identifying the possible problem physician, most states have enacted legislation requiring that such information be reported to the boards by certain persons, institutions, and organizations. Though these compulsory reporting laws vary from state to state, they usually require reports by health care facilities, liability insurance carriers, and individual licensees. They may also require reports from courts, law enforcement agencies, and medical societies. With the exception of individual licensees required to report apparent violations by colleagues, reporting laws usually involve informing the medical boards of specific formal disciplinary or legal actions. However, in situations in which a physician withdraws from a medical staff while under review for disciplinary reasons, that fact may also have to be reported, as it should be. Essential to mandatory reporting is protection for those who report. When acting in good faith, they must be assured immunity at both state and federal levels. Similar protection must extend to medical board members, staffs, and committees and to all those engaged or assisting in the process of legitimate professional peer review. Equally important, however, is the legal protection of the accused physician, who must be assured of due process and must be shielded from harassment.

The federation actively encourages these mandatory laws. The federation's *Guide to the Essentials of a Modern Medical Practice Act* states this position clearly.[1] A licensing and disciplinary board can be no stronger than the statute on which it is built. The statute, usually referred to as the Medical Practice Act, is the foundation. If it is weak, the board will be weak. The federation has placed strong emphasis on improving and modernizing medical practice acts. The *Guide*, revised every three years, has played an important role in this effort. In fact, the 1985 edition of the *Guide* has been instrumental in the partial or complete revision of the practice acts of at least 23 states—a notable achievement. The 1988 edition predictably will have further positive influence over the next several years.

LICENSE REREGISTRATION

Many boards have now adopted more effective license reregistration rules to require the filing of a form on which the licensee must report any sanctions taken against him or her during the reregistration period, usually one to three years. The number of states in which a physician could simply mail in a check to reregister a license is decreasing noticeably, as it should. Boards are coming to recognize that reregistration represents a specific moment in time when each licensee should be reviewed in a systematic way. This enhancement of reregistration, coupled with a strong federation effort advocating a system of review based on currently available markers or indicators of possible performance or fitness problems, is leading to the identification of more problem physicians while not burdening the vast majority of able physicians with needless and time-consuming procedures.

MARKERS AND INDICATORS

The markers and indicators of possible performance or fitness problems include, among others, disciplinary actions by other states, adverse privileging actions by hospitals, other sanctions springing from peer review processes, and malpractice judgments and awards. In the majority of states, these markers (referred to as red flags by many boards) are presently being made available through mandatory reporting laws and from other sources such as the federation's Physician Disciplinary Data Bank which in its present state of sophistication is indeed a powerful credentialing aid for both medical boards and hospitals. When this information is combined with required self-reporting on licensure reregistration forms, boards have available a coherent approach to identifying possible problem physicians by revealing significant events or, more likely, patterns of events indicating questionable performance or fitness. Once identified, physicians who present concerns to the medical boards can be more precisely evaluated through informal interviews, physical and/or mental examinations, cognitive and/or skills examinations, practice profile investigations, etc. In the federation's view, if the medical boards are properly funded to implement the markers and indicators system and if enforceable mandatory reporting is universally adopted, effort will be concentrated where it belongs—on those who have demonstrated inadequate professional ability or fitness.

I have not yet alluded to elaborate and costly systems for evaluating the competence (or standard of practice) of all physicians in the process of licensure reregistration or periodic relicensure. First, I seriously doubt that is possible. Competence, as simple as the word seems, is an elegantly complex concept that defies our limited assessment capabilities. Second, it is not economically justified—the cost benefit ratio of the effort, flawed in any case, would be terribly skewed. Third, I do not believe it is necessary in order to achieve the purpose of protecting the public. Years ago, during the trial of Willie Sutton—that intelligent, debonair, and highly successful bank robber—the presiding judge, with obvious reverence, asked, "Mr. Sutton, with all of your talents and grace, why in the world did you rob banks?" As many of you recall, Willie immediately replied, "Because, judge, that's where the money is!" And so it is with this issue—if we are striving to identify the problem physician, we have to look where he or she is to be found—not at the whole universe of physicians. Look for the signs of incompetence, not competence.

The federation has followed the work of the New York Advisory Committee on Physician Recredentialing with great interest for good reason. Clearly, the committee was given a difficult task and attempted to address its responsibility. The members of the committee in accepting this daunting challenge certainly deserve the respect of the medical community and the citizens of the state. Appreciating as I do the effort that went into the committee report, I wish it were possible for me to accept the proposals it presents. But I cannot. I shall not detail my concerns. My worst fear, which I believe to be a valid one, is that if these proposals go forward, the total effort may degenerate into a mass testing system keyed to the lowest common denominator. Even worse, it could possibly deceive the people of New York State for a time into believing they are actually protected from incompetence and turn attention away from effective methods the state could be using to identify those who are incompetent. That would be a tragic irony.

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Position paper of the Medical Society of the State of New York on physician recredentialing

The Report of the New York State Advisory Committee on Physician Recredentialing entitled "Phase One: General Principles, Proposed Process, Recommendations" was released in January of 1988. The Medical Society of the State of New York does not endorse this report or its recommendations.

Historically, quality assurance (optimal patient care) and physician competency have been the foundations upon which the policies and positions of the MSSNY have been developed. Over the past decade, the assurance of quality and maintaining of competence, a responsibility which rightly belongs within the purview of the profession (all professions), has been gradually assumed to a large extent by agencies external to the profession. Perhaps well intentioned, the regulatory mechanisms developed by these external agencies have had a deleterious effect on the delivery of medical care but have had little impact on physician clinical performance.

The MSSNY agrees with the statement, made on several occasions in the report, that the recredentialing process broadly outlined in the report "is not designed to measure medical competence." Indeed the report does little more than discuss those well known methods used to evaluate those various, individual components which taken collectively are used to define knowledge and cognitive skills, not performance.

We agree with the report that there does not exist a single methodology for measuring competency and agree that employment of a combination of methodologies to measure competency would be logistically and economically unrealistic. The evaluation of competence in the health professions has not yet reached maturity. Measurement of changes in practice as a consequence of additional education, assessment of the validity of examinations and the determination of goals for competence are all necessary parts of the ongoing development of competence evaluation.

As stated in the beginning of this statement, MSSNY is committed to quality assurance and maintaining competence of health professionals. However, we do not need further government intrusion to do what already is being done. Accordingly, the MSSNY subscribes to the following recommendations of the "Health Policy Agenda for the American People."

- Health professionals are individually responsible for maintaining their competence and for participating in continuing education; all health professionals should be engaged in self-selected programs of continuing education. In the absence of other financial support, individual health professionals should be responsible for the cost of their own continuing education.
- Professional schools and health professions organizations should develop additional continuing education self-assessment programs, should prepare guides to continuing education programs to be taken by practitioners throughout their careers and should make efforts to ensure that acceptable programs of continuing education are available to practitioners.
- Health professions organizations and faculty of programs of health professions education should develop standards for competence. Such standards should be reviewed and revised periodically.
- When reliable and cost-effective means of assessing continuing competence are developed, they should be required for continued practice.

This should be done without government interference or control.

This position paper was adopted by the House of Delegates of the Medical Society of the State of New York (MSSNY) at its April 1988 meeting.

The MSSNY is closely monitoring the developments generated by the report of the New York State Advisory Committee on Physician Recredentialing. In addition, it is preparing a comparative analysis of the positions stated by other interested parties. An ad hoc committee has been appointed by the president of the MSSNY to develop standards for the assessment of competence that are consistent with the MSSNY's position statement.

Position statement of the Hospital Association of New York State on the Report of the New York State Advisory Committee on Physician Recredentialing

The January 1988 Report of the New York State Advisory Committee on Physician Recredentialing contains a series of recommendations to institute a physician "recredentialing" system in New York State. The Report recommends that physicians be "recredentialed" once every nine years as a condition of continued eligibility to practice medicine. Under the proposed system, a physician could select one of three "recredentialing" options: (a) submit evidence of specialty recertification by a national specialty board; (b) pass a new, State-administered examination; or (c) submit evidence of successful completion of a peer review process using hospital recredentialing and reappointment processes (for physicians with significant hospital inpatient practices) or a medical records review (for physicians with primarily office-based practices).

The Hospital Association of New York State (HANYS) endorses the principle that the periodic evaluation of the standards of a physician's practice, to determine competency for continued practice, should be encouraged. Such a process promotes the provision of quality care to which all of our member institutions are committed. Whether the process proposed by the Advisory Committee, or any process, will achieve the Report's stated goal of "... prevention of medical errors" is an unanswered question.

In proposing a "recredentialing" system, the Report raises numerous complex questions involving 1) regulatory authority to implement the system, 2) confusing definitional terms, 3) potential imposition of conflicting purposes on existing hospital recredentialing activities, and 4) questionable testing procedures. Each of these issues is discussed below.

STATUTORY AUTHORITY

Included in Appendix 1 of the Advisory Committee's Report is an opinion by the General Counsel of the Department of Health that the Board of Regents currently possesses the statutory authority to adopt regulations to "... require the periodic re-examination of physicians' credentials as part of its authority to supervise the licensing and practice of medicine."

It is clear, however, that the "recredentialing" system as presently proposed is not authorized by statute. In addition to lacking statutory authority, the proposal advances a system which conflicts with existing statutes in many fundamental respects. In order to implement the proposal, legislative amendment to the Education Law will be necessary to overcome the many statutory barriers which prevent regulatory implementation. The following is an analysis of existing statutory provisions.

Under the Education Law, the privilege to practice is granted based on satisfaction of two conditions: obtention of a license and maintenance of registration. The question of statutory authority involves an analysis of each of these components since the two together are the basis for the privilege to practice. Failure to satisfy either may properly be grounds for loss of the privilege.

Subdivision 1 of section 6502 of the Education Law states: "A license shall be valid during the life of the holder unless revoked, annulled or suspended by the Board of Regents." Thus, it is clear that once obtained, a license is valid for life absent a revocation, annulment or suspension by the Board. Could the Board use the proposed "recredentialing" process to suspend, annul or revoke a license?

The Education Law specifies how and under what circumstances the Board of Regents may take action against a licensee. Section 6511 provides that certain penalties may be imposed on a licensee *found guilty* of professional misconduct. The penalties include censure and reprimand, whole or partial license suspension, license revocation, license or registration annulment, a civil fine, and other penalties. However, the penalty may be imposed *only after* the licensee is found guilty of professional misconduct.

The activities constituting misconduct are enumerated in sections 6509 and, for physicians and certain other professionals, 6509-a. Neither section refers to "credentialing" or "certification" actions. Rather, these sections refer to activities which may or may not lead to loss of credentials or certification. The statutory definitions of misconduct are neither coterminous nor synonymous with standards for determining credentials or certification. Thus, the activities used to determine credentials or certification are not within the ambit of the statutory definition of misconduct.

The Education Law further details the steps which must be taken before a determination of misconduct may be made. For physicians, the procedures are detailed in section 6510-a, which incorporates the procedural requirements found in section 230 of the Public Health Law. These statutes contain specific and detailed procedures which must be followed if misconduct is to be found. The Advisory Committee Report ignores these procedural requirements and instead proposes a completely different process for determining whether a license may be suspended, revoked, or annulled.

Thus, neither the substantive nor procedural laws of the State support the position that actions may be taken against a physician's license in the manner proposed by the Report. If the "periodic re-examination of credentials" is to result in license suspension, annulment or revocation, then it is only permissible and authorized if it accords with the provisions of sections 6502, 6511, 6509-a and 6510-a of the Education Law and section 230 of the Public Health Law.

Alternatively, it may be posited that the proposal in substance involves the imposition of new conditions on registration; failure to satisfy the conditions would prevent a licensee from registering and thus practicing. Here again, the Education Law is clear. Subdivision 3 of section 6502, in pertinent part succinctly states: "The department *shall renew* the registration of each licensee upon the receipt of a proper application, on a form prescribed by the department, and the registration fee" (emphasis added).

Section 6524 specifies the fees which must accompany a physician's registration. It is important to note that section 6502 imposes a statutory directive on the Education Department: it *must* renew the registration upon the submission of an application form and the fee. No discretion to deny registration is vested in the Education Department. The statutory mandate, together with the absence of other statutory language which even implies that the Department or the Board of Regents could adopt regulations to supersede or qualify the mandate, lead to the inescapable conclusion that the 6502 requirements are the *only* authorized limits on registration renewal. Thus, other regulatory modifications or other conditions would have no statutory foundation.

The General Counsel's opinion refers to the Education Department's authority to, *inter alia*, administer licensing examinations under section 6507. First, this is a ministerial authorization, not a grant of authority to create new mandates (beyond those specified in statute) as to who must take the exam. Secondly, the section refers to *licensing* examinations and reexaminations. Since section 6502 states that a license is for life, the 6507 provision cannot be read as meaning that a holder of a valid license must submit to a reexamination in order to remain a holder of the license (absent a formal misconduct finding of guilt). Third, the "reexamination" reference is further clarified in section 6524 which provides that, to *obtain* a license, an examination must be passed and that certain fees are required for taking an exam or reexamination upon application for an initial license. A "reexamination" is only necessary when an applicant for a license initially fails the first exam. Once an exam is passed, no further examination requirement is imposed, unless it is subsequent to a misconduct finding.

Since the Education Law governs the practice of medicine, its provisions are the guideposts for determining whether the proposed "recredentialing" system rests on an adequate statutory foundation. As the above references indicate, specific duties and conditions have been enacted which preclude the regulatory imposition of additional criteria for the retention of the privilege to practice medicine.

Definitional Issues

The Report of the Advisory Committee on Physician Recredentialing somewhat interchangeably employs three terms: recredentialing, certification, and relicensure. The use of the terms is confusing, misleading, and a distortion of their accepted definitions.

"Recredentialing" is a process whereby a private entity, usually a hospital, determines that the professional activities of a physician meet standards developed by that entity for the purpose of concluding that the physician may perform professional activities within and as a part of the operation of that entity. "Credentialing" and "recredentialing" result from a weighing of two factors: the expertise of the physician and the needs and standards of the hospital.

"Certification" is the process of documenting that a physician meets uniform standards of practice which are applied geographically, ie, nationwide. Thus, "certification" is documentation by a private national organization that a physician meets the organization's practice standards, usually regarding specialty or subspecialty practice. Certification, however, is not conditioned on an applicant's satisfaction of certain hospital-specific standards which are involved in the credentialing process.

"Relicensure," in terms of New York law, is a misnomer. Physicians, and all professionals licensed by the Board of Regents, are licensed only once. The license must be accompanied by a registration in order for the individual to practice legally. Currently, licensees must register every three years; they are not re-licensed nor are licenses "renewed." The periodic requirement for practice is registration, nor relicensure. The granting of the license and registration are State, not private, actions and are minimum threshold requirements in the credentialing and certification evaluations.

The Advisory Committee's use of these terms has blurred distinctions (in terms of purpose and process) among the three. Since the apparent purpose of the Report was to establish a framework within which the State could revoke the privilege to practice medicine every nine years, the issue is more appropriately framed in terms of either making registration contingent upon the satisfaction of certain conditions or acting to suspend or revoke a license. As explained above, however, either activity requires statutory amendment before it can be authorized under the laws of the State.

Impact on Current Hospital Credentialing Activities

Sections 2805-j and 2805-k of the Public Health Law and their implementing regulations require hospitals to perform periodic reviews of the credentials of physicians with staff privileges. Hospitals are required to collect and maintain information to determine if privileges should be limited, renewed, or not renewed by the hospital.

Much of the information now collected by hospitals is described in the Report as being part of the proposed State "recredentialing" information collection system. It might be concluded that the Committee envisions the mere submission by each hospital of the names of physicians it has recredentialed and that the underlying information collected by the hospital remains in and with the hospital. The submission of the physician's name, it could be argued, is therefore all that is required of the hospital. The text of the Report, however, contains little to support this view.

Rather, the Report implies extensive hospital-generated information for a physician to be "recredentialed" by the State. It specifies the type and nature of the "profile" which each hospital must maintain for each member of its medical staff and then indicates that a summary of each profile must be submitted to the State. Using this information, the State would then determine if the physician may be "recredentialed," ie, authorized to continue practice.

In effect, the Report would use the hospital's internal credentialing process for a State purpose: the renewing or revoking of the privilege to practice. It would convert the separate, hospital-specific process to a State determination of overall fitness for practice. Thus, the hospital credentialing process would carry the greatly expanded consequence of being, in effect, a licensing determination. The imposition of this expanded purpose will cause new and irreconcilable conflicts between hospitals and medical staffs and may, by raising the consequential "stakes," dilute the purpose and effectiveness of hospital credentialing programs.

HANYS firmly believes that the distinction between credentialing—a distinct, hospital function—and granting the privilege to practice—a State function—must be maintained. For this reason, the Association does not endorse the use of the individual hospital credentialing process for State purposes as proposed in the Report.

If competence is to be evaluated periodically by the State, the first two options, ie, specialty board recertification or an appropriate State examination, may be preferable. Resorting to use of the hospital credentialing system should only be allowed for a physician who has attempted but not achieved success via options one or two.

In these cases, the physician could submit documentation that he or she has been recredentialed by a hospital (without submission of the hospital's "profile" to the State) as "appeal" evidence only.

The issues of due process, confidentiality, hospital investigation of private office records, and other matters are all raised if hospitals are required to play a major role in the State "recredentialing" process. If this role is rejected, there is no need to explore these complex issues. If the State system proposed in the Report is implemented, however, all these issues—as well as the nature and extent of the data to be demanded from hospitals—must be examined in detail.

TESTING ISSUES

While the Association endorses the possible eventual use of a new State examination, certain testing issues must also be taken into account. Appendix 2 of the Report presents an outline of three tests (the SPEX examination, the CBX Simulation Model, and the Practice-Orientation Examination) that may be utilized as part of the "re-licensing" process. The following comments are offered relative to these three examinations and are based on reviews conducted by HANYS, the Greater New York Hospital Association, and a critique received from George Webster, MD, Senior Vice President for Evaluation and Research, American Board of Internal Medicine, Inc.

Of the three, the SPEX examination is the most detailed and appropriately reflects the matrix developed for the new FLEX. Based on expert opinion in the field of testing, HANYS strongly feels that, while the CBX Simulation Model holds great promise for further testing, the Report exaggerates the level of the current state of the art. It is felt that while a computer simulation approach may have the highest face validity and acceptability to physicians and the public, currently the CBX methodology is not yet an option. The Practice-Oriented Examination has a credible design, but further work may be required on this examination as well. For example, it is not yet extensive enough to identify specific areas of deficiency for diagnostic or educational purposes based on a 225-item, one-half day examination. Second, the conformance between the content of the written examination and the content of patient problem categories for office record audit may possibly pose a problem. In addition, the NBME pool of questions possibly may not have adequate representation with respect to very common conditions, and may not be focused on the criterion elements of care. Finally, current studies at the NBME indicate that after specialty differentiation has begun it may not be valid to assume that all the modules measure a single underlying dimension of knowledge for an individual physician.

SUMMARY

While agreeing that the periodic evaluation of physicians is in the public interest, HANYS believes that the January 1988 Report contains numerous legal and practical problems, contradictions, and invalid assumptions that all call for a re-evaluation of this particular approach. As written, the proposal raises serious concerns regarding the conduct of peer review activities. Further, it may jeopardize the concerted efforts hospitals and their medical staffs are making to improve quality through peer review. Without a more in-depth, thorough, and thoughtful review of these issues, the implementation of this Report's recommendations would not be in the best interests of the people of New York who are served by its physicians and hospitals.

Position statement of The Federation of State Medical Boards of the United States on the assessment of licensed physicians

The Federation of State Medical Boards of the United States believes it is essential that the population of licensed physicians be systematically assessed at appropriate intervals to identify those whose qualifications and/or fitness for continued licensure are open to reasonable question. This has been the federation's position since 1976. To assist its member boards in the development of workable assessment procedures, the federation established the Ad Hoc Committee to Study Physician Reevaluation for Reregistration, the final report of which was completed in 1982 and adopted by the federation in early 1983. The following principles are fundamental to the recommendations contained in that report and to the position of the federation.

PRINCIPLES OF ASSESSMENT

1. The intent of assessment should be to identify possibly unqualified and/or unfit physicians while not burdening able physicians with needless and time-consuming procedures.
2. Assessment should be an integral part of the license reregistration system system in each state and should occur once in every reregistration cycle (usually two years in duration).
3. Assessment should be based on the review of those markers and indicators of questionable physician performance and/or fitness which arise from peer review processes, liability actions, and other relevant mechanisms.
4. Assessment should work hand-in-hand with an enforceable and broad-based system for the mandatory reporting of possibly unqualified and unfit licensees to the medical board.
5. Assessment should not involve formal cognitive examination, clinical examination, physician examination, or mental examination. These are specific forms of evaluation which should be used only after the assessment process has identified particular licensees in need of such evaluation.

Based on its principles of assessment, the federation advocates the following licensing reregistration system as an approach to assessment.

1. The application form for license reregistration should be designed to require the licensee to update and/or add to the information in the board's file relating to the licensee and his or her professional activity. It should also require the licensee to report to the Board the following information.
   a. Any action taken against the licensee by:
      - any jurisdiction or authority (United States or foreign) which licenses or authorizes the practice of medicine;
      - any peer review body;
      - any health care institution;
      - any professional medical society or association;
      - any law enforcement agency;
      - any court; or
      - any governmental agency

      for acts or conduct similar to acts or conduct described in the medical practice act as grounds for disciplinary action.
   b. Any adverse judgment, settlement, or award against the licensee arising from a professional liability claim.
   c. The licensee's voluntary surrender of or voluntary limitation on any license or authorization to practice medicine in any jurisdiction, including military, public health, and foreign.
   d. Any denial to the licensee of a license or authorization to practice medicine by any jurisdiction, including military, public health, and foreign.
   e. The licensee's voluntary resignation from the medical staff of any health care institution or voluntary limitation of his or her staff privileges at such an institution if that action occurred while the licensee was under formal or informal investigation by the institution or a committee thereof.
   f. The licensee's voluntary resignation or withdrawal from a national, state, or county medical society, association, or organization if that action occurred while the licensee was under formal or informal investigation or review by the body.
   g. Whether the licensee has ever been addicted to or treated for addiction to alcohol or any chemical substance.
   h. Whether the licensee has had any physical in-

   jury or disease or mental illness within the registration period which could reasonably be expected to affect his or her practice of medicine.
   i. The licensee's completion of continuing medical education or other forms of professional maintenance and/or evaluation, including specialty board certification or recertification, within the registration period.
2. The licensee should be required to sign the application form for license reregistration. Failure to report fully and correctly should be grounds for disciplinary action by the board.
3. The board should be directed to establish an effective system for reviewing reregistration forms. It should also be authorized to initiate investigations and/or disciplinary proceedings based on information submitted by licensees for license reregistration.

When correlated with the information obtained through an effective mandatory reporting system, such as that described in *Guide to the Essentials of a Modern Medical Practice Act*[1], and from sources such as the federation's Physician Disciplinary Data Bank, the reregistration process outlined above would provide the medical board markers and indicators identifying possible problem physicians requiring special attention or investigation. With the demographic and practice pattern information which should also be gathered as part of the data base at the time of reregistration, the process would also provide a regularly updated profile of every licensee. The federation believes the information provided a state medical board by a reregistration process such as the one outlined here is the minimum a board should have at its disposal to systematically fulfill its obligation to assess its licensees. To assist boards in establishing such a process, the Ad Hoc Committee to Study Physician Evaluation for Reregistration designed a model reregistration form and process flowchart, both of which have been distributed to the federation's member boards. The federation recommends that all states not now having a meaningful assessment system tied to reregistration of the medical license move to adopt such a system as soon as possible.

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Ondine's curse revisited

CHARLOTTE COLP, MD; ROSLYN SCHNEIDER, MD; DAVID ALTARAC, MD

The story of Ondine is that of a sea nymph who married a handsome mortal. In the play by Giraudoux, when Ondine leaves her husband, he loses involuntary breathing regulation, becomes apneic, and dies.[1] The term Ondine's curse, first used as a medical eponym by Severinghaus and Mitchell,[2] appropriately describes an otherwise healthy patient with alveolar hypoventilation while awake due to primary idiopathic respiratory center malfunction.[3] Such patients are rare, compared to the far more common "Pickwickians" with obesity-related illness. Numerous recent reports have documented sleep abnormalities in obese patients, most frequently obstructive sleep apnea.[4,5]

We recently had the opportunity to study a patient with Ondine's curse and obstructive sleep apnea. The patient was 69 years of age at presentation, and is perhaps the oldest reported with this syndrome. Also of interest was a temporary ventilatory stimulating effect of theophylline in this patient.

CASE REPORT

A 69-year-old man first presented to Beth Israel Hospital on May 30, 1980, with a complaint of syncope and weakness of two days duration. The patient and his wife stated that he had been entirely well all his life prior to the present illness.

On initial evaluation in the emergency room, the physical examination was unremarkable. The patient's height was 62 in and his weight was 134 lb. The lungs were clear on auscultation. His blood pressure was 140/55 mm Hg; pulse, 120 min; stool, 4+ guaiac; hematocrit, 21%. Arterial blood gas showed PO_2 of 61 mm Hg; PCO_2, 47 mm Hg; pH, 7.37. After emergency endoscopy showed a bleeding peptic ulcer, Bilroth gastrectomy was performed. The postoperative course was complicated by multiple, bilateral pneumonias. In addition, the patient required prolonged intubation because attempts at weaning from the respirator repeatedly led to hypercapnea (Table I). A tracheostomy was performed on June 9. The pneumonia gradually cleared, so that by late June only minimal streaking remained on chest radiograph. Measurements performed through the tracheostomy tube indicated an inspiratory force of 30 cm H_2O and a vital capacity of 1.7L. The patient never displayed overt respiratory distress or complained of shortness of breath. Nevertheless, attempts at weaning with intermittent mandatory ventilation (IMV) led to hypercapnea when the IMV rate was 8 or less.

The patient's cannula was removed on July 6 and he was discharged from the hospital with a PCO_2 of 65 mm Hg. He was followed in the outpatient department with a presumptive diagnosis of chronic obstructive pulmonary disease, in spite of a lack of appropriate history. The first pulmonary function study, on August 13 (Table II), was within normal limits except for a minimal reduction of maximum mid-expiratory flow (MMF), which was normal when repeated after bronchodilator. The maximum breathing capacity (MBC) was normal.

The diagnosis of idiopathic hypoventilation syndrome was made in September 1982 when spirometry showed vital capacity and flow rates to be greater than predicted for the patient (Table II). At that time, blood gases remained as before; on inhalation of 100% O_2 for 15 minutes, there was a further rise of PCO_2 (Table I); inhalation of 7% CO_2 led to no apparent patient discomfort and no significant increase in his baseline ventilation of about 3.0 L/min. On voluntary hyperventilation, the patient was able to lower the PCO_2 to 30 mm Hg. The diagnosis of idiopathic hypoventilation was confirmed at

TABLE I. Initial Patient Course

Date	PO_2 (mm Hg)	PCO_2 (mm Hg)	pH	FIO_2	Comments
5/30/80	61	47	7.37	0.2	In emergency room for acute gastrointestinal bleed
5/31/80	64	35	7.48	—	Respirator
6/01/80	71	53	7.31	—	IMV* 6; respirator
6/02/80	110	40	7.53	0.45	Assist control; respirator
6/03/80	46	77	7.23	—	Extubated
6/03/80	69	34	7.59	0.5	IMV* 12; respirator
6/04/80	76	52	7.32	—	"T-piece"
6/09/80	78	42	7.42	—	Respirator/tracheostomy
7/06/81	71	65	7.35	—	Decannulated
9/20/82	59	65	7.34	0.2	Outpatient
9/20/82	453	80	7.25	1.0	Outpatient
9/20/82	—	82	7.20	1.0	7% CO_2
9/20/82	108	30	7.60	0.2	Voluntary hyperventilation
9/19/84	64	60	7.39	0.2	At rest
10/22/86	72	37	7.57	0.2	Voluntary hyperventilation
11/17/86	54	81	7.30	0.2	In hospital
11/19/86	80	56	7.40	0.2	Intravenous aminophylline
11/20/86	65	62	7.33	0.2	Intravenous aminophylline; theophylline serum level 10.2 μg/cc
12/01/86	54	76	7.32	0.2	Oral theophylline; serum level 17 μg/cc

* Intermittent mandatory ventilation.

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TABLE II. Pulmonary Function Tests

	Vital Capacity (L)	Total Lung Capacity (L)	Forced Expired Volume in 1 Second (L)	Maximum Mid-Expiratory Flow Rate (L/sec)	Maximum Breathing Capacity (L/min)	Carbon Monoxide Diffusing Capacity (Single Breath) mL/min/mm Hg
Predicted value	2.6	4.6	1.9	2.0	63	16
8/13/80	2.7	4.8	2.0	1.6	69	18 (113%)
Bronchodilator	2.8	—	2.5	3.2	—	—
9/19/84	2.9	4.4	2.5	4.0	95	22 (138%)
10/22/87	2.7	3.9	2.4	3.3	97	24 (148%)

New York University Medical Center with normal spirometry, effective voluntary hyperventilation, and lack of response to inspired CO_2 without increase of either ventilation or Po_2. In addition, normal maximal inspiratory pressure of -70 cm H_2O was documented. A sleep study, performed with monitoring of rib cage and abdominal motion as well as ventilation and ear oximetry, showed many episodes of apnea (up to 18/hour), of both central and obstructive types. Apneas lasted as long as 100 sec and were associated with a drop of O_2 saturation to as low as 50%.

The patient remained clinically well at this time and refused further therapy. He appeared phlegmatic but was still employed full time managing his own small business. He was described as a loud snorer, but his wife denied recent changes in his sleep pattern. Excessive daytime somnolence and personality changes were also denied, as was use of tobacco, alcohol, or drugs of any sort.

Physical examination remained normal. Thyroid function studies, nocturnal electrocardiographic monitoring, and a computerized axial tomographic (CAT) scan of the head were all within normal limits. In 1984, spirometry and blood gases remained stable.

On repeat study in October 1986, all ventilatory functions including MBC and inspiratory flow volume loop remained normal, and the patient was still able to lower Pco_2 to 37 mm Hg by voluntary hyperventilation. However, resting Pco_2 in November 1986 was above 80 mm Hg for the first time (Table I). The patient was therefore admitted to the hospital, and an intravenous infusion of aminophylline was given at 37 mg/hr. Within four hours, there was a drop of Pco_2 to 60 mm Hg, and to 56 mm Hg by the next day. However, although oral theophylline therapy was continued for the next ten days, and blood levels as high as 17 μg/mL were achieved, the Pco_2 rose again and remained over 70 mm Hg.

The patient returned home and still had no physical complaints, but over the next few months, systemic hypertension (blood pressure, 170/110 mm Hg) developed, as well as bilateral rales in the lower lung fields, leg edema, asterixis, and slight confusion. He was again referred to New York University Medical Center, where a repeat sleep study showed many episodes of obstructive sleep apnea, approximately one every 60 seconds. There was no central sleep apnea at this time. Use of nasal continuous positive airway pressure (CPAP) of 5 cm H_2O led to total abolition of the apneas; however, the patient found the mask uncomfortable and refused further use of it. CAT scan of the neck was normal.

The patient was readmitted to Beth Israel Hospital in March 1987 because of outpatient blood gas finding of Pco_2 of 85 mm Hg (Table III). Use of nasal CPAP was reattempted without success, and on March 15, an endotracheal tube was inserted and ventilator therapy begun, with prompt development of normocapnia and alkalosis, which was corrected with intravenous acetazolamide. Tracheostomy was performed on March 18. The postoperative course was complicated by bilateral pneumonia. For the next two weeks, attempts to discontinue ventilatory support repeatedly led to gradual development of hypercapnea with Pco_2 over 50 mm Hg. Progesterone treatment was begun on March 25 with no apparent effect. Intravenous aminophylline was given on April 3, 1987, with subsequent drop of Pco_2 to 45 mm Hg without mechanical ventilation. However, in spite of continuation of aminophylline until April 10, with rising theophylline blood levels, subsequent Pco_2 rose over 50 mm Hg, except when the patient was on a ventilator. Therefore, nightly ventilation was continued. Eventually, the patient was discharged to continue the same regimen at home.

DISCUSSION

The patient reported here presented with gastrointestinal bleeding due to peptic ulcer, requiring gastrectomy. There were no signs or symptoms of cardiopulmonary disease, or of sleep disturbance or mental changes. Because of the current practice of obtaining frequent arterial blood gas determinations, hypercapnea was detected. Hypoxemia also was present, presumably due to the complicating pneumonias and/or atelectasis and small airway disease. However, hypercapnea persisted both before and after intubation and tracheostomy, even after clearing of pneumonia and when ventilatory function was entirely normal.

Primary hypoventilation was proven by documenting ability to hyperventilate voluntarily and by absence of response to inhaled CO_2. The patient remained clinically stable for several years with Pco_2 of about 60 mm Hg. He was found to have frequent sleep apneas, both central and obstructive. However, by 1986, the patient's condition began to deteriorate, with increase of Pco_2 to a maximum of 85 mm Hg, concomitant worsening of mental function, and development of hypertension and possible heart failure. A repeat study showed severe and frequent sleep apnea, obstructive at this time.

With deteriorating status and lack of

TABLE III. Subsequent Patient Course

Date	Po_2 (mm Hg)	Pco_2 (mm Hg)	pH	FIo_2	Comments
3/06/87	50	85	7.30	0.2	Outpatient
3/15/87	83	39	7.52	0.4	Intubated on respirator
3/19/87	85	32	7.42	0.3	Acetazolamide given post tracheostomy; respirator
3/19/87	127	57	7.25	0.4	"T-piece".
3/20/87	136	41	7.36	0.3	Respirator
3/26/87	113	51	7.52	0.28	"T-piece"; progesterone
4/02/87	78	54	7.32	0.24	Tracheostomy collar
4/03/87	74	45	7.35	0.24	Tracheostomy collar; intravenous aminophylline
4/04/87	44	55	7.35	0.28	Tracheostomy collar; intravenous aminophylline
4/09/87	136	33	7.51	0.3	Respirator
4/18/87	66	64	7.26	0.24	Tracheostomy collar
5/05/87	55	64	7.32	0.2	Outpatient

improvement with nasal CPAP, a second tracheostomy was performed. Subsequently, hypercapnea still persisted, with P_{CO_2} generally over 60 mm Hg—confirming that the patient's hypoventilation was not secondary to obstructive sleep apnea, but was a true case of primary hypoventilation, or Ondine's curse.

The relationship between Ondine's curse and sleep apnea has not been well defined. Since Ondine's curse presumably is due to a lesion of the central medullary chemoreceptor, central sleep apnea might be expected, and has indeed been reported in two patients.[6] Obstructive sleep apnea, on the other hand, may be caused by a lesion of the carotid bodies, which are receptors that affect stimulation of the genioglossus muscles.[7] Obesity is a well known cause of obstructive sleep apnea (OSA), and Rapoport et al[4] reported obese patients with OSA and chronic hypoventilation. Both obstructive and central sleep apnea were found in 19 patients referred for diaphragmatic pacing for hypoventilation.[8] All but one of these patients were obese and/or had a specific central nervous system lesion which may have contributed to their abnormal breathing patterns. Diaphragmatic pacing increased the tendency to OSA and indeed brought it on de novo in a non-obese individual described by Hyland et al.[9] Finally, a slim patient, very similar to the one described in this report, with idiopathic hypoventilation syndrome, had both obstructive and central sleep apneas unrelated to diaphragmatic pacing.[5]

In the patient presented here, the relationship between the OSA and Ondine's curse is unclear. Of relevance may be the frequent occurrence of OSA in even otherwise healthy elderly males.[10] Over the years of observation, the patient exhibited physical and mental deterioration, with worsening daytime blood gases. Whether the abnormal sleep pattern was the cause or the effect is not known. In any case, a vicious cycle may have developed, whereby the respiratory failure both aggravated and was worsened by the OSA.

This patient was also of interest in regard to his transient response to aminophylline. Aminophylline has been considered a respiratory stimulant for some time. In a recent study,[11] an infusion of the drug increased ventilation in dogs; this effect was attributed to improved diaphragmatic contractility, but a central stimulatory effect was not excluded. Indeed, in a study of cats, theophylline clearly served as a respiratory stimulant by opposing the effect of adenosine, an inhibitor of ventilation.[12]

Use of aminophylline as a respiratory stimulant in patients dates back to 1957, when Galdston et al[13] noted improvement of hypercapnea and of response to CO_2 inhalation in patients with respiratory failure due to emphysema. Similar results were reported by Dowell et al[14]; the latter authors, unlike Galdston, did not rule out improvement in ventilatory function as the cause of the increased ventilation in their patients. But Dowell et al made a further contribution by demonstrating that Cheyne-Stokes respiration could be terminated with aminophylline, which increased minute ventilation and led to cessation of apneic periods.

Aminophylline has also been used in infants with frequent apneas, who were considered at risk for sudden infant death syndrome (SIDS). Normalization of their breathing patterns has been noted.[15,16]

Finally, some recent studies have indicated that theophylline is effective in preventing central, but not obstructive, sleep apneas. Reports have differed as to whether the effect lasts with long term oral therapy.[17,18] Available studies of aminophylline as a bronchodilator indicate no loss of efficacy over time.[19]

In the case of the patient reported here, it is clear that he had ventilatory stimulation from aminophylline on two occasions, each time lasting only a few days. The reason for this loss of therapeutic response is not clear. Further therapeutic trials of theophylline in patients with Ondine's curse would clearly be indicated.

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See also pp 196–198.

Peptic ulcer disease presenting as life-threatening diarrhea following subtotal colectomy

JOHN B. RODGERS, MD

Diarrhea can be life threatening if large quantities of fluid are lost. Patients with severe diarrhea develop hypovolemia, which can lead to renal failure and eventual vascular collapse. While multiple factors were involved in the case report presented here, aggressive treatment of gastric acid hypersecretion proved successful.

CASE REPORT

A 76-year-old man was admitted to a hospital other than ours for treatment of multiple benign and malignant tumors of the colon. His history was significant for chronic alcohol use, chronic obstructive pulmonary disease, and diabetes mellitus type II, which was being treated with diet. The patient had no history of prior gastrointestinal disease, although he had experienced heartburn for a number of years. The family history was unremarkable for gastrointestinal or endocrine disorders.

Physical findings and routine blood studies were also unremarkable. The serum creatinine level was 1.1 mg/dL. The patient underwent a subtotal colectomy and an ileosigmoid colostomy with placement of a gastrostomy tube. Pathologic diagnosis of tumors in the resected colon included two tubular adenomatous polyps; two villotubular adenomatous polyps, both containing foci of intramucosal adenocarcinoma, and adenocarcinoma of the transverse colon, Duke's stage A. The regional lymph nodes showed no metastases.

The next ten weeks were complicated by severe diarrhea with daily fecal output of 4 to 5 liters. Additionally, the patient complained of nausea and heartburn. Endoscopy showed small ulcers of the duodenum confirmed by biopsy. Sigmoidoscopy demonstrated 15 cm of normal rectosigmoid colon and a patent anastomosis. Stool culture and tests for *Clostridium difficile* toxin, as well as ova and parasites, were negative. Radiologic studies of the upper and lower gastrointestinal tract were normal. Assays of various hormone levels, which yielded normal results, included calcitonin, gastrin, glucagon, and vasoactive intestinal peptide. Urinary 5-hydroxyindoleacetic acid level was within normal limits.

Medical treatment was unsuccessful, and the patient's abdomen was subsequently reexplored for possible bowel obstruction. Many adhesions were lysed, and an ileostomy was performed. The patient did poorly after surgery, with the diarrhea continuing despite treatment. This included central intravenous alimentation; diphenoxylate with atropine, 2 tablets four times a day; pancreatic extract, 3 tablets with feedings; amitriptyline, 50 mg at bedtime; ranitidine, 150 mg twice a day; metronidazole, 250 mg four times a day; and diazepam, 5 mg three times a day. Renal failure developed, with the serum creatinine rising to 5.9 mg/dL. The patient was subsequently transferred to the Albany Medical Center Hospital.

At this time, the patient was complaining of nausea, anorexia, weakness, and diarrhea. Routine laboratory results were unremarkable except for a serum creatinine level of 7.8 mg/dL.

Central intravenous alimentation was continued. Oral feedings were withheld to determine daily basal ileostomy outputs. These varied from 1.2 to 1.5 liters. Continuous gastric aspiration via the gastrostomy tube was next performed to see the effect on ileostomy output. Daily volumes of gastric aspirate measured 1 to 1.5 liters, with hydrochloric acid concentration of 118 mEq/L, giving a secretory rate of 7 mEq/h in the basal state. During this time, the daily ileostomy output dropped to 200 mL. Additional serum gastrin level tests and a secretin stimulation test were performed to exclude a gastrinoma as a cause of the diarrhea. These tests were normal.

The patient was then given a high dose of famotidine, 40 mg twice a day, as treament for gastric hypersecretion. (This high dose was selected because the patient had previously been given ranitidine at the usual recommended dose without benefit.) Overnight collections of gastric acid dropped to 100 mL, with a hydrochloric acid concentration of 46 mEq/L.

Subsequently, oral feedings were restarted and intravenous feedings were discontinued. Loperamide, 2 mg three times a day, was also given. Daily ileostomy outputs fluctuated from 0.5 to 1.5 liters. The ileostomy was subsequently closed. The patient continued to have mild diarrhea with daily fecal outputs below 1 liter. Just prior to discharge, a basal acid output with famotidine held for 24 hours was 6.9 mEq/L. The patient was discharged on loperamide, 2 mg three times a day, and famotidine, 40 mg twice a day, which was later decreased to 40 mg at bedtime. When seen three months later, the patient was doing well, with a serum creatinine of 1.3 mg/dL. He has continued to do well during a year of follow-up.

DISCUSSION

In this patient, severe diarrhea developed following a subtotal colectomy. Gastrointestinal fluid losses in excess of several liters daily over a period of weeks to months impose severe stress on the body by producing chronic dehydration and malnutrition. If the complications of dehydration and malnutrition are not corrected, gastrointestinal function is likely to deteriorate further, thus increasing the problem of excessive fecal fluid loss. Acute and chronic renal failure is a common consequence, thus making this type of chronic diarrhea a life-threatening situation.

It is fortunate for such patients that total parenteral alimentation (TPN) has been developed to treat these complications of dehydration and malnutrition. In some patients with severe postoperative diarrhea, TPN given alone for a period of several weeks can produce resolution of the diarrhea as a result of improvement of the patient's nutritional status. This was not observed in this patient. Indeed, his requirements for parenteral fluid replacement were initially underestimated, causing the development of moderate renal failure.

When presented with a case of severe diarrhea requiring parenteral support, it is helpful to determine whether the diarrhea is the result of osmotically active exogenous agents in the lumen of

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the intestine, such as magnesium salts (osmotic diarrhea), or is "secretory" in nature. The distinction is not always clear, but when the diarrhea continues after a patient fasts for 24 hours (as noted in this patient), the diarrhea should be considered "secretory."

Secretory diarrhea has a number of causes including infection and inflammatory bowel disease. Infectious agents known to produce an enterotoxin that stimulates fluid secretion by the enterocytes are well described and include *Vibrio cholerae*, *Clostridium difficile*, and *Escherichia coli*, among others.[1] Infectious causes of diarrhea were investigated in this case but were not found, and contrast radiologic studies of the intestinal tract failed to demonstrate any abnormalities that would suggest a diagnosis of inflammatory bowel disease.

Another cause of secretory diarrhea is overproduction of a gastrointestinal hormone by a tumor. One such hormone that has been well described and characterized is gastrin, which is produced in excessive amounts by a gastrinoma. The stomach is the major target organ for this hormone, resulting in excessive gastric acid production.[2] This gastric hypersecretion can produce diarrhea, as noted in more than 30% of patients with gastrinoma.[2] It is postulated that in addition to the large volume of gastric juice being presented to the intestine for absorption, the diarrhea also results from a reduction of pH of the upper small bowel lumen. A pH much lower than 6 will inactivate pancreatic enzymes, interfering with digestion and absorption of dietary components, especially fats. As certain unabsorbed long chain fatty acids can inhibit water absorption by the intestine,[3] this malabsorption of fats can contribute to the problem of diarrhea.

Tumors that autonomously secrete other gastrointestinal hormones also cause diarrhea, with these other hormones having their major effect directly on the enterocytes, causing them to secrete excessive amounts of fluid into the lumen of the intestine. Hormones reported to have this effect include vasoactive intestinal peptide (VIP), calcitonin, gastric inhibitory peptide, serotonin, and prostaglandin E_1.[1] Studies of VIP under controlled conditions document the production of watery diarrhea when given to healthy subjects.[4] Patients who suffer secretory diarrhea from these hormones have been referred to as having the "WDHA" syndrome. This term indicates that most of these patients present with a combination of findings including watery diarrhea, hypokalemia, and achlorhydria.[5]

Some patients with secretory diarrhea from exposure to abnormal hormonal levels have elevated gastric acid, as seen in those with gastrinomas, while others have low levels of gastric acid, as is often the case in patients with WDHA. Therefore, gastric acid analysis can be useful in the evaluation of these patients. In the patient described here, it was soon apparent that the problem was overproduction of gastric acid, and that the diarrhea could be readily terminated by continuous gastric aspiration. These findings suggested the possibility of a gastrinoma, but subsequent studies failed to provide any indication of this diagnosis. The patient's clinical course over a 12-month period of follow-up has been uncomplicated on a regular therapeutic dose of famotidine, further excluding gastrinoma as the cause of diarrhea.

The data obtained on this patient indicate that the basic cause of the severe and chronic diarrhea following his subtotal colectomy was peptic ulcer disease. Some patients with duodenal ulcer disease have a large parietal cell mass[6] that causes hypersecretion of gastric acid in both the basal and stimulated state. In this patient, the condition likely caused large volumes of fluid to be secreted into the upper intestinal tract. As long as the colon was in place and functioning, however, the overproduction of gastric acid caused no overt clinical problem. The colon has a capacity to absorb three to four liters of isotonic saline per day.[7] It was not until this reserve capacity of the colon for water and electrolyte reabsorption was lost that the peptic ulcer disease in this patient caused severe, incapacitating diarrhea. Once it was recognized that the diarrhea was the result of overproduction of gastric acid, the delivery of adequate treatment was not difficult.

Gastric acid analysis is a simple, inexpensive, noninvasive procedure. In patients with incapacitating secretory diarrhea, this test should be performed to determine whether excessive production of gastric acid is the cause. In patients with peptic ulcer disease, treatment should be readily effective. If the cause is a gastrinoma secreting excessive quantities of gastrin, treatment will be more difficult, but high doses of various histamine-2 receptor blockers can often be of benefit.

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Endodermal sinus tumor and benign cystic teratoma of the ovary occurring simultaneously

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Endodermal sinus tumors (EST) of the ovary are rare and highly malignant germ cell tumors, primarily occurring in children and young adults. These germ cell tumors replicate the yolk sac and its derivatives; their presence is evident both microscopically and in the excessive production of alpha-fetoprotein (AFP). We present the case of a woman with a yolk sac tumor who also had a mature cystic teratoma in the contralateral ovary. The endodermal sinus tumor was in a clinically advanced stage with extension to the pelvis and metastases to the liver, omentum, and retroperitoneal lymph nodes.

CASE REPORT

A 24-year-old woman presented to the Emergency Room of St Vincent's Medical Center of Richmond with fever, malaise, and lower abdominal pain. Four months before, she had been hospitalized elsewhere for treatment of a tubo-ovarian abscess. She was then followed by a private physician in New York City and treated with danazol for supposed endometriosis. The surgical history was unremarkable.

On physical examination, the patient appeared pale and weak. Her blood pressure was 130/80 mm Hg; pulse, 90/min; temperature, 100.5°F; and respiratory rate, 22/min. The general physical examination was unremarkable except for the abdomen, which appeared tense, with ascites. A firm mass was palpable in the lower abdomen. Bowel sounds were present. The pelvic examination revealed a solid, cystic mass occupying the entire pelvis and extending to the umbilicus.

A complete blood count showed severe anemia and an elevated white blood cell count. On sonogram, a large pelvic solid, cystic mass was seen. A computed tomographic (CT) scan of the abdomen/pelvis showed a large pelvic mass with solid cystic components and multiple liver lesions.

Following intravenous antibiotic therapy, hydration, and blood replacement, the patient underwent exploratory laparotomy for acute abdominal pain. On entering the abdomen, 2,500 cc of hemorrhagic fluid was noted. After evacuation, a solid necrotic, 14.0 × 8.0-cm, tumor was evident in the right ovary. The left adnexa demonstrated a 7.0-cm smooth, encapsulated cystic tumor arising from the left ovary. Exploration of the abdomen and retroperitoneum demonstrated carcinomatosis. A total abdominal hysterectomy with bilateral salpingo-oophorectomy, total omentectomy, and tumor debulking was performed. Chemotherapy was planned at the New England Deaconess Medical Center.

Forty hematoxylin-eosin-stained slides of both tumors were examined. Two sections of the endodermal sinus tumor were stained with periodic acid-Schiff with and without diastase. Indirect immunohistochemical reaction for alpha-fetoprotein was performed on two deparaffinized sections of formalin-fixed tissue with the use of peroxidase antiperoxidase method.

The left ovary revealed an oval-shaped, lobulated mass measuring 14.0 × 8.0 × 5.0 cm. On cross section, it was solid, gray-white, with hemorrhagic and necrotic areas. The surface was mucoid (Fig 1). The right ovary revealed a cystic, oval-shaped mass with a smooth gray surface measuring 7.0 × 4.0 × 2.0 cm. On cross section, it was a unilocular cyst filled with hair and sebum (Fig 2).

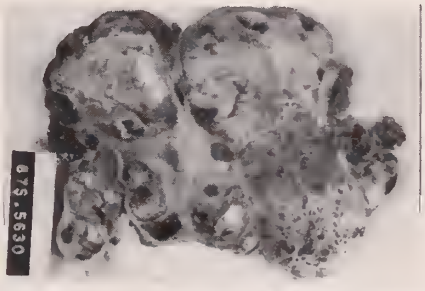

FIGURE 1. A cross-section of the endodermal sinus tumor. Note the lobulation, hemorrhage, and necrosis.

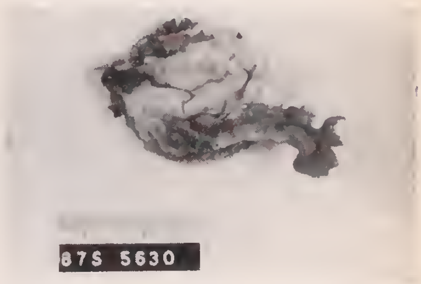

FIGURE 2. Benign cystic teratoma; cyst was evacuated. Note the hair and sebum.

The left ovarian tumor exhibited a papillary and glandular pattern (Fig 3). The stain for alpha-fetoprotein was positive. Hyaline globules which were periodic acid-Schiff positive were found. The right ovary contained mature squamous epithelium with skin adnexa, cartilage, and respiratory and colonic-type epithelium.

DISCUSSION

Endodermal sinus tumors (EST) are rapidly growing and usually of considerable size when encountered. They locally invade and metastasize early via the lymphatics to regional lymph nodes and also by the hematogenous route to

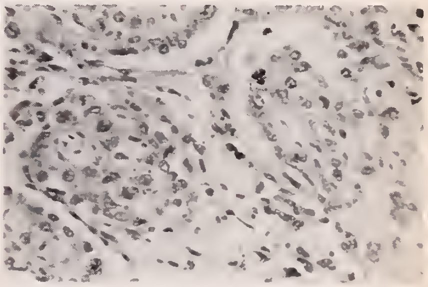

FIGURE 3. Microscopic picture of endodermal sinus tumor (original magnification ×100; hematoxylin-eosin stain). The tumor has a papillary arrangement with a vascular core. Notice one mitotic figure.

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the lungs, liver, and other organs. Metastases, as in the patient described here, are frequently present at the time of initial observation.[2]

In a series of 71 women with EST reported by Kurman et al[3,4] in 1976, ten patients had a mature (benign) cystic teratoma associated with EST in the same ovary, and in only two of these women the opposite ovary contained a benign cystic teratoma.

EST is radioresistant. Recently, the administration of cis-platinum, bleomycin, and vinblastine has completely revolutionized the treatment, with a dramatic increase in survival.[4]

The use of serial determination of alpha-fetoprotein may permit earlier detection of recurrent disease, and therefore, direct treatment of subclinical recurrent disease at an earlier stage when chemotherapeutic drugs are most effective.[5]

The benign cystic teratoma in the contralateral ovary was also a germ cell tumor, but in this case it was mature (benign).

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Pulmonary hypertrophic osteoarthropathy as the initial manifestation of interstitial fibrosis

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Pulmonary hypertrophic osteoarthropathy is a syndrome characterized by clubbing of the digits, arthralgia, and periostitis of the distal long bones.[1-3] Most cases of pulmonary hypertrophic osteoarthropathy are associated with neoplastic pulmonary diseases, especially bronchogenic carcinoma. We report a case of pulmonary hypertrophic osteoarthropathy associated with idiopathic pulmonary fibrosis in which the initial presenting complaint was arthralgia.

CASE REPORT

A 35-year-old man presented in April 1985 with complaints of bilateral knee and ankle pain and stiffness. The symptoms had first developed three years previously but had progressively worsened over time. For approximately three to four months prior to his presentation, the patient also noticed increasing dyspnea on exertion and a dry, nonproductive cough. He had a 15-year history of smoking one pack a day but denied any occupational exposure to asbestos. The patient had previously worked as a diamond polisher, but for the three years prior to his presentation had been working as a manager in a knitting factory. He denied any rhinitis, anorexia, weight loss, or any prior history of pain or tenderness in the digits, but stated that clubbing was present for 15 years. There was no family history of arthralgias, arthritis, or skin abnormalities. Physical examination revealed 4+ clubbing of his digits, with a normal appearing face. The lung examination was remarkable for bibasilar rales. The heart and abdomen were within normal limits. There was bilateral swelling of the ankles and knees but no tenderness. The distal tibiae were bilaterally enlarged without any pitting edema. There was poor range of movements in all joints, and there were no signs of arthritis.

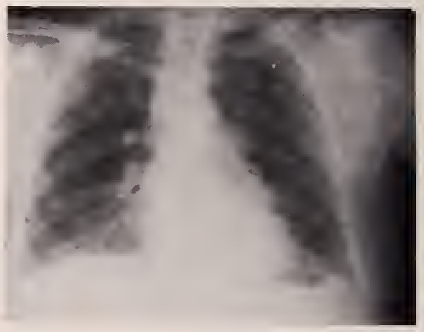

FIGURE 1. Chest film demonstrating a diffuse reticulonodular pattern.

A chest film revealed a diffuse reticulonodular pattern (Fig 1). Radiographs of the extremities revealed periostitis of the femur, tibia, and fibula consistent with hypertrophic osteoarthropathy (Fig 2). Pulmonary function tests showed a vital capacity that was 40% of predicted, total lung capacity 58% of predicted, and a diffusion capacity 37% of normal. Routine laboratory studies were within normal limits except for a slightly elevated lactose dehydrogenase at 285 mU/mL (normal, 100–225 mU/mL) and alkaline phosphatase of 480 mU/mL (normal, 30–115 mU/mL). Arterial blood gas levels on room air revealed a pH of 7.43, arterial carbon dioxide tension ($PaCO_2$) of 31 torr, and arterial oxygen pressure (PaO_2) of 63 torr. IgG, IgA, and IgM levels were within normal limits. A test for rheumatoid factor was negative. A Kveim test was negative. The patient underwent bronchoscopy with bronchoalveolar lavage. No endobronchial lesions were found, and the mucosa was normal. Bronchoalveolar lavage revealed 95% macrophages, 3% lymphocytes, and 2% polymorphonuclear leukocytes consistent with a low level macrophage alveolitis. Cobalt was not detected. An open lung biopsy was performed and revealed idiopathic pulmonary fibrosis. The patient was started on prednisone, 20 mg/day, with an initial improvement in both dyspnea and joint pain. After several weeks of therapy,

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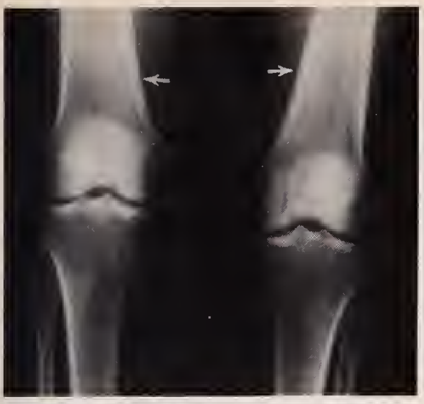

FIGURE 2. Radiograph of knees showing subperiosteal bone formation.

the patient experienced an exacerbation of his symptoms, which has persisted despite being placed on higher dosages of prednisone.

DISCUSSION

Pulmonary hypertrophic osteoarthropathy is a syndrome of unknown etiology consisting of periostitis of the long bones, digital clubbing, and polysynovitis.[1-3] Periostitis causes severe burning pain in the distal extremities, which are very tender to palpation. A unique feature of hypertrophic osteoarthropathy is the aggravation of bone pain on dependency. Clubbing generally tends to be asymptomatic, although patients may complain of burning and warmth of the finger tips. Synovitis is usually symmetrical and may affect the knees, ankles, wrists, and metacarpophalangeal joints. Autonomic dysfunction including flushing, blanching, and severe sweating may occur. Radiographic features include symmetrical subperiosteal new bone layering in the distal long bones, especially in the legs and forearms. Scintillography with 99m technitium pyrophosphate or diphosphynate demonstrates pericortical linear uptake of the tracer along the long bones.

Hypertrophic osteoarthropathy usually appears secondary to other systemic diseases, although a primary form exists. It is commonly associated with neoplastic and suppurative diseases of the lungs, mediastinum, and pleura.[4] Pulmonary infection, including bronchiectasis, and tuberculosis were commonly associated with hypertrophic osteoarthropathy.[5] With the advent of antibiotics, however, hypertrophic osteoarthropathy is most commonly associated with pulmonary neoplasms, especially bronchogenic carcinoma. Hypertrophic osteoarthropathy may be the initial presenting feature of a pulmonary neoplasm. Segal and Mackenzie[6] reported that hypertrophic osteoarthropathy, on average, preceded the discovery of pulmonary neoplasm by 4.9 months. Although this patient is a smoker and consequently at higher risk for the development of pulmonary neoplasm, there has been no evidence of a neoplasm during the more than two years that we have followed the patient.

The association between interstitial fibrosis and pulmonary hypertrophic osteoarthropathy is unusual, although the association between interstitial fibrosis and clubbing is well established. In recent reviews of interstitial fibrosis, clubbing was found in 50–61% of patients.[7-11] Pulmonary hypertrophic osteoarthropathy was reported, however, in only four patients.[12] A literature review has yielded only six cases of pulmonary hypertrophic osteoarthropathy associated with pulmonary fibrosis. We wish to report that arthralgias and other symptoms of pulmonary hypertrophic osteoarthropathy may, in fact, be the initial complaint of pulmonary interstitial fibrosis.

Acknowledgment. The authors thank Barbara Gudz for technical assistance in this work.

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Ben W. Aberman, MD

TO THE EDITOR: I read with sadness in the October 1988 issue the obituary of Dr Ben W. Aberman of East Hampton. He was my family doctor in the Washington Heights section of New York City 40 years ago. In 1947 when I was a summer camper in Connecticut, I developed gastroenteritis, later found to be nonparalytic poliomyelitis. Aberman drove immediately to the camp with my parents, correctly diagnosed the condition, and arranged hospitalization. He functioned as an emergency physician, an ambulance service, a crisis counselor, a family therapist, and a friend.

In this era of commercialized medicine dominated by big insurance companies and managed care systems with their acronyms, buzzwords, and logos, and their armies of claims clerks, auditors, reviewers, and functionaries, it is nice to reflect back to a simpler time when medical care was personalized and one decent fellow like Dr Aberman could make a difference. He'll be missed.

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Psychiatric teams and the psychiatrist's authority

TO THE EDITOR: Dr Irving Jabitsky's article,[1] "Psychiatric teams and the psychiatrist's authority in the New York State mental health system," has performed a useful service in discussing the role and authority of the psychiatrist in the treatment team. Just as in George Orwell's *Animal Farm*—"All animals are equal, but some are more equal than others," so Dr Jabitsky reminds us—"All members of the team are unique but not equal." The rationale underlying the team concept is that each member will bring his or her unique expertise to bear for the ultimate benefit of the patient.[2] Dr Jabitsky rightly observes that to do this successfully requires skillful management and leadership of the team. Clearly, the team member best qualified to succeed in this task, both by training and by experience, is the psychiatrist. He or she should have the most comprehensive training and experience not only in mental disorders, but also in general medicine, neurology, psychopharmacology, psychopathology, and, most currently, brain imaging. Additionally, because the psychiatrist should be most knowledgeable in group dynamics and in management, he or she should be best placed to deal with conflicts, rivalries, and antagonisms that interfere with the smooth functioning of the team. Yet there is nothing to prevent the psychiatrist as team leader from designating another member as temporary leader, if it is felt that the patient's problem warrants such a change. In geropsychiatry, for example, it is common for team leadership to shift between geropsychiatrist and geriatrician as the patient's needs dictate.

I think that within the New York State Office of Mental Health (OMH), if a team member successfully challenges the authority of the psychiatrist, who does have the final legal responsibility for the patient's care, that would represent a failure of proper team education and functioning. As mentioned elsewhere in the article, only one captain can fly the airplane or navigate the ship. This requires that part of the social contract within the team is to accept the leadership of the psychiatrist. Dr Jabitsky refers to the ambivalence always present between the leader and the led: "those who are subject to another's power both welcome and resent it." This problem, again, requires skilled and sensitive management. But it is important to remember that in any leaderless group, sooner or later a leader must emerge through group consensus and submission. If this leader is not to be the psychiatrist, who will accept that role? Not the nurse, who lacks advanced training in the subjects already mentioned; not the clinical psychologist, whose repertoire is largely devoid of the areas of general medicine and neurology; and not the social worker, whose training, while emphasizing the extrinsic socio-cultural and environmental factors in mental disorders, cannot address intrinsic genetic and biological factors. With the increasing age of the population and consequently the increasing importance of brain pathology and systemic disease in the genesis of mental disorders, today more than ever psychiatrists need a strong background in medicine and neurology. For in geropsychiatry we learn every day anew that iron fact—the brain is the organ of mind.

Public hospital psychiatrists with strong professional identities understand that they belong to a medical specialty and should therefore feel secure in their clinical leadership capability. If they observe competition between the team and themselves they must ask themselves the reason. Is it because, in some way, they have failed to earn the team's respect and thus acceptance of their broader and superior knowledge? Unfortunately, some of us may fail in that regard. It is worth recalling two examples from the past where failure to be guided by correct neuropsychiatric insights resulted in adverse consequences for patients. Thus, "patterning" therapy was prescribed for patients suffering from spasticity resulting from damage to the upper motor neurone. And patients with fluent aphasia (Wernicke aphasia) were incorrectly confined to mental hospitals because of their disorganized speech (jargon), for which they had an agnosia.

Dr Jabitsky cites figures showing the replacement of psychiatrists by nonpsychiatrists in public mental health programs in the decade from 1971 to 1981. This trend deserves further study to see if it is continuing, and to establish more precisely the causes. Is it to reduce expenditures? Is it because program directors believe that the psychiatrist's expertise is not necessary to run mental health programs, and that nonpsychiatrists can do an equally effective job? Is it because too many psychiatrists in public mental health programs flee from frustration, or to seek greener pastures?

Dr Jabitsky also describes a disturbing example of a (nonpsychiatrist) team leader overriding a psychiatrist's decision to talk to an agitated patient who "was screaming in the hallway," before resorting to PRN medication. In the OMH system, this would not only violate the principle of "the least restrictive environment," but, it seems to me, might also expose the agency to possible litigation in the event of adverse consequences from the failure to follow a psychiatric decision. I must therefore disagree with Dr Jabitsky's statement, "Unfortunately, senior administrative psychiatrists, who are also in management, tend to support nonmedical chiefs of service and team leaders and not on-ward clinical psychiatrists, who are labor." As an administrative psychiatrist (clinical director), I would always support a colleague where I agreed that his decision was correct and was made in the best interests of the patient. Clearly, if necessary, one would need to educate other nonpsychiatrist managers to accept such decisions.

Dr Jabitsky also cites some conflicting statements about foreign medical graduates (FMGs). On the one hand, he cites a study, "FMGs are culturally and linguistically alien to the patients they serve," and on the other hand writes, "Neither should the contributions FMGs make to the mental health system be undervalued." Many FMGs are American-born, so the first statement does not apply to them. And those FMGs coming from other countries become continuously acculturated like other immigrants.

Every psychiatrist working in the public sector will agree with Dr Jabitsky's call for the restoration of the prestige and clinical authority that properly belongs to the psychiatrist. However, this ideal requires that each of us in public mental health systems work tirelessly at educating our nonpsychiatric colleagues and team members about the true role of the psychiatrist. This is far more than the perception "that he simply signs prescriptions and medical records."

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Poor legal representation in a malpractice lawsuit

TO THE EDITOR: The letter by Dr Ralph Ger[1] in the November 1988 issue touched a nerve. I feel that I, too, had poor legal representation as a defendant in a recent malpractice lawsuit. Here are some of the difficulties I had.

Another physician "set up" this lawsuit, while being a defendant himself in several unrelated cases handled by a particular law firm. My malpractice insurance company, aware of the conflict of interest, nevertheless assigned a lawyer from that firm to defend me. It was left to me to request substitution of counsel.

The new lawyer told me that he had never before defended a case in my specialty, but that he thought he could handle it. Having already changed lawyers once, I was afraid of being considered uncooperative if I requested another change. I spent much time and effort in familiarizing this attorney with basic knowledge before we could discuss my case.

Despite my cooperation, my lawyer did not return the favor. He did not provide me with copies of any information about my case, not even the plaintiff's examination before trial, until I requested them in writing. He did not bother to call me when the trial date was postponed.

This advocate appointed by the insurance company was disorganized and distracted. He missed a meeting scheduled at my office, for which I had cancelled half a day's patients. At meetings he did attend, he regularly jumped up to phone his office or other clients in the midst of our discussions.

My case was not thoroughly prepared. Charts of two subsequent treating physicians were never requested; the third such chart reached me one week before the trial, only after repeated demands. Also, for reasons that the insurance company refuses to disclose, I was allowed only one witness, the expert. He was young, relaxed, and grinning, but broke down under cross-examination; whereupon my lawyer unashamedly explained that he had "forgotten" to review the most critical point in the case with him. This occurred in the face of the appearance of three well-rehearsed witnesses for the plaintiff.

My attorney's effort during the trial was entirely inadequate. In response to the sneering, shouting performance put on by the plaintiff's two lawyers, my attorney became increasingly apologetic and confused. I had to encourage and guide his thinking, and help him compose his closing statement, but unfortunately he substituted a meek and regretful summation. Losing the case was inevitable.

It is no consolation to recall that my lawyer had said that I was "the most cooperative client" he had ever had, and to learn that my insurance company's medical experts are certain that I did not commit malpractice. On the contrary, this strengthens my belief that my legal defense left me defenseless. Like Dr Ger, I would be interested to know how widespread this situation is, and what can be done in a concerted way about cases such as ours. Perhaps the Medical Society of the State of New York could help.

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Doctors' penmanship

TO THE EDITOR: I read Roger Miles Rose's[1] note about doctors' penmanship. At the bottom of my personal note paper, I use a statement which anyone may quote freely, "There is no correlation between handwriting and surgical skill."

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Documentation of maternal mortality

Maternal mortality is often preventable, yet the maternal mortality rate in New York State, 16.7 per 100,000 live births, is approximately twice the national average and far exceeds the national 1990 goal of five deaths per 100,000 live births set by the Surgeon General.

Coding accuracy may substantially affect reported rates. Traditionally, maternal deaths are identified through the examination of the underlying cause of death reported on the death certificate. Deaths are selected that are coded to pregnancy complications, childbirth and the puerperium according to the International Classification of Diseases, Ninth Revision (ICD-9) Codes. Vital statistics data indicated that during 1980–1986, a total of 289 maternal deaths occurred among New York State residents. The limitations of vital statistics data in measuring maternal mortality have been documented.[1] Essentially, death certificates often do not contain adequate information to identify maternal deaths. A current or recent pregnancy may go unreported on the certificate, or the relationship of other disease conditions to a known pregnancy is not clearly stated.

In order to assess the completeness of maternal death reporting and enhance data quality, a vital records match was performed to identify potential maternal deaths. Deaths to women age 10–49 during 1980–1986 were matched against live birth, fetal death and induced abortion files to determine if the deceased had terminated a pregnancy within 90 days of her death. This span of 90 days conforms with the definition of maternal mortality used by the American College of Obstetricians and Gynecologists.[2] This computerized matching resulted in the identification of an additional 193 deaths that occurred within 90 days of a pregnancy termination. Of these deaths, 135 occurred in hospitals and had available records that were reviewed by a team of physicians. Seventy-three were subsequently identified as maternal deaths after physician review. The state maternal mortality rate with the additional computer matched deaths was 20.9 per 100,000 live births, up from 16.7.

Regional Differences

New York City accounted for almost half the births over the study period. Approximately 80% of the maternal deaths identified from death certificates occurred in New York City, resulting in a maternal mortality rate of 29.2 per 100,000. The rate for the rest of the state was 6.7 per 100,000. There appears to be a much larger undercount in upstate rates as compared to New York City when the additional maternal deaths from record matching are considered (as shown in Table I). Fifty of the 73 matched record deaths occurred in upstate New York, resulting in a revised maternal mortality rate of 11.9 per 100,000. This rate is almost double that based on the death certificate alone. The rate for New York City increased to 31.9 per 100,000 with the addition of the remaining 23 record matched deaths.

Several reasons are proposed for the difference in the additional maternal deaths determined by record matching in New York City (10%) as compared to upstate (78%). The death certificate in New York City questions whether the deceased had been pregnant within the last three months. This information may affect the cause of death coding as well as the type of investigation into the cause of death. Second, there is an established independent reporting system in place from hospitals to the New York City Department of Health in the event of maternal deaths. The higher level of ascertainment of maternal deaths on the death certificate in New York City reduces the number of women for whom detection would potentially have been made through record matching. Also, New York City birth certificates do not contain a social security number which reduces the probability of a successful match with the death file. The form used to report abortions in New York City also makes it difficult to match death and abortion files because it contains limited identifying information. This limits the matching of deaths to upstate New York recorded abortions, or approximately 40% of the abortions occurring among New York State residents.

Maternal Race

The ratio of black-to-white maternal mortality is four to one in upstate New York and three to one in New York City. In New York City, the addition of the record matched deaths had little effect on the white maternal death rate. However, the black rate increased approximately 10% and the Hispanic rate approximately 23% with the inclusion of the direct and indirect obstetrical deaths (Table II).

TABLE I. Maternal Mortality Rates per 100,000 Live Births in New York State, 1980–1986

		Death Certificate Maternal Deaths		Total Maternal Deaths*		
Recorded Area	Births	Number	Rate	Number	Rate	Percent Increase
Upstate New York	954,550	64	6.7	114	11.9	78
New York City	777,266	225	29.2	248	31.9	10
Total state	1,731,816	289	16.7	362	20.9	25

* Includes death certificate and record matched deaths classified as either direct or indirect obstetric deaths.

TABLE II. Maternal Mortality Rates by Maternal Race, 1980–1986

	White		Black		Hispanic	
	Number	Rate	Number	Rate	Number	Rate
Death Certificate Only						
Upstate New York	42	5.1	18	21.5	4	12.8
New York City	40	16.1	119	47.9	53	23.6
Total	82	7.7	137	41.3	57	22.3
Death Certificate Plus Record Matching						
Upstate New York	75	9.1	31	37.1	7	22.4
New York City	43	16.3	130	52.4	61	27.2
Total	118	10.9	161	48.5	68	26.6

The ratio of black-to-white maternal mortality rates decreased from approximately five to one in 1980–1982 to the current level of four to one during 1984–1986. Three-year moving average rates were used in place of yearly rates to even out year-to-year fluctuations caused by the small number of maternal deaths. A comparison of upstate to New York City race specific rates showed that black New York City mortality rates were 1.5 times higher than those in upstate at the beginning of the study period (1980–1982). Since then, black maternal mortality in New York City has declined to the point where the city and upstate rates are approximately equal when birth certificate and record matching deaths are considered. White rates in New York City continue to be higher than those in upstate, although this differential has also decreased over time.

Cause of Death

The 289 maternal deaths based on death certificates in the state were distributed over 22 ICD-9 codes. The major causes of death were ectopic pregnancy and obstetrical pulmonary embolism, each with 46 deaths during 1980–1986. Hypertension was the second leading cause with a total of 38. The majority of the 73 deaths classified as either direct or indirect obstetric deaths determined by record matching were related to diseases of the circulatory system, including subarachnoid hemorrhaging and pulmonary embolism. Six of the maternal deaths were external relating either to surgery complications or suicides due to postpartum depression.

As a result of this study, the Department of Health has implemented a program for maternal death ascertainment and review beyond the traditional approach of death certificate review. Computer matching of all female deaths in the childbearing ages with live birth, fetal death and abortion files has been implemented as a routine process in vital statistics. In addition, the death certificate has been modified to include a question regarding whether the deceased female of childbearing age has been pregnant within the past six months. The department has also instituted a maternal mortality surveillance program in which the medical charts of all in-hospital identified deaths will be reviewed to determine if an obstetric death occurred and to classify that death as a direct or indirect obstetric death. For deaths occurring out of hospitals, a query program will be instituted to follow back to local offices of coroners and medical examiners for information regarding the death in order to determine if it was pregnancy related.

Material for this article was obtained from Maternal Mortality in New York State, a report prepared by the Information Systems and Health Statistics Group. A copy of the report is available from: Bureau of Information Science and Policy, New York State Department of Health, Empire State Plaza, Corning Tower, Room 308 Albany, NY 12237.

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Changing trends in malignant melanoma in New York State

The incidence of malignant melanoma, a type of skin cancer derived from melanin forming cells, has been rapidly increasing in New York State (excluding New York City) since 1950 (Fig 1). Between 1950 and 1985, the age-adjusted incidence rate has increased five-fold in males and more than tripled in females.[1] There are approximately 1,280 new cases of malignant melanoma reported each year, with 435 deaths attributed annually to this disease. If the cancer is diagnosed in its early stages, 89% of patients survive five years. However, if the diagnosis is made after the melanoma has spread, the proportion of patients reaching the five-year survival mark drops to 46%. Approximately 20% of diagnoses are made in the later stages of disease, reducing the overall five-year survival rate to 80%.[2]

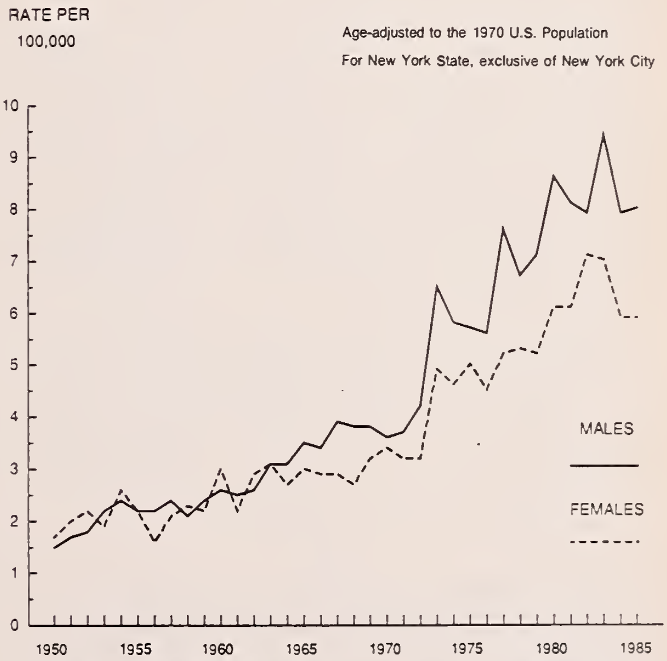

FIGURE 1. Age-adjusted malignant melanoma incidence rates by sex, New York State, 1950–1985.

Melanoma is generally more serious than nonmelanoma skin cancers, such as basal or squamous cell cancers, primarily due to the more aggressive treatment involved. In addition to the cost of treatment, the impact on the individual includes greater risk of premature death, a greater extent of physical disfigurement, and psychological and social stresses associated with more aggressive treatment. The impact of malignant melanoma on the medical care system can be measured by examining hospital utilization. Between 1980 and 1985, malignant melanoma accounted for an annual average of 1,661 hospital discharges, and 15,278 inpatient hospital days.

Etiologic Factors

Solar ultraviolet radiation is the most important exposure related to malignant melanoma. Other risk factors relate to the effects of solar ultraviolet radiation. For example, individuals with no tanning ability are shown to have a three- to four-fold increased risk of developing malignant melanoma. Whites have a much greater risk (>10-fold) of developing this cancer as compared to blacks due to less pigmentation. The risk of developing melanoma increases with proximity to the equator due to the increasing directness of ultraviolet rays. The risk is also higher for individuals with light complexions, fair hair, light eyes or others who burn easily. Malignant melanoma is also thought to have a genetic component since it is found more frequently among individuals with xeroderma pigmentosum and dysplastic nevus syndrome, two genetically determined diseases. In addition, individuals with a family history of malignant melanoma have between a two- to three-fold risk of developing the disease. Other risk factors that have been suggested include occupational exposure to ionizing radiation, use of oral contraceptives, menopausal estrogen use, immunosuppression and viral infections. Further research is needed to more fully understand the influences of these possible risk factors.

It is also common to find malignant melanoma arising at anatomic sites not usually exposed to the sun. This is not observed with other types of skin cancers. Malignant melanoma is frequently seen in professional and managerial workers, who spend much of their working days indoors. It is believed that for malignant melanoma to occur, the effect of total sun exposure must be modified by other factors such as intense sunburn at an early age, intermittent intense exposure to ultraviolet radiation or the presence of precursor lesions.

Rates of melanoma also vary by population density, with an increased risk noted in suburbia. This is consistent with the observation that melanoma risk is higher among upper socioeconomic groups. This association also raises the possibility that some of the increased risk may result from more frequent diagnoses among the more affluent who have better access to medical care.

The Public's Knowledge, Attitudes and Behaviors

There is little published research describing current knowledge, attitudes and behaviors with respect to sun exposure and skin cancer. It is clear that large numbers of people purchase sun tanning products and sunbathe. The belief is widely held that a tan looks healthy and is viewed by many as physically attractive. Further indication of these attitudes is found in the increasing popularity of commercial tanning salons and home tanning beds.

Despite the popularity of tanning, the increasing use of sunscreens and sunblocking products would indicate a growing awareness of the potential risks involved in sun exposure. Knowledge of these products is limited, however. Stern, Weinstein and Baker found individuals who reported sunscreen use were actually using products with little or no sun-protective qualities.[3] Johnson and Lookinbill found one-third of the adults surveyed reported using sunscreens in order to promote tanning. The ability of sunscreens to stimulate melanogenesis has yet to be proven.[4] More research is needed on current knowledge, attitudes and behaviors related to the sun and other ultraviolet radiation in order to develop effective educational and motivational techniques to encourage the public to reduce any excess ultraviolet exposure.

Limitations of Sunscreen Use

Sunscreens are designed to either act as a barrier to sun (eg, titanium dioxide) or to absorb sunlight in the wavelength region (290–320nm) considered to be responsible for skin cancers. The most popular of the latter type is para-aminobenzoic acid (PABA).[5] A grading system is in place that rates sunscreens according to their sun protective factor (SPF). This represents the ratio of the minimal ultraviolet dose needed to produce erythema 24 hours after exposure with and without sunscreen. The ability of sunscreens to prevent cancers has been demonstrated experimentally in animals. Their effectiveness, however, has not been shown in human studies due to the difficulty in replicating conditions encountered by human users.[6]

Sunscreen effectiveness depends mainly on proper application. A study was conducted involving 50 regular users of sunscreen with previous solar skin damage, a group expected to be highly motivated. It was found that many sun-exposed areas such as eyebrows, ears, behind the ears, hands and neck were not protected on a daily basis. In addition, sunscreen effectiveness is also limited by other factors such as heat, wind, humidity, perspiration and water exposure.[6] The risk of skin cancer could conceivably increase if individuals apply sunscreens improperly and depend on the sunscreen for protection while increasing their sun exposure.

The most effective preventive measures are avoiding excess sun exposure and using sun barriers such as clothing and hats. The proper application of sunscreen is necessary for maximal effectiveness that includes covering all sun-exposed parts and frequent application, especially after swimming or physical activity.

Early Detection

Early detection of malignant melanoma is important to increase the individual's chance of survival. Melanomas often begin as growths similar to moles that grow in size, change color, become ulcerated and bleed easily when injured. Malignant melanoma can rapidly metastasize, but is curable if detected early and given proper treatment. Individuals should be reminded of the warning signs of melanoma which include: an asymmetrical mole where one half does not match the other; ragged, notched or blurred edges; nonuniform pigmentation; a sudden or continuing increase in the size of a mole.

Exposure to ultraviolet radiation is the most important risk factor for malignant melanoma. The evidence suggests that reduction of unprotected sun exposure could substantially reduce morbidity and mortality from both malignant melanoma and other forms of skin cancer. The New York State Department of Health is planning to conduct a survey to assess the public's knowledge, attitudes and behaviors with respect to ultraviolet exposure. Results from this survey will be used to develop and target a health education campaign.

For further information about malignant melanoma contact: Patricia Kraichy, Bureau of Cancer Epidemiology, Corning Tower Room 565, Albany, NY 12237, (518) 474-2354.

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CRITICAL CARE OBSTETRICS

By Steven L. Clark, MD, Jeffrey P. Phelan, MD, and David B. Cotton, MD. 508 pp. Oradell, NJ, Medical Economics Books, 1987. $52.95 (hardcover)

When I received my training in obstetrics and gynecology in the mid-1960s, it was felt that almost all patients could be cared for by a generalist with a relatively limited body of knowledge. At the present time adequate care for many patients requires the services of a subspecialist whose chosen area is now so complex that even he has difficulty in keeping up with the entire range of his chosen area of work. With such a discrepancy between the needs of the patient and human limitations, it is a joy to encounter such a book as *Critical Care Obstetrics*. The editors and other contributors to this volume are well known for their work in critical care of the obstetric patient.

The book is clearly written and the individual chapters appear to be relatively uniform and of very high quality. That result stems from the fact that a third of the chapters involve the editors themselves. Additionally, the book is published by Medical Economics, the publisher of *Contemporary OB/GYN*, whose articles share a similar type of format and no-nonsense manner of conveying information. The level of presentation appears most appropriate to generalists and to perinatologists who do not specialize in critical care. By the nature of their work, practicing obstetricians are called upon at a variety of times to take care of patients who are desperately ill. In the course of care for those patients, a variety of specialists involved in the administering of intensive care usually become involved. While those specialists may be very expert in the administering of intensive care, the experts are almost universally not experts in the care of the pregnant patient. That fact makes it mandatory that someone who is knowledgeable concerning the pregnant patient remain actively involved in her care.

The list of subjects covered in this book is quite comprehensive. One can, however, question why one subject is included and another is not. It is this reviewer's opinion that perhaps systemic lupus erythematosis is not a critical care item, but that renal failure is. With the exception of such questions, the book can unequivocally be recommended for anyone charged with the care of obstetric patients with acute life threatening disease, which is almost all of the OB/GYN community. It is even appropriate for the intensivist who is not obstetrically trained, since many of the issues involved in rendering care to the pregnant patient are in fact clouded by the pregnancy itself.

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PRINCIPLES OF SURGICAL TECHNIQUE. THE ART OF SURGERY

Second Edition. By Gary G. Wind, MD, and Norman M. Rich, MD. 280 pp, illustrated. Baltimore, Md, Urban & Schwarzenberg, 1987. $34.50 (softcover)

This book is geared for third and fourth year medical students and junior surgical residents, but the lessons outlined are worth the review of even the most experienced surgeon. The recurrent themes of asepsis, proper tissue handling, hemostasis, and exposure are reinforced throughout the text. A reasonably successful attempt is made to lay down the foundations of bridging the gap between "knowing" and "doing."

Both Drs Wind and Rich have an engaging style of writing and keep the theme of the book on course. The illustrations by Dr Wind highlight the text at appropriate times with great clarity. As with the first edition, this book should be well received by all students of surgery and is a welcome addition to the medical literature.

The text is divided into two parts: Part I—Definition of the Principles, and Part II—Application of the Principles, with each section complementing the other. The expanded chapter, "Historical Notes on Surgical Technique," is an excellent condensed review of the subject. Particularly strong chapters are those on wound healing, basic surgical maneuvers, and surgical stapling devices. In Part II, the authors build on the basics to provide an excellent review of the more common surgical procedures. It would have been nice, however, to see a chapter on mastectomy/axillary dissection and tracheotomy/cricothyroidotomy. A separate chapter on surgical anatomy of the abdomen is clear, concise, and an integral part of the text.

One of the overall highlights of the book is the helpful "tips" on surgical techniques for the neophyte that generally only come with experience. This book is very easy to read and digest and is must reading for those at all levels of surgical education.

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OTOLARYNGOLOGY. HEAD & NECK SURGERY: PRINCIPLES & CONCEPTS

Edited by Jerry Templer, MD, and William E. Davis, MD. 190 pp, illustrated. St Louis, Mo, Ishiyaku EuroAmerica, Inc, 1987. $35.00 (hardcover)

The authors have stated that *Otolaryngology—Head and Neck Surgery: Principles and Concepts*, is another introductory textbook that attempts to provide information that most medical graduates should know about the field of otolaryngology. This is indeed an ambitious and laudable task, and one that can ultimately lead to criticism. The scope of contemporary otolaryngology/head and neck surgery is so vast that the authors of introductory texts always have difficult decisions about what to include and how much detail to provide.

This book is written in an easy reading style that, combined with numerous illustrations and diagrams, permits the reader to cover the book's contents in a short period of time. The chapters cover most areas of the field including: the cochlea and vestibular system, the nose and the paranasal sinuses, the oral cavity, nasopharynx, pharynx, facial, plastic and reconstructive surgery, head and neck cancer, and allergy.

The strength and value of this textbook are that it provides the reader with an overview of the field of otolaryngology/head and neck surgery. As such, it is most suited for medical students who are looking for a comfortable review of this specialty. The book does not provide enough in-depth information on specific subjects to warrant its use as a reference for diagnosis or treatment. As such, it will have little practical value for the practicing physician.

This reviewer is also involved in teaching medical students and has continued to search for a text that provides just enough and not too little or too much material regarding what medical students need to know about this field. Although starting out with laudable goals, in my opinion this text falls short of being one I would use for my students. There are too many areas in which insufficient information is given, as for important subjects like cholesteatoma or tinnitus. Vasomotor rhinitis is allotted a paragraph, as is nasal encephaloceles. Yet, medical students will see thousands of patients with vasomotor rhinitis and probably will never see anyone with a nasal encephalocele. The inclusion of a color photograph section is a good idea, but clinical pathologic photos of common tympanic membrane, laryngeal, and nasal pathologic conditions would have much more practical value than photos of facial trauma. Also, the chapter on endoscopy and descriptions of surgical techniques and figures of anastomosing parotid ducts may provide information not appropriate to an introductory text. A glaring omission is the failure to mention nasal steroid sprays as a treatment for nasal allergy in the allergy chapter.

This book admirably serves to provide a review of the broad field of otolaryngology and head and neck surgery. However, although others may disagree, I am still looking for a practical introductory book that provides usable information on the common diseases of the head and neck region that would be read by medical students and used by primary care physicians.

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HERNIA REPAIR WITHOUT DISABILITY

Second Edition. By Irving L. Lichtenstein, MD. 252 pp, illustrated. St Louis, Mo, Ishiyaku EuroAmerica, Inc, 1987. $85.00 (hardcover)

Many principles and prejudices have arisen in the discussion of abdominal wall herniae. This wide variety of opinion involves the pathophysiology of the hernia defect and the various techniques of reconstruction of the abdominal wall. This second edition of Dr Lichtenstein's book, originally published in 1970, is based on the author's experience with 6,231 operations performed for abdominal wall herniae over a 14-year period. The book clarifies some of the common misconceptions regarding herniorraphy and proposes a standard method of repair, based on sound anatomic and surgical principles.

The early chapters explain the principles of diagnosis and anatomy and represent one of the most useful aspects of the book. The art work and legends are clear, giving good three-dimensional representations of various hernia defects and the technique of repair. The overall organization is good. However, there is much repetition, and chapters could have been consolidated. For example, chapter four, entitled "Why Is Man Peculiarly Liable to Hernia?" is five pages in length and should be included in chapter three, "Surgical Anatomy of the Inguinal Region." Also, statements and conclusions concerning direct and indirect herniae are made without substantiation by data.

The focus of this book is different from that of other works on this topic, such as the multiple (86) author book *Hernia* edited by Nyhus and Condon. The latter is an encyclopedic overview, which is not the intent of the present work. However, since Dr Lichtenstein briefly discusses the history of hernia repair, recent data on other methods of repair are called for but not presented. The strength of the book lies in the fact that over 6,000 patients with herniae have been successfully treated by the author, thereby supporting his observations.

However, the analysis of this data seems cursory for such a large number of patients and leaves many questions unanswered. First, with a follow-up rate of 91%, the long term results in 9% of patients cannot be evaluated and in some scientific analyses would be considered failures of therapy. Secondly, the author apparently altered the technique of repair after 1984, and currently employs mesh as a reinforcing layer. The data influencing this decision and, as the author admits, long term follow-up are also lacking. Moreover, the analysis of the results does not distinguish the two groups of patients (before and after 1984). Third, the true lack of disability encountered by the patients treated could be better quantitated, eg, days at home, days at work, part time vs full time, and analysis by occupation. This would allow the reader to conclude, as the title suggests, that his method of repair is associated with lack of disability.

This text is a useful addition to the library of general surgeons, since it offers a well-described and illustrated technique for the repair of abdominal wall herniae based on sound principles and extensive clinical experience.

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INFECTIVE DISEASE IN PRIMARY CARE: A SYMPTOMATIC APPROACH

By Robin Hull, MB. 234 pp, illustrated. Dobbs Ferry, NY, Sheridan House, Inc, 1987. $17.50 (paperback)

This book, *Infective Disease in Primary Care*, is written by an author who is a practicing primary care physician with extensive clinical experience in other countries and a strong interest in infectious diseases. According to him, the book is meant to educate and help physicians of first contact who lack experience in infectious diseases to recognize such diseases in early stages, and to take appropriate steps for prompt management. In addition, the book is to serve as "stimulus to that first thought" and advise the clinician to seek further details from other infectious disease books.

The manual is written in a very simplified manner, easy to understand and read. The content is divided into various chapters dealing with different symptoms such as malaise and fever; spots and rashes; gastrointestinal, respiratory, or neurological symptoms; sexually transmitted disease, etc. Individual diseases causing those symptoms are arranged in alphabetical order, punctuated by good two-line histories of the disease and case histories as encountered by the author in his practice.

Unfortunately, the information contained in the book is superficial, insufficient, and at times outdated. For example, the author advises that in diagnosing endocarditis, blood cultures be taken twice a day for three to five days, and elsewhere states that the etiology of Lyme disease is unknown. Inaccurate statements include the assertion that splenic rupture occurs because of heavy handed palpation and that tetanus, malaria, or melioidosis are commonly seen in IV drug addicts. Very little guidance is offered as to what abnormalities to expect in laboratory evaluation. There is a glaring omission of detailed treatment plans except for a few diseases such as malaria (even here specific drugs and dosages are not mentioned). There is frequent repetition of the same information related to various diseases under different headings. Considerable emphasis is given to certain exotic diseases like Crimean Congo hemorrhagic fever, Ebola fever, Marburg disease, and Ockelbo disease, which are rarely encountered in the United States. There is little information as to how to differentiate between diseases presenting with identical clinical manifestations. The final chapter deals with the medical side of organizing expeditions. The author points out some practical and good suggestions for physicians who are likely to participate in such adventures.

In summary, the book falls short of expectations as a manual to be consulted by practicing physicians because of a lack of adequate detailed information. It is an ideal introductory book on infectious diseases and tropical medicine for medical students. However, it has limited place on the bookshelves of either emergency room physicians, family practitioners, or general practitioners since better infectious disease books are available.

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MANUAL OF CLINICAL ONCOLOGY

Fourth Edition. Edited by C. D. Sherman, MD, K. C. Calman, MD, S. Eckhardt, MD, I. Elsebai, MD, D. Firat, MD, D. K. Hossfeld, MD, J. P. Paunier, MD, and B. Salvadori, MD. 372 pp, illustrated. New York, Springer-Verlag, 1987. $28.00 (softcover)

This volume represents the fourth attempt of the Union Internationale Contre de Cancer (UICC) to provide a synopsis of all areas of human oncology in a small volume. The prime audience is stated to be graduating medical students and young practitioners of medicine by whom "it should be read and understood in its entirety." There is probably no chance and no way that this goal will be achieved, and probably that is just as well.

The manual consists of three parts, distinctive one from another in much more than their subject matter. Part One surveys the nature, occurrence, cause, prevention, diagnosis, and general strategies of management of human cancers. The text is well presented, and much current conventional wisdom is presented; indeed, certain areas are dealt with in quite considerable detail. While the table of contents is helpful in finding specific information, no index is provided, perhaps because of the above noted intention that everyone should read it from start to finish without interruption. There is also no adequate list of references.

The second and longest section presents a series of brief clinical descriptions, and summary suggestions for management, for all major malignancies. These summaries vary in their lucidity and accuracy from excellent to barely tolerable. There are more frequent inaccuracies than one would wish or expect, and in addition points of view are presented as facts, and alternate opinions are not suggested. This is particularly bothersome since in this section also references are virtually absent. Finally, new therapeutic approaches such as autologous bone marrow transplantation for lymphomas and solid tumor are not covered here, and relatively new drugs such as α-interferon or pentostatin for hairy cell leukemia are not mentioned despite the striking results which have been reported for these treatments during the last five years. These, and other similar innovations, should appear in a 1987 publication, particularly in one with such an authoritarian gestalt and tone.

Part Three is a departure from its predecessors. It contains concise summaries of recent advances in clinical and pre-clinical oncology: biomarkers, imaging, oncogenes, molecular biological approaches to cancer diagnosis, laser therapy including the use of photoactivated dyes, bone marrow transplantation (finally), and drug containing lysozymes and lymphokines, and concludes with a short review of AIDS. This section was clearly written last. It contains more current information, and provides a copious bibliography for each of its many subjects, eg, 36 references on tumor stem cell assays, and 34 references on magnetic resonance imaging.

While the *Manual of Clinical Oncology* contains much valuable information, it is not consistently presented. I doubt very much whether any new physician will spend the day or two required to read it cover to cover—when will he or she find the time, even in a post-Bell-Commission residency, or in a new practice situation? Conversely, the absence of an index will discourage those who might wish to look up specific facts, and the absence of references in the first two segments will preclude checking or expanding on specific points in question. The final part is more appropriate for someone specializing in oncology who wishes a quick epitome plus a large reference update in a particular area of cancer research. For students, house staff, practitioners, and cancer specialists alike, there exist superior texts and references. I hope the well motivated members of the UICC's Manual Revision Committee will make their next edition more practical, better referenced, and better tailored to the audience they wish to reach.

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REMEMBER ME. A COLLECTION OF POEMS

By Albert D. Anderson, MD. 39 pp. New York, Albert D. Anderson, MD, 1987. (softcover)

It was difficult to write a meaningful review of this unique little book. The first reading took only 30 minutes but to completely understand the deep emotions reflected in each poem required much more. It helped to study the frontispiece depicting two index fingers "almost touching" and the photograph of the author's facial expression on the back cover. By reciting aloud each poem while imagining both pictures, the reader can capture the inner feelings Dr Anderson poured into his book.

The slow progression of the author's physical deterioration resulting from the rare disease syringomyelia obviously influenced the selection of topics for his poems. Typical examples are the following four: "Touch me," "I must move," "Fear," and "Loneliness." Stricken at age 23 as a junior medical student, Dr Anderson completed his studies at Harvard and was forced soon after to abandon his surgical training for a short career in internal medicine. Finally he turned to the rehabilitation of patients more or less disabled than he is.

If you need a "profile in courage" to overcome a period of depression, read *Remember Me* by Dr Albert Anderson.

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RESOURCES IN MEDICAL HISTORY. ON INSANITY AND ITS CLASSIFICATION

By Vincenzo Chiarugi. Translated with a critical introduction by George Mora, MD. 380 pp. Canton, Mass, Watson Publishing International, 1987. $35.00 (clothbound)

This book, published during the Enlightenment, is an example of the introduction of the practice of humane treatment of the mentally ill. Its main purpose is to classify psychiatric disorders, but it also attempts to explain their etiology and offers forms of treatment. The amount of scientific and medical information established during the 17th and 18th centuries made codification the order of the day, so that by 1800 Chiarugi was one of a large number of clinicians who had classified his observations. His classification was very similar to that of Philippe Pinel in France, and is of course quite dissimilar to DSM III-R.

Chiarugi was well acquainted with the psychiatric literature, and worked hard at introducing innovative practices in the treatment of the mentally ill. The success of his reforms in Florence led to his being invited by the government of Venice to serve as consultant for improving the mental hospital of S. Servolo. He was also appointed honorary professor at the medical faculty of the University of Pisa. He taught psychiatry but maintained an interest in agriculture, chemistry, and dermatology and published papers on these topics. In 1818 he was named director of postgraduate training for physicians at Santa Maria Nuova.

Chiarugi, like other authors at the time, was very limited by a lack of real understanding of the causes of psychological disorders. Despite the fact that belief in reason was replacing tradition and faith, his ideas incorporate those of Descartes, Aristotle, Plato, and Saint Thomas.

The book is composed of three volumes. Its emphasis is on the classification of mental illness, but etiological and treatment issues are also included. The first volume addresses the causes and treatment of insanity in general. The second volume covers melancholia, mania, and amentia. The third deals with the nosology of insanity and includes clinical observations with anatomical pathological findings.

Chiarugi maintained that nerves are small tubes containing a fluid secreted by the blood. Motor activity is due to action of the nervous fluid from the brain to the periphery. Sensation is due to stimulation of nervous fluid from the periphery carried to central organs. He attempted to resolve the body and soul dichotomy by postulating the existence of the "sensorium commune," an organ located at the base of the brain where "the soul has the closest connection with the body."

Chiarugi repeatedly stressed the importance of the sensorium commune for the understanding of mental illness. For example, in mania there is increased activity in this organ due to organic and psychological causes. Symptoms would be controlled by reestablishing the harmonious balance in the organ. Treatment modalities might include the administration of sedatives as well as phlebotomy. The development of trust between patient and physician is emphasized in the book, as well as the physician's role in instilling a sense of hope in the patient.

This book was written in 18th century Latin-style Italian which makes it difficult to read despite the excellent translation by Dr George Mora. The contents could have been better organized. There are numerous repetitions. All of this takes away from the clarity of the book.

Nonetheless the book is well worth the effort. It is an important contribution to the history of psychiatry. It provides a fascinating view of the influence of the religious and political climate of the time on the comprehension of mental illness. The book gives us an invaluable understanding of a key developmental phase in the field of psychiatry.

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BOOKS RECEIVED

AIDS

How to Find Information About AIDS. By Virginia A. Lingle, MSLS, and M. Sandra Wood, MLS. 130 pp. New York, The Haworth Press, Inc, 1988. $14.95 (hardcover)

Private Acts, Social Consequences: AIDS and the Politics of Public Health. By Ronald Bayer. 282 pp. New York, The Free Press, 1989. $22.95 (hardcover)

GENETICS

Medical Genetics Handbook. By Harold Chen, MD. 384 pp, illustrated. St Louis, Mo, Warren H. Green, Inc, 1988. $72.50 (softcover)

GERIATRICS

Geriatric Medicine Annual 1989. Edited by Richard J. Ham, MD. 320 pp, illustrated. Oradell, NJ, Medical Economics Books, 1989. $42.95 (hardcover)

INTERNAL MEDICINE

Contemporary Internal Medicine: Clinical Case Studies. Volume 1. Edited by Juan Bowen, MD, and Ernest J. Mazzaferri, MD. 319 pp, illustrated. New York, Plenum Publishing Corp, 1988. $45.00 (hardcover)

Physician's Guide to Insulin-Dependent (Type I) Diabetes. Diagnosis and Treatment. 150 pp, illustrated. Alexandria, Va, American Diabetes Association, 1988. $22.45 (spiralbound)

Physician's Guide to Non-Insulin-Dependent (Type II) Diabetes. Diagnosis and Treatment. Second Edition. 98 pp. Alexandria, Va, American Diabetes Association, 1988. $22.45 (spiralbound)

LEGAL MEDICINE

Life-Sustaining Treatment: Making Decisions and Appointing a Health Care Agent. 180 pp. New York, The New York State Task Force on Life and the Law, 1987. $8.50 (softcover)

Surrogate Parenting: Analysis and Recommendations for Public Policy. 143 pp. New York, The New York State Task Force on Life and the Law, 1988. $7.00 (softcover)

MEDICAL HISTORY

Yankee Surgeon. The Life and Times of Usher Parsons, 1788–1868. By Seebert J. Goldowsky, MD. 450 pp, illustrated. Boston, Mass, The France A. Countway Library of Medicine, 1988. $24.50 (hardcover)

MISCELLANEOUS

The New Aerobics for Women. By Kenneth H. Cooper, MD, and Mildred Cooper. 324 pp. New York, Bantam Books, 1988. $12.95 (paperback)

Sensible Living: Winning Your Fight Against Arthritis. By Virgil Harbert. 143 pp, illustrated. New York, William Morrow & Co, Inc, 1988. $15.95 (clothcover)

Patient Care Flowchart Manual. Fourth Edition. Edited by Steven R. Alexander, MD. 646 pp. Oradell, NJ, Medical Economics Books, 1988. $52.95 (hardcover)

Symptoms After 40. What They Mean, What to Do About Them, What's Normal With Aging and What's Not. By Kenneth Anderson. 324 pp. New York, Arbor House Publishing Co, 1987. $8.95 (paperback).

In Quest of the Mythical Mate. A Developmental Approach to Diagnosis and Treatment in Couples Therapy. By Ellyn Bader, PhD, and Peter T. Pearson, PhD. 288 pp. New York, Brunner/Mazel Publishers, 1988. $27.95 (hardcover)

Black Families in Crisis: The Middle Class. Edited by Alice F. Coner-Edwards, DSW, and Jeanne Spurlock, MD. 320 pp. New York, Brunner/Mazel Publishers, 1988. $30.00 (hardcover)

A Piece of My Mind. America's Doctors Share Their Most Dramatic, Inspiring, and Moving Experiences. Edited by Bruce B. Dan, MD, and Roxanne K. Young. 256 pp. New York, Feeling Fine Programs, Inc, 1988. $18.95 (hardcover)

The New Good Housekeeping Family Health and Medical Guide. By the Editors of Good Housekeeping. 788 pp, illustrated. New York, William Morrow & Co, 1989. $24.95 (cloth)

When Doctors Get Sick. Edited by Harvey Mandell, MD, and Howard Spiro, MD. 464 pp. New York, Plenum Publishing Corp, 1987. $25.00 (hardcover)

The Doctor as a Person. Edited by Jane Eys, MD, and John P. McGovern, MD. 151 pp, illustrated. Springfield, Ill, Charles C. Thomas, Publisher, 1988. $29.75 (hardcover)

The Penguin Medical Encyclopedia. Third Edition. By Peter Wingate, MD, and Richard Wingate. 519 pp, illustrated. New York, Viking Penguin, Inc, 1988. $8.95 (paperback)

Medical Mavericks. Volume One. By Hugh Desaix Riordan, MD. 102 pp. Wichita, Kan, Bio-Communications Press, 1988. $7.95 (paperback)

OTOLARYNGOLOGY

Otolaryngology: Problems In Primary Care. Edited by D. Thomas Upchurch, MD. 350 pp, illustrated. Oradell, NJ, Medical Economics Books, 1989. $42.95 (softcover)

PEDIATRICS

Dr. Eden's Healthy Kids. The Essential Diet, Exercise, and Nutrition Program. By Alvin N. Eden, MD. 285 pp. New York, Nal Penguin, Inc, 1987. $8.95 (paperback)

PHARMACOLOGY

Drug Interactions. Sixth Edition. By Philip D. Hansten, PHARM D, and John R. Horn, PHARM D. 448 pp, Philadelphia, Pa, Lea & Febiger, 1989. $65.00 (3-ring looseleaf binder)

PSYCHIATRY

Psychiatry and Mental Health Science Handbook. Co-edited by Caudewell S. Thomas, MD, and Jacob Jay Lindenthal, PhD. 267 pp. St Louis, Mo, Warren H. Green, Inc, 1988. $29.50 (paperback)

Issues In Community Mental Health: Housing. Edited by Steven J. Friedman, MS, and Kenneth G. Terkelsen, MD. 105 pp. Canton, Mass, Prodist, 1988. $6.95 (paperback)

Psychotherapy of the Disorders of the Self: The Masterson Approach. Edited by James F. Masterson, MD, and Ralph Klein, MD. 460 pp. New York, Brunner/Mazel Publishers, 1989. $39.95 (hardcover)

PSYCHOLOGY

Manual for Clinical Psychology Trainees. Second Edition. By James P. Choca, PhD. 192 pp. New York, Brunner/Mazel Publishers, 1988. $16.95 (paperback)

TOXICOLOGY

Comprehensive Review in Toxicology. Second Edition. By Peter D. Bryson, MD. 696 pp, illustrated. Rockville, Md, Aspen Publishers, Inc, 1989. $137.00 (hardcover)

TRANSPLANTATION MEDICINE

Transplantation in New York State: The Procurement and Distribution of Organs and Tissues. 164 pp. New York, The New York State Task Force on Life and the Law, 1988. $8.00 (softcover)

Plan to reform Medicaid system is on the table

The Health Policy Agenda for the American People (HPA), a coalition of 172 public and private sector groups representing consumers, labor, health care providers, and the government, has prepared an eight-point plan for overhauling Medicaid. The plan emphasizes expanded and fair allocation of services and funds to those in need as well as standardization of benefits nationwide.

The HPA, an American Medical Association sponsored group, is pressing President Bush and Congress for immediate Medicaid reform. According to the coalition, approximately 11 million people with incomes below the federal poverty level do not qualify for Medicaid under current eligibility guidelines, and are therefore unable to obtain adequate health care. In addition, as each state currently determines the local dispensation of funds and services, health care benefits under Medicaid vary greatly from state to state. The HPA plan mandates national standards for eligibility, benefits, and reimbursement, with all Americans having incomes below the federal poverty level receiving standardized health care benefits from Medicaid, and most Medicaid reimbursements being at cost or at least at a level comparable to that of Medicare.

Specifically, the HPA proposes that there no longer be a link between Medicaid eligibility and welfare programs. Rather, the federal poverty level would be the cutoff to qualify for Medicaid. The plan also calls for a "medically needy" program that would provide health care benefits for persons with incomes above the eligibility level but who are impoverished due to catastrophic medical expenses. Included in the standardized benefits package contained in the HPA plan are the following: physician services; inpatient and outpatient hospital services; laboratory and x-ray services; prescription drugs; institutional care for the elderly and the physically or mentally disabled; dental services; early and periodic screening, diagnosis, and treatment; family planning services; home health and personal care services; and other medically necessary professional services.

The net cost of these reforms, taking into account the cost of the programs and the potential savings resulting from an improved Medicaid system, is an estimated $13.2 billion, according to a cost analysis performed by the Harvard University School of Public Health.

FDA issues warning to contact lens wearers

An FDA Safety Alert entitled "Homemade Saline Solutions for Contact Lenses" and sent to about 50,000 opthalmologists, optometrists, and opticians, warns of the potential dangers when using homemade saline solutions in contact lens care. Improper lens care can lead to infections and can result in blindness.

Homemade solutions, prepared by dissolving salt tablets in distilled water, are not sterile and can contain bacterial contaminants and such infectious microorganisms as *Acanthamoeba*. If homemade saline solutions are used before or during heat disinfection of contact lenses, the heat treatment will destroy the infectious agents. However, eye care professionals should instruct their patients never to use homemade saline solutions as a rinse after heat disinfection, as an eyedrop or wetting solution, or in any stage of a chemical or hydrogen peroxide disinfection process. In addition, it is important to prepare fresh saline solution each day and to sterilize the saline solution bottle with boiling water at least once a week.

The Centers for Disease Control has demonstrated an association between the use of homemade saline solutions and *Acanthamoeba* infection, a painful condition that is difficult to treat and can cause blindness.

Preschool eye screening gets a boost

Early detection of eye and visual disorders is important for planning and implementing an effective treatment regimen. A promising new instrument called the Visiscreen 100 may prove useful in screening children as young as three months for vision problems. In general, it is difficult to test children who cannot speak and cannot yet read an eye chart. The Visiscreen 100 overcomes this problem by taking photographs of the eye, which are then evaluated by an ophthalmologist.

The device has been used to diagnose cataracts, nearsightedness, far-sightedness, astigmatism, and strabismus in a group of 57 children aged three months to eight years. The investigators found an 84% agreement rate between the results of conventional eye screening examinations and findings of photorefraction evaluations.

While this method requires further testing and refinement, the technology has opened the door to early detection of eye disorders in infants and young children.

Cyclosporine shows promise against dry eye

Dry eye, a condition characterized by insufficient tear production, causes pain and, eventually, blindness. Researchers believe that one form of the disorder is associated with an autoimmune response, in which the body attacks its own tear glands. Preliminary studies suggest that localized application of small amounts of cyclosporine may be an effective treatment for this condition (*Science News* 1988; 134:215).

Cyclosporine, which suppresses the immune system, had not been considered a feasible treatment for dry eye because of its toxic side effects when administered systemically. Investigators at the University of Georgia (Athens) experimented with cyclosporine eye drops in dogs and found that the drops "substantially restored tearing, reduced corneal scar tissue and speeded the healing of corneal ulcers . . . and may prevent further tear-gland destruction."

By limiting cyclosporine exposure to the affected eye, the researchers were able to achieve therapeutic effects with a dosage equal to only 1% of the oral dose. Cyclosporine had been shown to be effective in treating autoimmune-mediated dry eye for up to two years, with no adverse effects. Promising results have been reported from a

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NEWS BRIEFS are compiled and written by Vicki Glaser.

study in Scotland using a cyclosporine ointment to treat patients with dry eye. It is hoped that the results of a larger clinical trial, which will begin in early 1989 in the United States, will support these encouraging findings.

Victims of SIDS demonstrate abnormal heart rate at birth

Approximately 8,000 babies in the United States are victims of sudden infant death syndrome (SIDS) each year, according to the Association of Professional Sleep Societies. Efforts to prevent this fatal disorder focus, in part, on identifying babies at risk for SIDS. Now, researchers at the University of California (Los Angeles) have found that victims of SIDS have an abnormal heart rate in their first month of life—weeks or months before their death.

Previous studies had shown victims of SIDS to have high heart rates, but it was unclear whether these babies had abnormal heart rates or were simply awake for longer period of time (heart rates are higher during awake periods). In this study, the researchers were able to differentiate between heart rate recordings taken during sleep and waking periods.

In comparison to healthy newborns, SIDS victims were shown to have significantly higher heart rates both when awake and during sleep in the first month of life. This correlation does not imply a cause and effect relationship. The abnormal heart rate and SIDS may be related; alternatively, there could be a third factor that causes both disorders. The higher heart rate among SIDS victims was also found during active sleep in the second month of life. This concurs with the findings from previous studies that heart rate abnormalities in SIDS victims are most apparent during active sleep.

Implantable defibrillators undergoing clinical tests

Defibrillators save lives by delivering an electric shock to a heart that is in ventricular fibrillation—a nonproductive, uncoordinated contraction of the heart muscle. The defibrillator reinstitutes a normal heart beat and restores circulation. Currently, defibrillation is performed externally by a physician or paramedic. Implantable defibrillators that could sense the onset of ventricular fibrillation and automatically provide an electric shock directly to the heart are under development.

Smaller, lighter devices have made implantation easier. However, attachment of the defibrillator's electrodes to the heart still requires open-chest surgery, a very expensive procedure. New models of defibrillators, now being tested, would enable transvenous implantation of the electrodes, in a procedure similar to that used for pacemaker implantation. In addition, improvements in the design and positioning of the electrodes and in the shape of the shock waveform have resulted in less energy being needed to achieve defibrillation.

Other design improvements would enable the defibrillator to be combined with a pacemaker and a device to regulate heart beat arrhythmia in one device that would control any heart rhythm abnormality. Programmable defibrillators would allow physicians to tailor the monitoring and energy variables for each patient.

New osteoporosis treatment combines fluoride and calcium

Combination therapy with fluoride and calcium has shown success in the treatment of spinal osteoporosis, according to researchers at the University of Texas Southwestern Medical Center in Dallas (*Science News* 1989; 135:36). After 3½ years of testing in 65 patients, the researchers report increased spinal bone mass of 3% to 6% each year of treatment—good news for the approximately five million people in the United States who suffer from spinal osteoporosis.

Previous efforts to use fluoride-calcium therapy for bone restoration were hampered by severe adverse effects, including damage to the stomach lining and subsequent bleeding due to excessive exposure of the stomach to the fluoride, as well as abnormal bone development and joint pain and swelling. Development of a gradual-release fluoride tablet has sharply reduced the incidence of gastrointestinal problems and joint pain and swelling, giving new life to the prospects for combination fluoride-calcium therapy.

During treatment, the fluoride stimulates osteoblasts to increase bone mass by producing a protein matrix to be filled with calcium deposits. The calcium supplements taken by the patients ensure sufficient calcium supply to make strong new bone.

The researchers plan to present the fluoride-calcium therapy to the Food and Drug Administration this spring for approval as a treatment of spinal osteoporosis. They hope that future research will demonstrate the effectiveness of this therapy for all forms of osteoporosis, which affects about 20 million people in this country.

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

William Aronson, MD, New York. Died January 4, 1989; age 92. Dr Aronson was a 1924 graduate of the New York University School of Medicine. He was a Fellow of the American College of Pathologists and a Diplomate of the American Board of Pathology. His memberships included the New York State Society of Pathologists, the New York Pathological Society, the American Society of Clinical Pathologists, the Bronx County Medical Society, and the Medical Society of the State of New York.

John Richard Bongiovanni, MD, Ogdensburg. Died December 29, 1988; age 57. Dr Bongiovanni was a 1957 graduate of the State University of New York at Buffalo School of Medicine. He was a Diplomate of the American Board of Urology. His memberships included the New York State Surgical Society, the New York State Urological Society, the American Urological Association, the American Fertility Society, the American Geriatrics Society, the Medical Society of the County of St Lawrence, and the Medical Society of the State of New York.

Milton J. Brothers, MD, Fort Lee, New Jersey. Died January 9, 1989; age 63. Dr Brothers was a 1953 graduate of the State University of New York Health Science Center at Brooklyn. He was a Diplomate of the American Board of Internal Medicine. His memberships included the Clinical Society, the New York Diabetes Association, the New York County Medical Society, and the Medical Society of the State of New York.

Dong Sun Chu, MD, New York. Died November 15, 1988; age 63. Dr Chu was a 1960 graduate of the College of Medicine, Seoul National University, Seoul, South Korea. He was a Diplomate of the American Board of Physical Medicine and Rehabilitation. He was a member of the American Academy of Physical Medicine and Rehabilitation, the New York County Medical Society, and the Medical Society of the State of New York.

Robert Sohn Coles, MD, New York. Died January 4, 1989; age 69. Dr Coles was a 1945 graduate of the New York University School of Medicine. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Ophthalmology. His memberships included the New York Academy of Medicine, the New York Society for Clinical Ophthalmology, the American Academy of Ophthalmology and Otolaryngology, the Pan American Association of Ophthalmology, the New York County Medical Society, and the Medical Society of the State of New York.

Emerick Demeter, MD, Great Neck. Died January 25, 1989; age 67. Dr Demeter was a 1950 graduate of the Charles Univerzita Fakulta of Ped-Gen Medicine, Praha, Czechoslovakia. He was a Diplomate of the American Board of Radiology. He was a member of the New York Roentgen Society, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Frank Alexander Dick, MD, Vero Beach, Florida. Died January 23, 1989; age 77. Dr Frank was a 1937 graduate of the Medical College of Virginia Commonwealth University School of Medicine, Richmond. He was a member of the New York Academy of Medicine, the Medical Society of the County of Fulton, and the Medical Society of the State of New York.

Michael L. Gilbert, MD, Manhasset. Died January 24, 1989; age 70. Dr Gilbert was a 1944 graduate of the New York University School of Medicine. He was a Fellow of the American Academy of Pediatrics and a Diplomate of the American Board of Pediatrics. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Leon N. Goldenberg, MD, New York. Died December 29, 1988; age 75. Dr Goldenberg was a 1938 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a Fellow of the American Academy of Pediatrics and a Diplomate of the American Board of Pediatrics. He was a member of the American Public Health Association, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Jacob Warren Goldstein, MD, Lawrence. Died September 27, 1988; age 71. Dr Goldstein was a 1942 graduate of the Faculte de Medecine de l'Universite de Lausanne, Lausanne, Switzerland. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Ophthalmology. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

M. Allen Greenwood, MD, Staten Island. Died January 15, 1989; age 74. Dr Greenwood was a 1942 graduate of the Faculte de Medecine de l'Universite de Lausanne, Lausanne, Switzerland. He was a Qualified Fellow of the International College of Surgeons and a Diplomate of the American Board of Ophthalmology. His memberships included the Pan American Medical Association, the American Academy of Ophthalmology and Otolaryngology, the Richmond County Medical Society, and the Medical Society of the State of New York.

Paul Allen Hansch, MD, New York. Died November 16, 1988; age 66. Dr Hansch was a 1955 graduate of the Cornell University Medical College, New York. He was a Diplomate of the American Board of Psychiatry and Neurology. He was a member of the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Alexander Sandor Honig, MD, Bronx. Died December 7, 1988; age 80. Dr Honig was a 1932 graduate of the Universitaet Wien, Medizinische Fakultaet, Wien, Austria. His memberships included the New York State Society of

Internal Medicine, the American Geriatrics Society, the Bronx County Medical Society, and the Medical Society of the State of New York.

Laszlo Kadar, MD, New York. Died December 30, 1988; age 79. Dr Kadar was a 1934 graduate of the Orvosi Fakultas Tudomanyegyetem, Budapest, Hungary. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the Academy of Psychosomatic Medicine, the American Geriatrics Society, the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Harry H. Kaufman, MD, Miami Beach, Florida. Died November 26, 1988; age 80. Dr Kaufman was a 1931 graduate of the New York University School of Medicine. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Auguste Kron, MD, New York. Died December 24, 1988; age 86. Dr Kron was a 1926 graduate of the Medizinische Fakultaet der Julius Maximiliams Universitaet, Wurzburg, Bayern, Germany. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

David Leisten, MD, Forest Hills. Died January 25, 1989; age 65. Dr Leisten was a 1952 graduate of the New York University School of Medicine. He was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the American Gynecological Society, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Lloyd H. Leve, MD, Rochester. Died January 11, 1989; age 58. Dr Leve was a 1958 graduate of the State University of New York at Buffalo School of Medicine. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Orthopedic Surgery. He was a member of the American Academy of Orthopaedic Surgeons, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Robert Daniel Levinson, MD, New York. Died November 21, 1988; age 82. Dr Levinson was a 1933 graduate of the Faculte de Medecine de l'Universite de Geneve, Geneva, Switzerland. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

David Osias Linder, MD, Lawrence. Died December 21, 1988; age 78. Dr Linder was a 1938 graduate of the Faculte de Medecine de l'Universite de Lausanne, Lausanne, Switzerland. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Irwin I. Lubowe, MD, New York. Died January 4, 1989; age 84. Dr Lubowe was a 1930 graduate of the New York Medical College. He was a Fellow of the American College of Allergists and a Diplomate of the American Board of Dermatology, Inc. His memberships included the New York State Dermatological Society, the American Academy of Dermatology, the American Public Health Association, the American Geriatrics Society, the Pan American Medical Association, the Society for Investigative Dermatology, the New York County Medical Society, and the Medical Society of the State of New York.

Michael Joseph Napolitano, MD, Flushing. Died January 5, 1989; age 61. Dr Napolitano was a 1954 graduate of the Facolta di Medicina e Chirurgia dell' Universita di Roma, Rome, Italy. He was a Fellow of the American College of Radiology and a Diplomate of the American Board of Radiology. His memberships included the Radiological Society of New York State, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Abraham Penner, MD, New York. Died January 25, 1989; age 82. Dr Penner was a 1931 graduate of the Columbia University College of Physicians and Surgeons. He was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. His memberships included the New York Academy of Medicine, the American Gastro-enterological Association, the New York County Medical Society, and the Medical Society of the State of New York.

John Pugliese, MD, Bronx. Died November 24, 1988; age 67. Dr Pugliese was a 1946 graduate of the Medical College of Wisconsin, Milwaukee, Wisconsin. He was a Fellow of the American Academy of Family Physicians. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Norman Davis Rochman, MD, Plainview. Died January 15, 1989; age 63. Dr Rochman was a 1961 graduate of the University of Tennessee Center for Health Sciences, Memphis, Tennessee. He was a member of the American Society of Abdominal Surgeons, the Nassau County Medical Society, and the Medical Society of the State of New York.

Salvatore F. Sorgi, MD, Manhasset. Died September 7, 1988; age 92. Dr Sorgi was a 1920 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

L. Jackson Spitz, MD, Brooklyn. Died October 2, 1988; age 77. Dr Spitz held Registrable Qualification Granted by the Scottish Conjoint Board in 1939. He was a member of the American Academy of Family Physicians, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

George L. Sprenger, MD, Plandome. Died July 23, 1988; age 85. Dr Sprenger was a 1932 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

John E. Welch, MD, Brooklyn. Died December 26, 1988; age 72. Dr Welch was a 1952 graduate of Meharry Medical College, Nashville, Tennessee. He was a member of the American Society of Anesthesiologists, Inc, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Kurt Zinner, MD, Wellsville. Died December 1, 1988; age 82. Dr Zinner was a 1931 graduate of the Medizinische Fakultaet, Albert Ludwigs Universitaet, Freiburg im Breisgau, Baden-Wurttemberg, Germany. He was a Fellow of the American College of Cardiology and the American College of Chest Physicians. He was a member of the Medical Society of the County of Allegany and the Medical Society of the State of New York.

FOR MY PATIENTS

NUMBER 8/1989

ADULTS NEED SHOTS TOO

Dear Patient,

"Doctor, when will my baby begin her 'shots'?"

Very often that's one of the questions asked by a new mother after she delivers her baby. During the past fifty years in the United States, the immunization of infants and young children has become a routine way of life. All states now require basic vaccines for all children entering nursery, day care or elementary schools. As a result, the immunization levels of school-age children are 90% or better.

The widespread use of childhood immunizations has prevented the occurrence of many diseases which were common in the 1930s and 1940s. Serious and fatal cases of **measles, tetanus, diphtheria, whooping cough, and poliomyelitis** are now rare diseases. Thanks to the worldwide success of the smallpox vaccine, the fearful outbreaks of **smallpox** have been completely eliminated from our planet. Birth defects due to **rubella (German measles)** infections in pregnancy are now "a thing of the past" in all countries with childhood vaccine programs.

Despite all this medical progress, many vaccine-preventable diseases now crop up throughout the country in **adults and adolescents.** These persons never had these diseases when they were children or were never given some of the important "shots" when they were infants or children.

National surveys now show that about 50% or more of Americans over 18 years of age are **not immune (protected) against tetanus, diphtheria, measles, polio, rubella, and mumps.** All of these can lead to major complications or death. It is important to remember that we have all the available vaccines necessary to prevent these diseases, and they are almost 100% ***effective*** in adults. And they are all safe—with very few, if any side-reactions.

GUIDELINES

The Public Health Service, Centers for Disease Control, and several national physicians' organizations have recently begun a campaign to promote adult immunizations. They have issued guidelines so that physicians and their patients will know which vaccines should be given to ***which*** adults and ***when.***

In order to help me decide which "shots" you may need for your health maintenance, you should provide me with the following information to be added to your personal medical record:

- ☐ history of all previous immunizations (type and date if known)
- ☐ history of any bad reactions (if any) to any previous immunizations
- ☐ history of any vaccine-preventable diseases such as measles, mumps, or rubella
- ☐ occupational risk such as health care worker, laboratory worker, animal handler, sewage worker, or gardener
- ☐ foreign traveler
- ☐ foreign student
- ☐ lifestyle risk such as homosexuality or illicit drug use

IMMUNIZATIONS RECOMMENDED FOR HEALTHY ADULTS

AGE	18-30	31-64	65+
Diph.-Tetanus	×	×	×
Measles*	×		
Mumps*	×	×	
Rubella*	×	×	
Flu			×
Pneumovax			×

*ONLY FOR ADULTS WITH NO HISTORY OF VACCINE AND/OR NO HISTORY OF THE DISEASE EARLIER IN LIFE

- ☐ Diph.-Tetanus "booster" given every 10 years
- ☐ Measles, Mumps, and Rubella given only once in lifetime
- ☐ Flu vaccine given every year
- ☐ Pneumovax given only once in lifetime

Now is the right time to ask me which immunizations you will need to stay well. I can tell you what's best for you at this time and what you may need if you plan to travel overseas.

MEDICAL SOCIETY OF THE STATE OF NEW YORK
ORGANIZED 1807

Office Handouts for Your Patients

This issue of the *New York State Journal of Medicine* contains another "For My Patients" letter, which appears on the preceding page.

Use this letter series to keep your patients informed about timely issues concerning diseases, treatments and health maintenance. Simply make photocopies of the page and distribute them to your patients with your newsletter or other correspondence, or place them in your reception area for your patients to read and take home.

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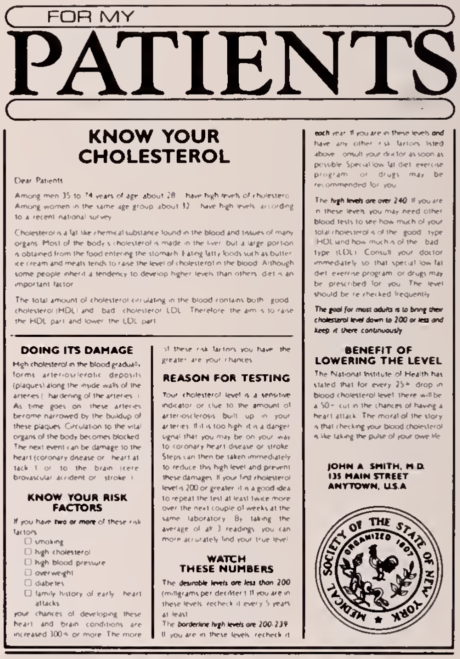

FOR MY
PATIENTS

KNOW YOUR CHOLESTEROL

Dear Patients

Among men 35 to 74 years of age about 28% have high levels of cholesterol. Among women in the same age group about 12% have high levels according to a recent national survey.

Cholesterol is a fat like chemical substance found in the blood and tissues of many organs. Most of the body's cholesterol is made in the liver but a large portion is obtained from the food entering the stomach. Eating fatty foods such as butter, ice cream and meats tends to raise the level of cholesterol in the blood. Although some people inherit a tendency to develop higher levels than others, diet is an important factor.

The total amount of cholesterol circulating in the blood contains both "good" cholesterol (HDL) and "bad" cholesterol (LDL). Therefore the aim is to raise the HDL part and lower the LDL part.

DOING ITS DAMAGE

High cholesterol in the blood gradually forms arteriosclerotic deposits (plaques) along the inside walls of the arteries ("hardening of the arteries"). As time goes on, these arteries become narrowed by the buildup of these plaques. Circulation to the vital organs of the body becomes blocked. The next event can be damage to the heart (coronary disease or "heart attack") or to the brain (cerebrovascular accident or "stroke").

KNOW YOUR RISK FACTORS

If you have **two or more** of these risk factors

- ☐ smoking
- ☐ high cholesterol
- ☐ high blood pressure
- ☐ overweight
- ☐ diabetes
- ☐ family history of early heart attacks

your chances of developing these heart and brain conditions are increased 300% or more. The more of these risk factors you have, the greater are your chances.

REASON FOR TESTING

Your cholesterol level is a sensitive indicator or clue to the amount of arteriosclerosis built up in your arteries. If it is too high, it is a danger signal that you may be on your way to coronary heart disease or stroke. Steps can then be taken immediately to reduce this high level and prevent these damages. If your first cholesterol level is 200 or greater, it is a good idea to repeat the test at least twice more over the next couple of weeks at the same laboratory. By taking the average of all 3 readings, you can more accurately find your true level.

WATCH THESE NUMBERS

The *desirable levels are less than 200* (milligrams per deciliter). If you are in these levels, recheck it every 5 years at least.

The *borderline high levels are 200-239*. If you are in these levels, recheck it *each* year. If you are in these levels *and* have any other risk factors listed above, consult your doctor as soon as possible. Special low fat diet, exercise program or drugs may be recommended for you.

The *high levels are over 240*. If you are in these levels, you may need other blood tests to see how much of your total cholesterol is of the "good" type (HDL) and how much is of the "bad" type (LDL). Consult your doctor immediately so that special low fat diet, exercise program or drugs may be prescribed for you. The level should be re-checked frequently.

The goal for most adults is to bring their cholesterol level down to 200 or less and keep it there continuously.

BENEFIT OF LOWERING THE LEVEL

The National Institute of Health has stated that for every 25% drop in blood cholesterol level there will be a 50% cut in the chances of having a heart attack. The moral of the story is that checking your blood cholesterol is like taking the pulse of your own life.

JOHN A. SMITH, M.D.
135 MAIN STREET
ANYTOWN, U.S.A.

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CONTRAINDICATIONS: PARAFON FORTE DSC chlorzoxazone is contraindicated in patients with known intolerance to the drug.
WARNINGS: The concomitant use of alcohol or other central nervous system depressants may have an additive effect. *Usage in Pregnancy.* The safe use of PARAFON FORTE DSC chlorzoxazone has not been established with respect to the possible adverse effects upon fetal development. Therefore, it should be used in women of childbearing potential only when, in the judgment of the physician, the potential benefits outweigh the possible risks.
PRECAUTIONS: PARAFON FORTE DSC chlorzoxazone should be used with caution in patients with known allergies or with a history of allergic reactions to drugs. If a sensitivity reaction occurs such as urticaria, redness, or itching of the skin, the drug should be stopped. If any symptoms suggestive of liver dysfunction are observed, the drug should be discontinued.
ADVERSE REACTIONS: After more than twenty-six years of extensive clinical use of chlorzoxazone-containing products in an estimated thirty-two million patients, it is apparent that the drug is well tolerated and seldom produces undesirable side effects. Occasional patients may develop gastrointestinal disturbances. It is possible in rare instances that chlorzoxazone may have been associated with gastrointestinal bleeding. Drowsiness, dizziness, lightheadedness, malaise, or overstimulation may be noted by an occasional patient. Rarely allergic-type skin rashes, petechiae, or ecchymoses may develop during treatment. Angioneurotic edema or anaphylactic reactions are extremely rare. There is no evidence that the drug will cause renal damage. Rarely, a patient may note discoloration of the urine resulting from a phenolic metabolite of chlorzoxazone. This finding is of no known clinical significance. Approximately thirty-six patients have been reported in whom the administration of chlorzoxazone-containing products was suspected as being the cause of liver damage. In one case, the jaundice was subsequently considered to be due to a carcinoma of the head of the pancreas rather than to the drug. In a second case, there was no jaundice but an elevated alkaline phosphatase and BSP retention. In this patient there was a malignancy with bony and liver metastases. The role of the drug was difficult to determine. A third and fourth case had cholelithiasis. Diagnosis in a fifth case was submassive hepatic necrosis possibly due to abusive use of the drug for approximately one year. The remaining cases had a clinical picture compatible with either a viral hepatitis or a drug-induced hepatitis. In all these latter cases, the drug was stopped, and with one exception, the patients recovered. It is not possible to state that the hepatitis in these patients was or was not drug-induced.
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For information on symptoms and treatment of overdosage, see full prescribing information.

12/16/87

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MAY 15–19, 1989: CONSULTANT'S COURSE IN CARDIOLOGY. The Waldorf Astoria, New York, NY. Sponsored by the American College of Cardiology. Inquiries: Registration Secretary, Extramural Programs Department, American College of Cardiology, 9111 Georgetown, Bethesda, MD 20814. Tel. (800) 253-4636; in Maryland (301) 897-5400, ext. 228.

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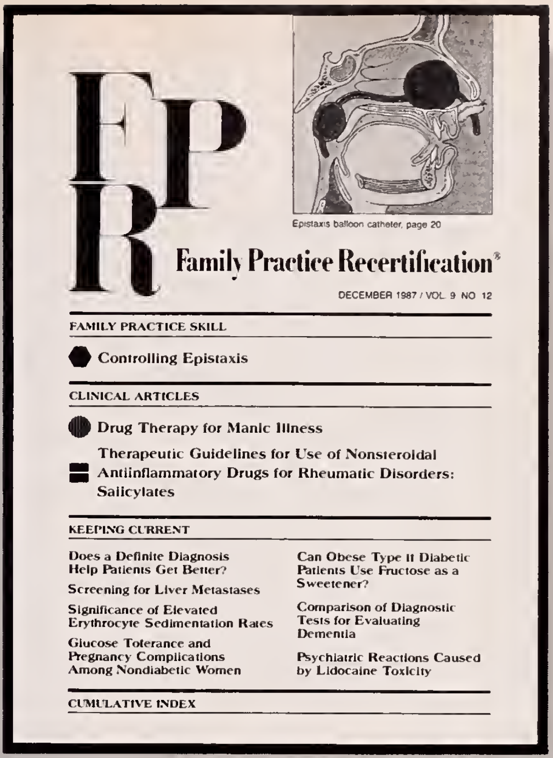

FPR

Epistaxis balloon catheter, page 20

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Drug Therapy for Manic Illness

Therapeutic Guidelines for Use of Nonsteroidal Antiinflammatory Drugs for Rheumatic Disorders: Salicylates

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June 17, 1989

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Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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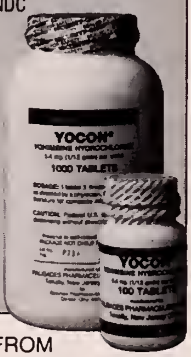

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ciate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

May 19. **Clinical Toxicology. Industrial/Occupational Emergencies.** NYC Poison Control Center. Contact: Sue Callaghan, RN, MA, Bellevue Hospital Center. Tel: (212) 561-6561.

May 24. **Practical Diagnosis and Management of Metabolic Bone Diseases.** Columbia University. Contact: Center for Continuing Education, College of Physicians & Surgeons of Columbia University, 630 W 168th St, New York, NY 10032. Tel: (212) 305-3682.

May 31–June 2. **Micronutrient Effects on Immune Function.** Sheraton Centre. Contact: Conference Director, The New York Academy of Sciences, 2 E 63rd St, New York, NY 10021.

AROUND THE NATION

CALIFORNIA

May 25–26. **Fifth Annual Current Issues in Anatomic Pathology: 1989.** Holiday Inn Union Square, San Francisco. Contact: Extended Programs in Medical Education, University of California, Room U-569, San Francisco, CA 94143-0742. Tel: (415) 476-4251.

DISTRICT OF COLUMBIA

May 1–3. **Conference on Emerging Viruses: Evolution of Viruses and Viral Diseases.** The Hotel Washington. Contact: Dr Stephen S. Morse, The Rockefeller University, 1230 York Ave, New York, NY 10021-6399. Tel: (212) 570-7722.

May 8–9. **Seventh Annual Conference on Prescription Medicine Information and Education.** Capitol Hilton Hotel. Contact: National Council on Patient Information and Education, 666 11th St, #810, Washington, DC 20001. Tel: (202) 347-6711.

May 24–27. **Second International Conference on Drug Research in Immunologic and Infectious Diseases: Acquired Immune Deficiency Syndrome (AIDS).** Hyatt Capitol Hill. Contact: Confer-

(*continued on p 26A*)

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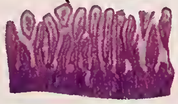
Aspirin and other nonsteroidal anti-inflammatory drugs weaken mucosal defenses, which may lead NSAID users to become prone to duodenal ulcers.[1] For those NSAID users who do develop duodenal ulcers, CARAFATE® (sucralfate/Marion) is ideal first-line therapy. Carafate rebuilds mucosal  defenses through a unique, nonsystemic mode of action. Carafate enhances the body's natural healing ability while it protects damaged mucosa from further injury. So the next time you see an arthritis patient with a duodenal ulcer, prescribe nonsystemic Carafate: therapy for the ulcer-prone patient.

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BRIEF SUMMARY

CONTRAINDICATIONS
There are no known contraindications to the use of sucralfate

PRECAUTIONS
Duodenal ulcer is a chronic, recurrent disease. While short-term treatment with sucralfate can result in complete healing of the ulcer, a successful course of treatment with sucralfate should not be expected to alter the post-healing frequency or severity of duodenal ulceration.

Drug Interactions: Animal studies have shown that simultaneous administration of CARAFATE (sucralfate) with tetracycline, phenytoin, digoxin, or cimetidine will result in a statistically significant reduction in the bioavailability of these agents. The bioavailability of these agents may be restored simply by separating the administration of these agents from that of CARAFATE by two hours. This interaction appears to be nonsystemic in origin, presumably resulting from these agents being bound by CARAFATE in the gastrointestinal tract. The clinical significance of these animal studies is yet to be defined. However, because of the potential of CARAFATE to alter the absorption of some drugs from the gastrointestinal tract, the separate administration of CARAFATE from that of other agents should be considered when alterations in bioavailability are felt to be critical for concomitantly administered drugs.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Chronic oral toxicity studies of 24 months' duration were conducted in mice and rats at doses up to 1 gm/kg (12 times the human dose). There was no evidence of drug-related tumorgenicity. A reproduction study in rats at doses up to 38 times the human dose did not reveal any indication of fertility impairment. Mutagenicity studies were not conducted.

Pregnancy: Teratogenic effects. Pregnancy Category B. Teratogenicity studies have been performed in mice, rats, and rabbits at doses up to 50 times the human dose and have revealed no evidence of harm to the fetus due to sucralfate. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when sucralfate is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS
Adverse reactions to sucralfate in clinical trials were minor and only rarely led to discontinuation of the drug. In studies involving over 2,500 patients treated with sucralfate, adverse effects were reported in 121 (4.7%).

Constipation was the most frequent complaint (2.2%). Other adverse effects, reported in no more than one of every 350 patients, were diarrhea, nausea, gastric discomfort, indigestion, dry mouth, rash, pruritus, back pain, dizziness, sleepiness, and vertigo.

OVERDOSAGE
There is no experience in humans with overdosage. Acute oral toxicity studies in animals, however, using doses up to 12 gm/kg body weight, could not find a lethal dose. Risks associated with overdosage should, therefore, be minimal.

DOSAGE AND ADMINISTRATION
The recommended adult oral dosage for duodenal ulcer is 1 gm four times a day on an empty stomach.

Antacids may be prescribed as needed for relief of pain but should not be taken within one-half hour before or after sucralfate.

While healing with sucralfate may occur during the first week or two, treatment should be continued for 4 to 8 weeks unless healing has been demonstrated by x-ray or endoscopic examination.

HOW SUPPLIED
CARAFATE (sucralfate) 1-gm tablets are supplied in bottles of 100 (NDC 0088-1712-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1712-49). Light pink scored oblong tablets are embossed with CARAFATE on one side and 1712 bracketed by C's on the other.
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Reference:
1. Eliakim R, Ophir M, Rachmilewitz D: *J Clin Gastroenterol* 1987; 9(4):395-399

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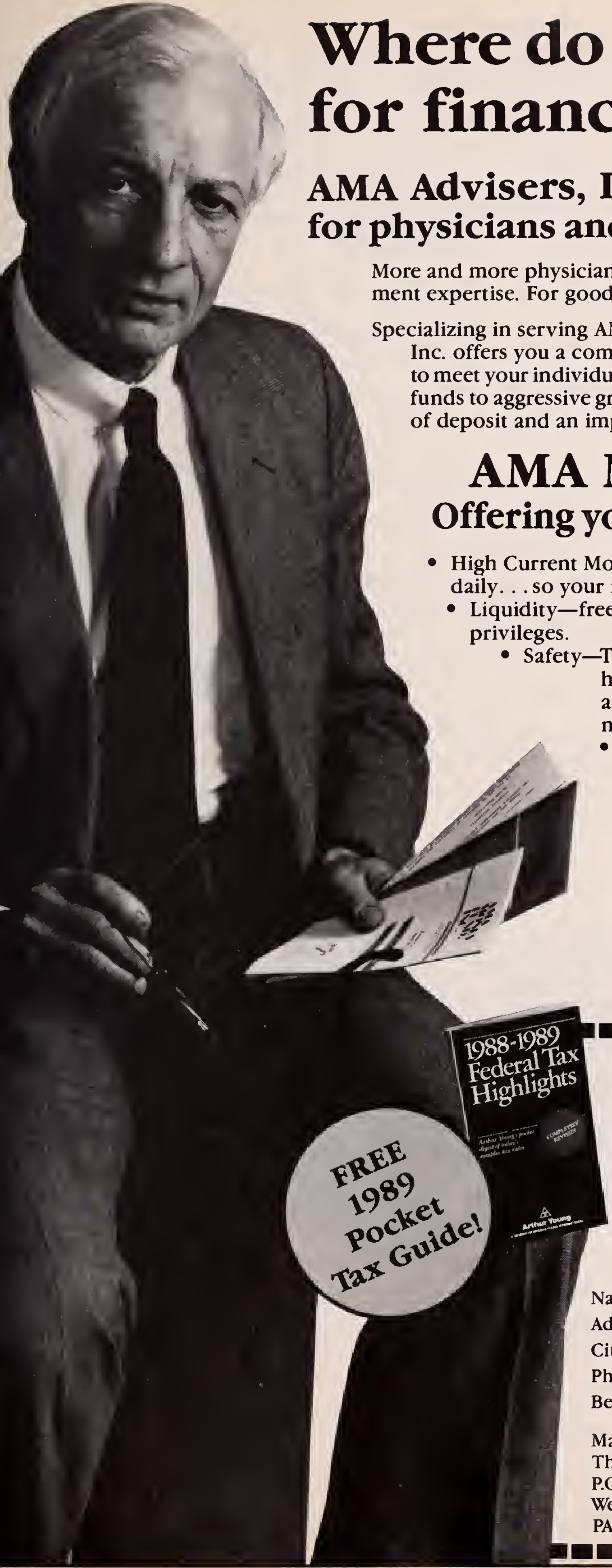

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(*continued from p 22A*)

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May 18–20. **Advances in Pediatrics.** 16 Cat I Credits. Mariner's Inn, Hilton Head Island. Contact: CME Registration, American Academy of Pediatrics, PO Box 927, Elk Grove Village, IL 60009-0927.

VIRGINIA

May 4–6. **Embryonic Origins of Defective Heart Development.** Key Bridge Marriott, Arlington. *May 18–20.* **Transforming Growth Factors-BETA: Chemistry, Biology and Therapeutics.** Key Bridge Marriott, Arlington. Contact: Conference Director, The New York Academy of Sciences, 2 E 63rd St, New York, NY 10021.

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BERMUDA

May 25–29. **Neuro-Ophthalmology Conference.** 18 Cat I Credits. Southampton Princess. Contact: Ann J. Boehme, CMP, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

COLOMBIA

May 2–5. **IV Pan American Congress of Epilepsy.** Cartagena. Contact: Dr Carlos Medina-Malo, Calle 77 No. 15, 80 Of. 406, Bogota, Colombia.

JAPAN

May 23–26. **Sixth International Conference on Human Tumor Markers.** Tokyo. Contact: IATMO Tokyo '89, c/o Kinki Nippon Tourist Col, Ltd, Inbound Sales Office Tokyo Kintetsu Bldg, 2F 19-2, Kanda Matsunaga-cho, Chiyods-ku, Tokyo, 101, Japan.

WEST INDIES

May 3–7. **Sixth Annual Meeting of the Southern Orthopaedic Association.** 12 Cat I Credits. Royal Antiguan Hotel, Antigua. Contact: Education Department, Southern Medical Association, 35 Lakeshore Dr, Birmingham, AL 35219-0088. Tel: (205) 945-1840. □

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Index to Advertisers

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Classified Advertising 4A, 12A, 14A, 20A, 21A, 24A
Cliggott Publishing 11A
Family Practice Recertification Magazine 21A
Eli Lilly and Company .... 2nd Cover
Marine Midland Bank 15A
Marion Laboratories 1A, 2A, 23A, 24A
McGraw Hill Publications 8A
Merck, Sharp and Dohme 9A, 10A
McNeil Pharmaceuticals 19A, 20A
New York Beef Industry Council 7A
Palisades Pharmaceuticals 22A
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Contents

COMMENTARIES

The enigma of extrapulmonary tuberculosis 251
MATTHEW R. WEIR, MD

Abdominal tuberculosis: A disease poised for reappearance 252
JAY J. ROHWEDDER, MD

Drug use in pregnancy 255
IRA J. CHASNOFF, MD

RESEARCH PAPERS

Birth outcomes for infants of drug abusing mothers 256
KATHERINE KAYE, MD, MPH; LISA ELKIND, MS; DORIS GOLDBERG, MD, MPH; ALEX TYTUN, DrPH

Wood burning related injuries 262
JAMES J. NICHOLSON, MD, PATRICK A. DIETZ, MD

CLINICAL CONFERENCE

Transfusion reactions 264

INTERNATIONAL HEALTH

Health and health care in Nepal 272
SUSAN L. PURETZ, EdD

HISTORY

Trends in American gynecology, 1800–1910: A short history 278
JOHN S. HALLER, JR, PHD

SPECIAL ARTICLE

Asthma in Timisoara: Diary of a glimpse at the Romanian medical system 283
RICHARD SELTZER

CASE REPORTS

Distal colonic tuberculosis with tuberculous enteritis, peritonitis, and pericarditis 286
JOSEPH C. SCIAMMARELLA, JR, MD, ALEX H. BRUCKSTEIN, MD

Mammary tuberculosis 288
ALEXANDER A. McMEEKING, MD; RAIMUNDO GONZALEZ, MD; BRUCE HANNA, PhD

Congenital splenic cysts presenting after blunt abdominal trauma 289
STEVEN STYLIANOS, MD, THOMAS V. SANTULLI, MD

Malignant melanoma of the anal canal 291
PAUL Y. LIN, MD; JAMES SMITH-BEHN, MD; LUDWIG M. DEPPISCH, MD

LETTERS TO THE EDITOR

Paget-Schroetter syndrome in a patient on the contraceptive pill 293
K. A. EARLE, MB, BS, M. H. LLOYD, MB

Professional medical conduct 293
KATHLEEN TANNER

Legislator-physician relationships 294
RANDALL D. BLOOMFIELD, MD

The *New York Medical Journal* and cigarette smoking 294
ZIRA DeFRIES, MD

ESSAY REVIEW

The wickedest woman in New York 296
NAOMI R. BLUESTONE, MD

LEADS FROM EPIDEMIOLOGY NOTES 298

BOOK REVIEWS 302

NEWS BRIEFS 308

OBITUARIES 310

GUIDELINES FOR AUTHORS 312

MEDICAL MEETINGS AND LECTURES 6A

Annual CME Assembly, September 15–16, 1989, New York Hilton, New York City
(See July issue for Preliminary Program)

ARMY RESERVE MEDICAL PROFILE NO.3

DALE L. TIPTON, M.D.

Associate Clinical Professor, Department of Otolaryngology, Head and Neck Surgery, University of California School of Medicine, San Francisco, California.
Chairman, Division of Otolaryngology, Franklin Hospital, San Francisco, California.
Lieutenant Colonel, U.S. Army Reserve.

EDUCATION University of California at Berkeley, A.B. Physiology; University of California School of Medicine, San Francisco, M.D. and Master of Science, Pharmacology.

RESIDENCY University of California School of Medicine, San Francisco: General Surgery—2 years; Otolaryngology—3 years.

FELLOWSHIPS National Institute of Health Fellow; Cancer Research Institute, University of California, San Francisco.

OUTSTANDING ACHIEVEMENTS Freshman Medical Student Research Award; Class President—2nd year medical school; Student Body President—senior year medical school; Special Award by National Institute of Health to attend and present paper at International Congress of Otolaryngology in Tokyo, Japan; Chairman, Department of Otolaryngology, San Francisco General Hospital 1970-76; Chief of Medical Staff, Franklin Hospital 1982-84.

Dr. Tipton and residents examining post-operative patient in recovery room.

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CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, (3) patients with hypotension (less than 90 mm Hg systolic), (4) patients who have demonstrated hypersensitivity to the drug, and (5) patients with acute myocardial infarction and pulmonary congestion documented by x-ray on admission.

WARNINGS

1. **Cardiac Conduction.** CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (nine of 2,111 patients or 0.43%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure.** Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt). An acute study of oral diltiazem in patients with impaired ventricular function (ejection fraction 24% ± 6%) showed improvement in indices of ventricular function without significant decrease in contractile function (dp/dt). Experience with the use of CARDIZEM (diltiazem hydrochloride) in combination with beta-blockers in patients with impaired ventricular function is limited Caution should be exercised when using this combination.
3. **Hypotension.** Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury.** Mild elevations of transaminases with and without concomitant elevation in alkaline phosphatase and bilirubin have been observed in clinical studies Such elevations were usually transient and frequently resolved even with continued diltiazem treatment. In rare instances, significant elevations in enzymes such as alkaline phosphatase, LDH, SGOT, SGPT, and other phenomena consistent with acute hepatic injury have been noted. These reactions tended to occur early after therapy initiation (1 to 8 weeks) and have been reversible upon discontinuation of drug therapy. The relationship to CARDIZEM is uncertain in some cases, but probable in some. (See PRECAUTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile As with any drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued In dogs, doses of 20 mg/kg were also associated with hepatic changes, however, these changes were reversible with continued dosing.

Dermatological events (see ADVERSE REACTIONS section) may be transient and may disappear despite continued use of CARDIZEM. However, skin eruptions progressing to erythema multiforme and/or exfoliative dermatitis have also been infrequently reported. Should a dermatologic reaction persist, the drug should be discontinued.

Drug Interaction. Due to the potential for additive effects, caution and careful titration are warranted in patients receiving CARDIZEM concomitantly with any agents known to affect cardiac contractility and/or conduction. (See WARNINGS.) Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM (See WARNINGS.)

As with all drugs, care should be exercised when treating patients with multiple medications. CARDIZEM undergoes biotransformation by cytochrome P-450 mixed function oxidase. Coadministration of CARDIZEM with other agents which follow the same route of biotransformation may result in the competitive inhibition of metabolism. Dosages of similarly metabolized drugs, particularly those of low therapeutic ratio or in patients with renal and/or hepatic impairment, may require adjustment when starting or stopping concomitantly administered CARDIZEM to maintain optimum therapeutic blood levels.

Beta-blockers: Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated, but available data are not sufficient to predict the effects of concomitant treatment in patients with left ventricular dysfunction or cardiac conduction abnormalities.

Administration of CARDIZEM (diltiazem hydrochloride) concomitantly with propranolol in five normal volunteers resulted in increased propranolol levels in all subjects and bioavailability of propranolol was increased approximately 50% If combination therapy is initiated or withdrawn in conjunction with propranolol, an adjustment in the propranolol dose may be warranted (See WARNINGS.)

Cimetidine: A study in six healthy volunteers has shown a significant increase in peak diltiazem plasma levels (58%) and area-under-the-curve (53%) after a 1-week course of cimetidine at 1,200 mg per day and diltiazem 60 mg per day Ranitidine produced smaller, nonsignificant increases The effect may be mediated by cimetidine's known inhibition of hepatic cytochrome P-450, the enzyme system probably responsible for the first-pass metabolism of diltiazem. Patients currently receiving diltiazem therapy should be carefully monitored for a change in pharmacological effect when initiating and discontinuing therapy with cimetidine An adjustment in the diltiazem dose may be warranted

Digitalis: Administration of CARDIZEM with digoxin in 24 healthy male subjects increased plasma digoxin concentrations approximately 20%. Another investigator found no increase in digoxin levels in 12 patients with coronary artery disease Since there have been conflicting results regarding the effect of digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing CARDIZEM therapy to avoid possible over- or under-digitalization. (See WARNINGS.)

Anesthetics: The depression of cardiac contractility, conductivity, and automaticity as well as the vascular dilation associated with anesthetics may be potentiated by calcium channel blockers. When used concomitantly, anesthetics and calcium blockers should be titrated carefully

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests No intrinsic effect on fertility was observed in rats.

Pregnancy. Category C Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. Diltiazem is excreted in human milk One report suggests that concentrations in breast milk may approximate serum levels. If use of CARDIZEM is deemed essential, an alternative method of infant feeding should be instituted

Pediatric Use. Safety and effectiveness in children have not been established

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded from these studies.

The adverse events described below represent events observed in clinical studies of hypertensive patients receiving either CARDIZEM Tablets or CARDIZEM SR Capsules as well as experiences observed in studies of angina and during marketing The most common events in hypertension studies are shown in a table with rates in placebo patients shown for comparison. Less common events are listed by body system, these include any adverse reactions seen in angina studies that were not observed in hypertension studies In all hypertensive patients studied (over 900), the most common adverse events were edema (9%), headache (8%), dizziness (6%), asthenia (5%), sinus bradycardia (3%), flushing (3%), and 1° AV block (3%). Only edema and perhaps bradycardia and dizziness were dose related. The most common events observed in clinical studies (over 2,100 patients) of angina patients and hypertensive patients receiving CARDIZEM Tablets or CARDIZEM SR Capsules were (ie. greater than 1%) edema (5.4%), headache (4.5%), dizziness (3.4%), asthenia (2.8%), first-degree AV block (1.8%), flushing (1.7%), nausea (1.6%), bradycardia (1.5%), and rash (1.5%).

DOUBLE BLIND PLACEBO CONTROLLED HYPERTENSION TRIALS

Adverse	Diltiazem N=315 # pts (%)	Placebo N=211 # pts (%)
headache	38 (12%)	17 (8%)
AV block first degree	24 (7.6%)	4 (1.9%)
dizziness	22 (7%)	6 (2.8%)
edema	19 (6%)	2 (0.9%)
bradycardia	19 (6%)	3 (1.4%)
ECG abnormality	13 (4.1%)	3 (1.4%)
asthenia	10 (3.2%)	1 (0.5%)
constipation	5 (1.6%)	2 (0.9%)
dyspepsia	4 (1.3%)	1 (0.5%)
nausea	4 (1.3%)	2 (0.9%)
palpitations	4 (1.3%)	2 (0.9%)
polyuria	4 (1.3%)	2 (0.9%)
somnolence	4 (1.3%)	—
alk phos increase	3 (1%)	1 (0.5%)
hypotension	3 (1%)	1 (0.5%)
insomnia	3 (1%)	1 (0.5%)
rash	3 (1%)	1 (0.5%)
AV block second degree	2 (0.6%)	—

In addition, the following events were reported infrequently (less than 1%) or have been observed in angina trials. In many cases, the relation to drug is uncertain.

Cardiovascular:	Angina, arrhythmia, bundle branch block, tachycardia, ventricular extrasystoles, congestive heart failure, syncope.
Nervous System:	Amnesia, depression, gait abnormality, hallucinations, nervousness, paresthesia, personality change, tinnitus, tremor, abnormal dreams
Gastrointestinal:	Anorexia, diarrhea, dysgeusia, mild elevations of SGOT, SGPT, and LDH (see hepatic warnings), vomiting, weight increase, thirst.
Dermatological:	Petechiae, pruritus, photosensitivity, urticaria.
Other:	Amblyopia, CPK increase, dyspnea, epistaxis, eye irritation, hyperglycemia, sexual difficulties, nasal congestion, nocturia, osteoarticular pain, impotence, dry mouth.

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM. alopecia, gingival hyperplasia, erythema multiforme, and leukopenia Definitive cause and effect relationship between these events and CARDIZEM therapy cannot yet be established.

Issued 1/89

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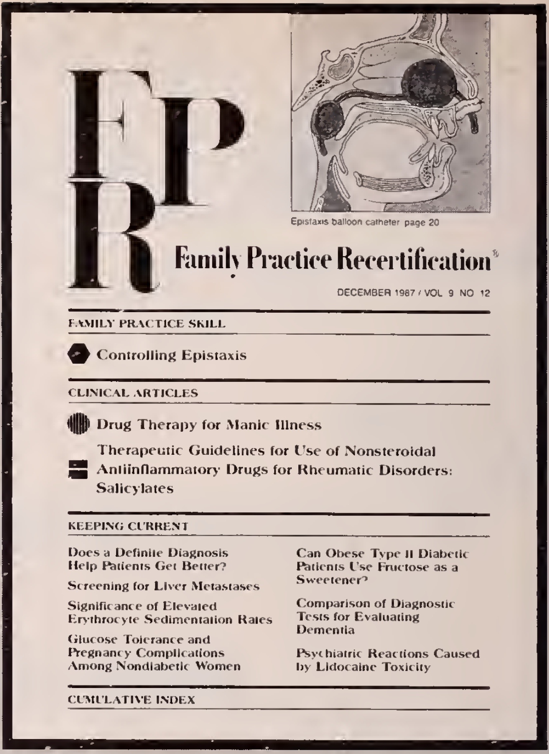

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Epistaxis balloon catheter page 20

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Controlling Epistaxis

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Drug Therapy for Manic Illness

Therapeutic Guidelines for Use of Nonsteroidal Antiinflammatory Drugs for Rheumatic Disorders: Salicylates

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Does a Definite Diagnosis Help Patients Get Better?

Screening for Liver Metastases

Significance of Elevated Erythrocyte Sedimentation Rates

Glucose Tolerance and Pregnancy Complications Among Nondiabetic Women

Can Obese Type II Diabetic Patients Use Fructose as a Sweetener?

Comparison of Diagnostic Tests for Evaluating Dementia

Psychiatric Reactions Caused by Lidocaine Toxicity

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PRACTICAL ▪ CLINICAL ▪ EDUCATIONAL ▪ CURRENT

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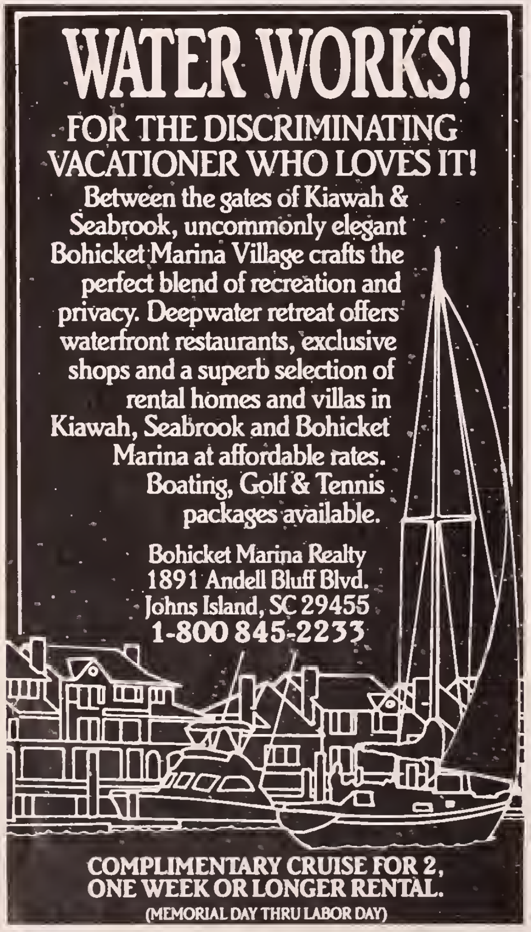

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June 4–17. **Methods of Immunologic Research and Diagnosis.** Ernest Witebsky Center for Immunology. Contact: James F. Mohn, MD, Director, The Ernest Witebsky Center for Immunology, 317 Sherman Hall, Buffalo, NY 14214. Tel: (716) 831-2848.

GARDEN CITY

June 7. **A Clinical Day in Ophthalmology: Fluorescein Angiography: Basic and Applied.** 7 Cat I Credits. Garden City Hotel. Contact: Ann J. Boehme, CMP, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042.

MANHATTAN

June 7–10. **Comprehensive Seminar on Fine Needle Aspiration: A Review & Update.** 32 Cat I Credits. *June 9–10.* **Sixth Annual Seminar in Obstetrical and Gynecological Ultrasound.** *June 11.* **Practical Training in the Technique of Fine Needle Aspiration of the Thyroid Gland.** 8 Cat I Credits. *June 12–16.* **Emergency Medicine 1989.** 28 ½ Cat I Credits. NYU Medical Center. *June 19–22.* **Selected Topics in Pediatrics.** 22 Cat I Credits. *June 23–24.* **Manifestations and Management of HIV Infection for Primary Care Physicians.** 10 ½ Cat I Credits. NYU Medical Center. Contact: NYU Medical Center Post-Graduate Medical Center, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

June 9. **Management of Stroke: Medical, Neurological and Rehabilitation Aspects.** Beth Israel Medical Center. Contact: Beverley R. Baptiste, Medical Education, Beth Israel Medical Center, First Ave at 16th St, New York, NY 10003. Tel: (212) 420-2849.

June 12–16. **Update Your Medicine 1989.** 33 ½ Cat I Credits. New York Hospital-Cornell. Contact: Office of CME, New York Hospital, 1300 York Ave, New York, NY 10021. Tel: (212) 746-4752.

June 19–23. **The New York Anesthesiology Review.** 31 Cat I Credits. Omni Park Central. Contact: Office of Continuing Medical Education, Albert Einstein College of Medicine, Montefiore Medical Center, 3301 Bainbridge Ave, Bronx, NY 10467. Tel: (212) 920-6674.

SARATOGA SPRINGS

June 3–4. **Ophthalmology Clinical Update 1989.** Gideon Putnam Hotel. Contact: Diane M. Martin, Office of CME, Albany Medical College, 47 New Scotland Ave, Albany, NY 12208. Tel: (518) 445-5828.

SOUTHAMPTON

June 12. **Diagnosis and Treatment of Head and Neck Abscesses.** 1 Cat I Credit. Southampton Hospital, Southampton. Contact: Charlotte Henry-Sullivan, Southampton Hospital, 240 Meeting House Lane, Southampton, NY 11968. Tel: (516) 283-2600.

AROUND THE NATION

CALIFORNIA

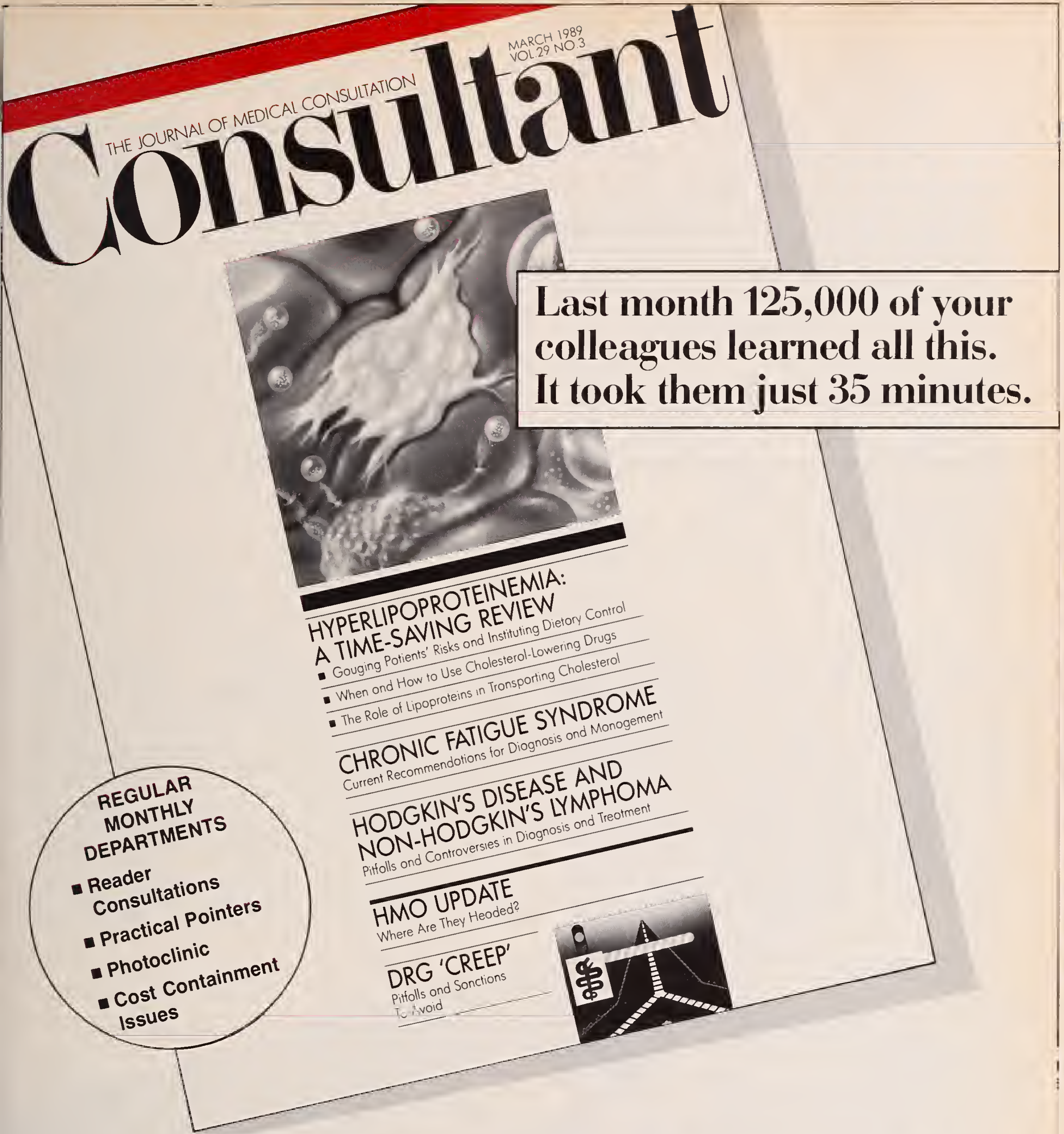
June 8–12. **18th Annual Scientific Assembly.** Rancho Bernardo Inn, Rancho Bernardo. Contact: American College of Emergency Physicians, 505 N Sepulveda Blvd, Suite 12, Manhattan Beach, CA 90266. Tel: (213) 374-4039.

June 15–17. **Second World Congress and Vaginosonography in Gynecology.** LeMeridien Hotel, San Francisco. Contact: American Association of Gynecologic Laparoscopists, 13021 E Florence Ave, Santa Fe Springs, CA 90670. Tel: (213) 946-8774.

June 22–24. **Basic and Advanced Colposcopy.** Fairmont Hotel, San Francisco. Contact: American College of Obstetricians and Gynecologists, 409 12th St, SW, Washington, DC 20024-2188.

June 24–25. **11th Annual Poison Center**

(*continued on p 16A*)



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How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

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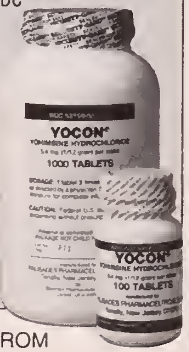

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Precautions: *General* – 1. Symptomatic response to nizatidine therapy does not preclude the presence of gastric malignancy

2. Because nizatidine is excreted primarily by the kidney, dosage should be reduced in patients with moderate to severe renal insufficiency

3. Pharmacokinetic studies in patients with hepatorenal syndrome have not been done. Part of the dose of nizatidine is metabolized in the liver. In patients with normal renal function and uncomplicated hepatic dysfunction, the disposition of nizatidine is similar to that in normal subjects.

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Drug Interactions – No interactions have been observed between Axid and theophylline, chlordiazepoxide, lorazepam, lidocaine, phenytoin, and warfarin. Axid does not inhibit the cytochrome P-450-linked drug-metabolizing enzyme system; therefore, drug interactions mediated by inhibition of hepatic metabolism are not expected to occur. In patients given very high doses (3,900 mg) of aspirin daily, increases in serum salicylate levels were seen when nizatidine, 150 mg b.i.d., was administered concurrently

Carcinogenesis, Mutagenesis, Impairment of Fertility – A two-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a two-year study in mice, there was no evidence of a carcinogenic effect in male mice; although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups. The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, mouse lymphoma assay, chromosome aberration tests, and a micronucleus test.

In a two-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy – Teratogenic Effects – Pregnancy Category C – Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in one fetus and at 50 mg/kg it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in one fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers – Studies conducted in lactating women have shown that <0.1% of the administered oral dose of nizatidine is secreted in human milk in proportion to plasma concentrations Caution should be exercised when administering nizatidine to a nursing mother.

Pediatric Use – Safety and effectiveness in children have not been established.

Use in Elderly Patients – Ulcer healing rates in elderly patients are similar to those in younger age groups. The incidence rates of adverse events and laboratory test abnormalities are also similar to those seen in other age groups. Age alone may not be an important factor in the disposition of nizatidine Elderly patients may have reduced renal function.

Adverse Reactions: Clinical trials of nizatidine included almost 5,000 patients given nizatidine in studies of varying durations Domestic placebo-controlled trials included over 1,900 patients given nizatidine and over 1,300 given placebo. Among reported adverse events in the domestic placebo-controlled trials, sweating (1% vs 0.2%), urticaria (0.5% vs < 0.01%), and somnolence (2.4% vs 1.3%) were significantly more common in the nizatidine group A variety of less common events was also reported; it was not possible to determine whether these were caused by nizatidine

Hepatic – Hepatocellular injury, evidenced by elevated liver enzyme tests (SGOT [AST], SGPT [ALT], or alkaline phosphatase), occurred in some patients and was possibly or probably related to nizatidine In some cases, there was marked elevation of SGOT, SGPT enzymes (greater than 500 IU/L) and, in a single instance, SGPT was greater than 2,000 IU/L. The overall rate of occurrences of elevated liver enzymes and elevations to three times the upper limit of normal, however, did not significantly differ from the rate of liver enzyme abnormalities in placebo-treated patients. All abnormalities were reversible after discontinuation of Axid.

Cardiovascular – In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in two individuals administered Axid and in three untreated subjects

CNS – Rare cases of reversible mental confusion have been reported.

Endocrine – Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to Axid. Impotence and decreased libido were reported with equal frequency by patients who received Axid and by those given placebo. Rare reports of gynecomastia occurred.

Hematologic – Fatal thrombocytopenia was reported in a patient who was treated with Axid and another H_2-receptor antagonist. On previous occasions, this patient had experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported

Integumental – Sweating and urticaria were reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported

Hypersensitivity – As with other H_2-receptor antagonists, rare cases of anaphylaxis following administration of nizatidine have been reported. Because cross-sensitivity in this class of compounds has been observed, H_2-receptor antagonists should not be administered to individuals with a history of previous hypersensitivity to these agents Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported

Other – Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine administration have been reported.

Overdosage: Overdoses of Axid have been reported rarely The following is provided to serve as a guide should such an overdose be encountered

Signs and Symptoms – There is little clinical experience with overdosage of Axid in humans Test animals that received large doses of nizatidine have exhibited cholinergic-type effects, including lacrimation, salivation, emesis, miosis, and diarrhea. Single oral doses of 800 mg/kg in dogs and of 1,200 mg/kg in monkeys were not lethal Intravenous median lethal doses in the rat and mouse were 301 mg/kg and 232 mg/kg respectively

Treatment – To obtain up-to-date information about the treatment of overdose, a good resource is your certified regional Poison Control Center. Telephone numbers of certified poison control centers are listed in the *Physicians' Desk Reference (PDR)* In managing overdosage, consider the possibility of multiple drug overdoses, interaction among drugs, and unusual drug kinetics in your patient.

If overdosage occurs, use of activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. Renal dialysis for four to six hours increased plasma clearance

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Additional information available to the profession on request.

1. Data on file, Lilly Research Laboratories.

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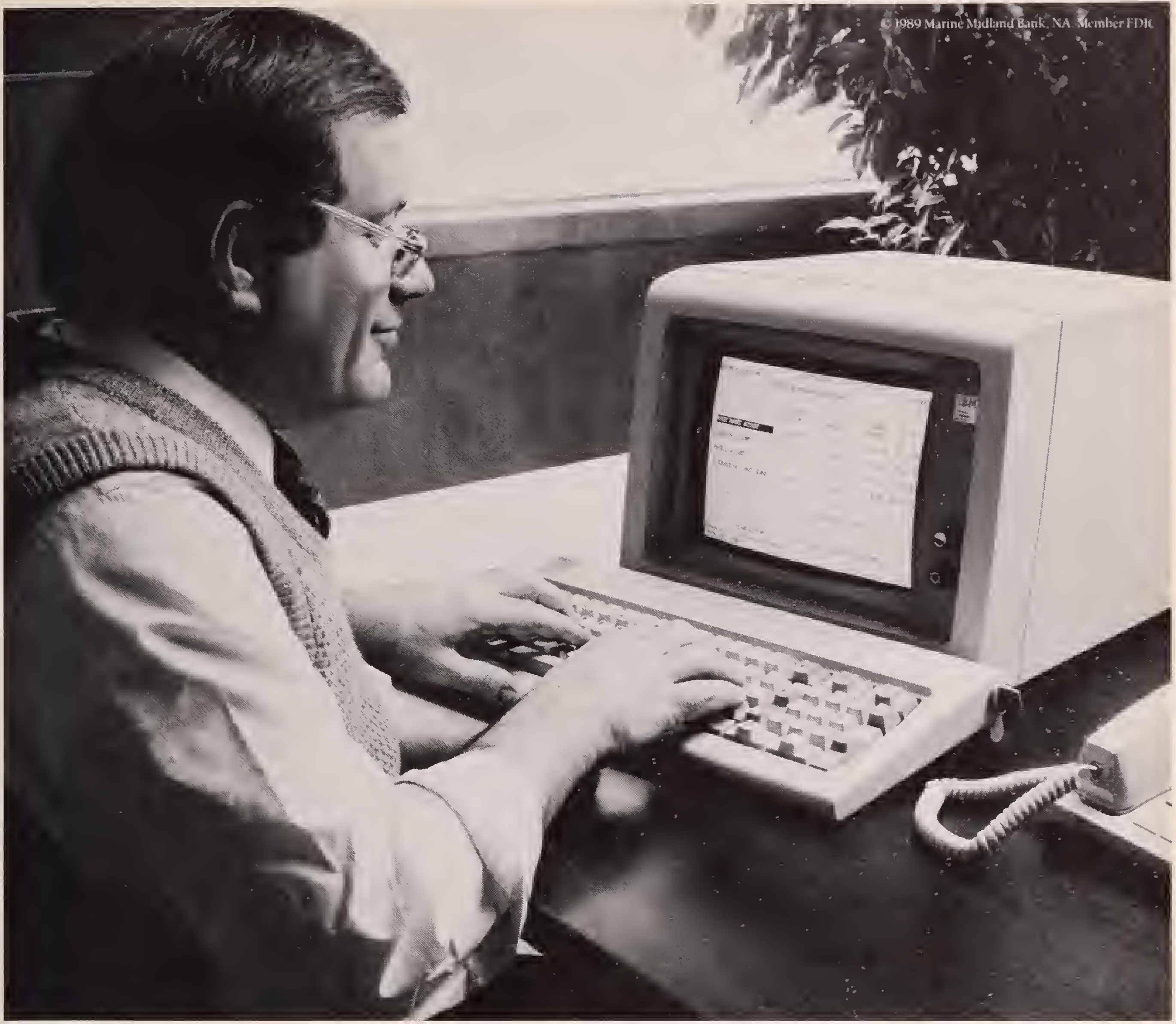

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FRIDAY, SEPTEMBER 15

8:30 a.m.–9:30 a.m.	Medical Technology, *Nassau A*
9:30 a.m.–12:30 p.m.	Gastroenterology/Colon & Rectal Surgery, *Sutton Center*
	Computers in Medicine, *Nassau A*
	Occupational & Environmental Health/Family Practice, *Murray Hill B*
	Physical Medicine & Rehabilitation, *Murray Hill A*
	Medical Liability Mutual Insurance Company, *Sutton South*
2:00 p.m.–5:00 p.m.	Chest Diseases/Radiology, *Sutton Center*
	Cardiovascular Diseases, *Sutton South*
	American Medical Women's Association/Orthopaedic Surgery Society, *Murray Hill A*
	Neurology, *Nassau A*
	Neurosurgery, *Murray Hill B*
	Emergency Medicine/Critical Care, *Nassau B*

SATURDAY, SEPTEMBER 16

8:30 a.m.–9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m.–12:30 p.m.	Substance Abuse, *Sutton North*
	Internal Medicine (All Day), *Sutton Center*
	Neurology/Ophthalmology (All Day), *Beekman Parlor*
	Psychiatry (All Day), *Nassau B*
	Obstetrics & Gynecology/Urology, *Murray Hill A*
	General Surgery, *Sutton South*
	Dermatology/Plastic Reconstructive Surgery, *Nassau A*
	Allergy & Immunology, *Sutton North*
2:00 p.m.–5:00 p.m.	Internal Medicine (All Day), *Sutton Center*
	Neurology/Ophthalmology (All Day), *Beekman Parlor*
	Psychiatry (All Day), *Nassau B*
	Pediatrics/Forensic Medicine, *Sutton North*
	Otolaryngology/Neurosurgery/Plastic Reconstructive Surgery, *Sutton South*
	Plastic Surgery, *Regent Parlor*
	Dermatology/Plastic Reconstructive Surgery (All Day), *Nassau A*

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Technical Exhibits

NEW YORK STATE
JOURNAL OF MEDICINE

May 1989 • Volume 89, Number 5

COMMENTARIES

The enigma of extrapulmonary tuberculosis

Infection with *Mycobacterium tuberculosis* can result in a protean disease that can involve multiple organ systems. It may be indolent or may disseminate rapidly, leading to death.[1] Over the past two decades the number of reported cases of extrapulmonary tuberculosis in the United States has remained relatively constant, with approximately 4,000 cases per year, despite the steady fall in the number of reported cases of pulmonary tuberculosis.[2]

Thus there is an apparent increase in the proportion of cases of extrapulmonary tuberculosis that are reported. A number of theories may explain this observed increase in frequency of extrapulmonary tuberculosis[3]: extrapulmonary tuberculosis may be increasingly recognized and reported; it may be overdiagnosed; the incidence could be increasing in a reservoir of previously infected patients; or any combination of these three factors may be involved. It is certainly possible that improved recognition and reporting could increase the number of observed cases of extrapulmonary tuberculosis. Previous studies have suggested that a large number of cases of active tuberculosis may be missed during life and are only ultimately found during autopsy.[4,5]

It is unlikely that extrapulmonary tuberculosis is overdiagnosed, as a number of studies have demonstrated high recovery rates of the organism on culture.[3,6] Certainly the diagnosis of extrapulmonary infection is much more difficult to establish than tuberculosis within the lung because of the protean nature of the disease and the lack of a simple screening test such as a chest radiograph.

Although the number of cases of extrapulmonary tuberculosis in the United States has remained relatively constant over the last two decades, this actually represents a falling rate in view of the steady population growth. This makes it unlikely that there is an increasing reservoir of infected patients. In addition, tuberculin testing and chest film screening have helped identify patients who may need preventive chemotherapy. All of these procedures have helped contribute to the fall in the incidence of pulmonary tuberculosis. However, a number of patients with extrapulmonary tuberculosis may have a normal chest film[7,8] and thus may not be identified as potential candidates for preventive chemotherapy.

In this issue of the *Journal*, Sciammarella and Bruckstein[9] describe a 66-year-old woman with distal colonic tuberculosis, tuberculous enteritis, peritonitis, and pericarditis. The report reminds us that a high index of suspicion is necessary in making a diagnosis of extrapulmonary tuberculosis. A number of clinical factors should arouse suspicion, including long term steroid use, chronic renal failure, blood dyscrasias, chronic obstructive pulmonary disease, gastrectomy, alcoholism, and atherosclerotic cardiovascular disease. All have been reported to increase the risk for reactivation of previously acquired tuberculous infection.[10,11]

Epidemiologic factors may also predict risk. Individuals of foreign birth or those exposed to others with active tuberculous infection appear to have an increased risk for the development of extrapulmonary tuberculosis.[6,12] In addition, individuals with increased risk for human immunodeficiency virus (HIV) infection may also have an increased risk for the development of primary tuberculous infection with subsequent dissemination to extrapulmonary sites.[13] Extrapulmonary tuberculosis can occur at any age, but tends to predominate in the third through sixth decades of life.[2,6,14]

Constitutional symptoms may also suggest the possibility of a chronic infectious disease process such as extrapulmonary tuberculosis. However, symptoms such as fever, weight loss, night sweats, malaise, and fatigue can also be seen in patients with malignancies, chronic connective tissue disease, chronic renal failure, and numerous other medical processes.

Physicians may also miss the diagnosis of extrapulmonary tuberculosis because of a normal chest film or a negative tuberculin test. However, both of these can occur in individuals who have disseminated disease.[7,8,15] Most negative tuberculins occur among older patients or those who are severely ill.[15] Chest radiography, although usually positive, may be negative in as many as a third of all patients with extrapulmonary tuberculosis.[12]

Establishing the diagnosis of extrapulmonary tuberculosis is difficult unless direct examination and proper culture techniques are used, since the clinical manifestations are so numerous and diverse. It is essential that all chronic infectious disease processes be considered as possible tuberculosis and appropriate cultures and biopsy be performed. Laboratory abnormalities such as thrombocytopenia, anemia, monocytosis, eosinophilia, leukocytosis, leukopenia, leukemoid reactions, hyponatremia, and alkaline phosphatase elevations have been described in other series, in patients with extrapulmonary tuberculosis.[15] However, these variables may be normal or only slightly abnormal in a large percentage of patients with extrapulmonary disease.[12]

The organ system involvement with extrapulmonary tuberculosis can be solitary or disseminated. Infections of the genitourinary system, lymphatic system, and respiratory system other than the lung make up the majority of extrapulmonary cases[2,6,14]; but any organ system can be involved. Again, appropriate tissue examination through biopsy and culture must always be performed.

In conclusion, it is important to realize that extrapulmonary tuberculosis is not an uncommon disease and can occur in divergent age groups and in multiple organ systems. A high index of suspicion is necessary in making the diagnosis, as the symptomatology may be protean, the chest radiographic findings negative, and, occasionally, tuberculin tests indeterminant. All chronic infectious processes should be considered as possible tuberculosis, with appropriate tissue examination and cultures being performed. Epidemiologic factors may assist in identifying patients who might be at risk, including persons of foreign birth, and those who have a history of prior exposure to tuberculosis and who may have other associated medical illnesses that could predispose them to contracting the disease.

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Abdominal tuberculosis: A disease poised for reappearance

The report in this issue of the *Journal*[1] of a Jamaican immigrant with tuberculous enteritis, peritonitis, and pericarditis emphasizes the prevalence of tuberculosis among Third World immigrants. In some cities, the majority of patients with gastrointestinal tuberculosis are immigrants. The number of new cases of tuberculosis in the United States had been declining 5% per year until 1980, when disease in Asian and Hispanic entrants reversed the favorable trend. In one survey, 14% of Asian male immigrants over the age of 56 had active tuberculosis.[2] This turnabout occurred even before the problem of tuberculosis among AIDS patients was recognized.

Today 9% of AIDS patients admitted to Bellevue Hospital have disease from *Mycobacterium tuberculosis*.[3] Extrapulmonary hematogenous and lymphatic tuberculosis are more common in patients with AIDS. This may increase the number of patients with tuberculous peritonitis because mesenteric lymph node infection causes most peritonitis.

There is little evidence that the pasteurization of milk had much effect on the prevalence of gastrointestinal tuberculosis. The incidence of enteric and laryngeal tuberculosis has long been recognized to increase as patients' pulmonary tuberculosis advanced from minimal to moderate to advanced. Patients whose sputa teem with bacilli are prone to infection of the larynx and bowel. The incidence of laryngeal and gastrointestinal tuberculosis showed a striking decline between the first and second World Wars as collapse therapy (pneumothorax, pneumoperitoneum, thoracoplasty) reduced the tubercle bacilli in contact with patients' laryngeal and gastrointestinal mucosa. Drug treatment for pulmonary tuberculosis caused a further sharp reduction in the incidence of laryngeal and gastrointestinal disease. Moreover, gastrointestinal disease has always been more common in poor countries where access to cow's milk is limited but where advanced pulmonary disease is common. It remains an everyday problem in impoverished areas even where boiled milk is used. Then, too, *M bovis* was not distinguished from *M tuberculosis* in early reports.

Tuberculous enteritis is rare in US citizens unless there is antecedent advanced pulmonary tuberculosis. In contrast, in India the majority of patients with enteritis are reported to have normal chest roentgenograms.[4,5] Similar discrepancies exist with tuberculous peritonitis. In some reports, there is a 2:1 female predominance, yet in others the sexes are equally represented.[6,7] The conclusions investigators draw regarding the frequency with which tuberculous enteritis and peritonitis occur together (uncommon) or are associated with active pulmonary tuberculosis (common with enteritis, less so with peritonitis) is influenced by the nationality studied. The tubercle bacillus behaves differently in various peoples and epidemiologic circumstances.

This muddle is further complicated because the complaints emphasized are biased by the author's practice. Surgeons stress abdominal pain in tuberculous peritonitis, particularly in immigrant children in whom it mimics appendicitis. Internists are impressed that peritonitis is indolent. The relative absence of pain and rebound tenderness often result in misdiagnosis of alcoholic liver disease with ascites. Tuberculous enteritis also is treacherous. One third of enteritis patients finally present as surgical emergencies with acute abdominal pain, but the duration of antecedent symptoms often suggests Crohn disease or occult malignancy.

There is common agreement on the following. Only 25–50% of US patients with gastrointestinal tuberculosis have active pulmonary tuberculosis.[8] Only a small minority have diabetes, take corticosteroids, or are immunosuppressed. In immigrants, two to five years typically elapse between entry into the United States and the appearance of symptomatic gastrointestinal tuberculosis.[6] A negative tuberculin skin test, or a chest roentgenogram that shows only remote tuberculous sequelae, do not exclude the diagnosis of gastrointestinal tuberculosis. Tuberculous peritonitis and enteritis seldom coexist because most peritonitis results from reactivation in mesenteric lymph nodes rather than transmural extension from the bowel. A chest film, pelvic examination, and urinalysis should be done when enteritis or peritonitis are suspected, but if there are no symptoms, physical findings, or laboratory data to suggest extra-abdominal disease, it is not necessary to perform a salpingogram, intravenous pyelogram, or liver biopsy.

The diagnosis of tuberculosis should be considered in every patient ill with abdominal pain, ascites, intestinal obstruction, internal fistula, malabsorption, or perirectal fistula. Patients with these complaints must have a chest film and a two-step tuberculin skin test when first seen. These tests are pivotal. Delays in diagnosis begin when these simple, inexpensive studies are put off. The combined US experience indicates gastrointestinal tuberculosis is rare among afebrile patients whose tuberculin skin tests and chest roentgenograms are truly negative.

The two-step tuberculin test is done when the first skin test is negative or indeterminate (5–9 mm of induration). In the second step, the same intermediate strength antigen (5 tuberculin units) is applied one week later. Induration of 10 mm or more constitutes a positive reaction. The two-step test is necessary because tuberculin reactivity wanes with age and degree of health. When the two-step technique is used in nursing home patients, up to 15% of those whose first skin test is negative will have a positive skin test when retested one week later. This booster phenomenon reflects remote primary infection or impaired cell mediated immune response.

It is important to remember that when the patient is debilitated, has fever, and has evidence of new or old granulomatous disease on the chest roentgenogram, abdominal tuberculosis must be considered even if the second skin test is negative.

In this country peritoneal tuberculosis is more common than tuberculous enteritis.[8] Direct hematogenous seeding of the peritoneum from primary pulmonary tuberculosis causes the abdominal lymph node infection that later reactivates to involve the peritoneum. This typically occurs years after the primary infection so tuberculous peritonitis usually is not accompanied by pleuropulmonary disease. Less commonly, tuberculous peritonitis is seen in younger patients as a complication of progressive primary infection. In these patients, peritonitis closely follows hematogenous dissemination; therefore, this peritonitis is sometimes accompanied by residua of the primary lung infection. These remnants help establish the diagnosis. They include a pulmonary infiltrate, hilar adenopathy, or pleural effusion. Cryptic pericarditis also is found in such patients.

Tuberculous peritoneal fluid is a lymphocytic exudate but when tuberculosis complicates cirrhosis with ascites, the resulting fluid may have a low specific gravity and protein content. Lymphocyte counts are between 200 and 2,000 cells/mm^3. Tuberculous ascites can loculate as bowel, omentum, and mesentery adhere, sometimes forming a palpable mass. Bowel obstruction may result. Pericentesis and percutanous peritoneal biopsy therefore are best done with ultrasound guidance. Laparoscopy or laparotomy are safer than percutaneous Cope needle biopsy. Caseating granulomas usually are demonstrated in histologic sections but acid fast bacilli commonly are absent even in material obtained at laparotomy. Smears and cultures of peritoneal fluid for acid fast bacilli usually are negative. Antituberculous treatment therefore must be based on the epidemiologic circumstances, clinical findings, and the presence of granulomas. Bloody peritoneal fluid is very much against the diagnosis of tuberculous peritonitis. Neither computed tomography nor magnetic resonance imaging can reliably distinguish gastrointestinal tuberculosis from cancer.

In a ten-year review of their experience with tuberculous enteritis in South Africa, Gilinsky and his co-authors[9] found that their patients' initial complaints were abdominal pain (66%), weight loss (66%), vomiting (50%), diarrhea (50%), and fever (50%). In some other series, constipation has been as common as diarrhea. Tuberculosis tends to localize in the ileocecal area and ascending colon, so pain and tenderness are most common in the right lower quadrant of the abdomen. A mass may be palpable there. Bowel distal to the ileocecal area is involved in about one third of the cases. Tuberculous enteritis causes bowel obstruction and malabsorption with equal frequency. In recent US series, the mean duration of symptoms before diagnosis was nearly five months and the patients' presenting complaints closely approximated those of Gilinsky's patients in South Africa.[9] The association of advanced pulmonary tuberculosis with laryngitis and enteritis is still common in this country, but the diagnosis requires painstaking inquiry about changes in voice timbre and quality combined with a careful review of gastrointestinal complaints. These diagnoses are overlooked because minor symptoms are ignored and rapidly resolve with drugs given for the pulmonary tuberculosis that dominates the picture.

Tuberculous enteritis must be considered when diagnoses of Crohn disease, appendicitis, abdominal carcinomatosis, lymphoma, ischemic colitis, ulcerative colitis, actinomycosis, and fungal or amebic infection are being considered. Ileocecal tuberculosis is difficult to distinguish from Crohn disease clinically or radiographically. Mucosal noncaseating granulomas are found in Crohn disease, whereas tuberculosis classically causes caseating granulomas in the submucosa of the bowel. Nevertheless, in some studies only a minority of biopsies taken at laparotomy show caseation. In these patients stains and cultures for tuberculosis often are negative. A tuberculin skin test should be done in all patients with Crohn disease and a trial of antituberculosis drugs given if they fail to respond to treatment. Crohn disease is uncommon in immigrants and affects younger patients, most of whom would be expected to be tuberculin negative.

Maglinte and Kelvin[10] stress that a cursory small bowel follow-through tacked on to an upper gastrointestinal contrast series does not adequately define ileocecal disease. A conscientious small bowel examination or enteroclysis provides the detail needed. Tuberculosis and Crohn disease both can produce a string sign (persistent narrowing) in the barium contrast column in the small bowel. Tuberculous strictures tend to be less than three centimeters in length. Longer strictures suggest Crohn disease. In tuberculosis, the ileum proximal to the stricture is more likely to be dilated with a small bowel silhouette that is irregular. Crohn disease seldom causes ileal dilation and the mesenteric border of the small bowel proximal to the stricture tends to be smooth. Perforation is uncommon in tuberculosis and rare in Crohn disease. Calcifications in the liver or abdominal lymph nodes, pleuritis, pericarditis, and coexistent pulmonary disease each may serve to distinguish tuberculous ileocolitis from Crohn disease or ulcerative colitis.

The terminal ileum often can be examined with the fiberoptic colonoscope. Ileocecal and colon biopsies should always be cultured when tuberculosis is being considered. Cultures may be positive when stained histologic sections show neither tubercle bacilli nor caseating granulomas.

Colonic tuberculosis is segmental, almost never diffuse. Ileocecal tuberculosis tends to produce a foreshortening of the ascending colon not seen with cancer. Tuberculosis, lymphoma, and colon cancer all may show a tumor-like blush with angiography. Gastrointestinal hemorrhage rarely is caused by tuberculosis but tuberculosis should be considered if the bleeding patient is an immigrant or has active pulmonary disease. A tuberculin positive patient with a rectal fistula in any position other than the posterior or mid-line may well have tuberculosis. Tuberculin positive patients with chronic, obscure abdominal pain may have tuberculous mesenteric lymphadenitis and be cured by a therapeutic trial of antituberculous chemotherapy.

Granulomatous hepatitis can develop in any patient with miliary tuberculosis. Stains and cultures for tubercle bacilli should be done on liver biopsy specimens taken from AIDS patients with fever, hepatomegaly, and abnormal liver function tests even when the histologic sections fail to show granulomas. Tubercle bacilli also may reach the liver through the portal vein or abdominal lymphatics. This can result in a hard nodular liver that mimics cancer, or obstructive jaundice from enlarged lymph nodes in the porta hepatis.

Because drug resistant tuberculosis is common in immigrants and ghetto populations prone to abdominal tuberculosis, more than two antituberculous drugs are used. The use of isoniazid, rifampin, and pyrazinamide for two months followed by isoniazid and rifampin for a total of one year is effective. Many would add daily ethambutol or streptomycin to this regimen in the first two months when dealing with patients who might fail to complete treatment. It is not uncommon for internal fistulae or even subacute bowel obstruction to resolve with drug treatment without surgery; therefore early on it is often wise to confine laparotomy to diagnostic biopsy. Few patients initially treated with antituberculous drugs subsequently come to surgery for complications. English surgeons experienced with immigrant populations seem to favor right hemicolectomy over ileotransverse bypass when resection must be considered.[11,12] Sketchy data suggest that bowel obstruction and constrictive pericarditis are reduced if prednisone is given, 30 mg each day for three months, and then tapered.

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Drug use in pregnancy

The problems of drug abuse in pregnancy have been with us since antiquity, but it was not until 1973, with the modern description of the fetal alcohol syndrome,[1] that public attention in the United States was directed toward the possible teratogenic effects of recreational drugs consumed during pregnancy. It was also at this time that descriptions of withdrawal suffered by neonates exposed in utero to narcotics[2] emphasized the need for further study of the behavioral and neurological effects of drugs on the developing fetus and child. The article by Kaye et al[3] in this issue of the *Journal* provides important new information on this subject. It reminds us of the special efforts that need to be made to deal with the various facets of this problem.

In the last few years, the impact of cocaine on American society has been repeatedly documented,[4–6] but its effect on human reproduction has only recently been appreciated. The first published report on the effects of cocaine on pregnancy and neonatal outcome[7] documented the high rate of spontaneous abortions, abruptio placentae, and neonatal neurobehavioral deficiencies. Subsequent studies have confirmed these preliminary findings and have documented a high rate of premature labor and delivery,[8,9] intrauterine growth retardation,[8] sudden infant death syndrome,[9] and malformations of the genitourinary tract[10] in cocaine-exposed infants. Additionally, risks for intrauterine cerebral vascular accidents associated with maternal cocaine use have been described.[11] Other studies published in the obstetric and pediatric literature have corroborated these findings. Bingol et al[12] found an increased rate of malformations and intrauterine growth retardation in a population of cocaine-exposed infants. Oro and Dixon[13] found a high rate of prematurity and intrauterine growth retardation in neonates exposed perinatally to cocaine or methamphetamines. Thus, it is becoming clear that cocaine use in pregnancy places the pregnancy and the neonate at high medical and neurobehavioral risk.

The only published information concerning the postneonatal period with regard to the infant exposed to cocaine in utero reports that passively exposed infants at four months of age continue to demonstrate motor disabilities with tremulousness, hypertonicity, and retention of primitive reflexes.[14] There is no reliable information regarding long term outcome of infants passively exposed to cocaine or other drugs of abuse. To best evaluate these children at school age, environmental factors must be taken into account. These environmental factors are not only socioeconomic but should include aspects of the maternal/infant relationship, including maternal psychopathology and personality disorders.

The problems involved in evaluating the effects of maternal substance abuse in the developing fetus and infant are multiple, not the least of which are the difficulties involved in following these infants over a long period of time. The chaotic and transient nature of the drug seeking environment impairs the intensive follow-up and early intervention processes necessary to ensure maximum development by each infant. In addition, most women from substance-abusing backgrounds lack a proper model for parenting. These factors, compounded by the early neurobehavioral deficits of the drug-exposed newborns, earmark these infants to be at high risk for continuing developmental and later school problems.

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Birth outcomes for infants of drug abusing mothers

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ABSTRACT. Birthweight, gestational age, and Apgar scores of drug-exposed infants were compared to those of drug-free infants using data collected from birth certificates. Infants born to abusers of opiates and cocaine as well as opiates and cocaine together were also compared to one another. The presence of adverse neurologic signs, need for intensive care, and length of hospitalization after delivery were compared among different types of drug-exposed infants using data collected in a program for infants at risk for developmental delay.

Infants of all drug abusers weighed an average of 423 g less than controls; mean gestational age for infants in the different drug groups ranged from four to ten days less than for controls. Opiate-exposed infants were significantly more likely to show adverse neurologic signs than were those with exposure only to cocaine, and were more likely to need intensive care. Infants of polydrug (cocaine + opiate) abusers fared worse than infants born to abusers of single drugs with respect to birthweight, gestational age, and length of hospital stay. Outcomes for infants of "crack" abusers were worse than those for infants exposed to other forms of cocaine with respect to birthweight and adverse neurologic signs. Our findings suggest that special outreach and follow-up efforts may be needed to facilitate the optimal development of these infants.

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An estimated five million people in the United States are believed to use cocaine regularly.[1,2] It is now the leading substance abused by pregnant women in New York City.[3] The adverse effect of maternal drug abuse on fetal and neonatal development is not a new phenomenon, but one that has a disconcerting tendency to manifest itself in different forms. While the problems of infants born to opiate addicts have been extensively described,[4–8] there have been relatively few studies of cocaine's impact on the neonate.

Bingol et al[3] were the first to report low birthweight and small size for gestational age in infants born to abusers of cocaine alone. These authors also noted in this group an increased rate of stillbirths (related to abruptio placentae) and congenital malformations. Prior to their report, published studies of cocaine's effect on birth outcomes had involved relatively few infants.[9–11] Ryan et al[12] noted that infants who were exposed to both cocaine and opiates prenatally fared somewhat worse (in terms of fetal loss, infant mortality, birthweight, length, and head circumference) than did those who were exposed only to opiates. Infants born to opiate and cocaine abusers in New York City have been noted to have a mortality rate much higher than that reported for the city overall: from 1978 to 1984, the mortality rate for infants born to drug abusers was 40 per 1,000 live births, approximately 2.5 times as high as that for New York City infants in general.[13]

The purpose of this study was to compare birth outcomes of infants born to cocaine, opiate, and polydrug (cocaine + opiate) abusers to one another, and to those of infants born to drug-free mothers. Given the theoretically greater potency of "crack" (a smokable form of cocaine) as compared to other forms of cocaine,[2] an attempt was made to discover whether birth outcomes of children born to crack addicts differed from those of children born to women who abused other forms of the drug. The prevalence of polydrug abuse in the sample provided the opportunity to study the status of these infants in relation to those of cocaine or opiate addicts, and thus to test the hypothesis that simultaneous exposure to central nervous system depressants and stimulants produces an interaction in which each drug antagonizes the effects of the other.[14]

We examined birthweight, gestational age, one- and five-minute Apgar scores, presence of neurologic signs, need for intensive care, and length of hospitalization after delivery. The selection of this last variable was made with the understanding that it could reflect at least three processes: illness, severity of withdrawal, and social factors (ie, difficulty in placing some drug-exposed infants in foster care). In order to study more precisely the effect of maternal drug abuse on these birth outcomes, we controlled for various confounding socioeconomic variables.[15]

Information collected in New York City's Infant Health Assessment Program (IHAP) was used to identify 585 drug-exposed infants. The purpose of IHAP is to identify and monitor the development of infants at risk for developmental delay, as well as to refer them to appropriate health care, educational, and social services. Currently, the program operates in 12 of New York City's hospitals, 11 of which are municipal and one voluntary. The population that is served by the city's municipal hospitals tends to be of low socioeconomic status; 54% of deliveries

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at the municipal hospitals are covered by Medicaid. Overall, this population contains a large proportion of recent immigrants. However, proportions of black and Hispanic patients differ considerably among municipal hospitals.

Maternal drug abuse is just one of the factors that IHAP defines as risks for delayed development. Other risk factors include prematurity (gestational age less than 33 weeks), low birthweight (less than 2,001 g), presence of a major congenital anomaly or inherited metabolic disease, hospitalization (for more than ten days) in the neonatal intensive care unit for serious illness or complications, maternal alcohol abuse, and maternal age less than 16 years. Use of data from this program to study problems associated with maternal drug abuse is warranted for two main reasons: It permits study of a much larger population than would be found in any one hospital and thus furnishes a better understanding of the problem's nature and dimensions; and knowledge gained from the study can be used to improve services offered by the program, as well as to try to reduce drug abuse during pregnancy.

PATIENTS AND METHODS

Infants born to drug abusing mothers at 12 New York City hospitals during June, July, August, and September of 1986 were identified on the basis of information collected in registration forms used by the Infant Health Assessment Program. The decision to identify drug-exposed infants using data from IHAP registration forms rather than data recorded on birth certificates was based on the fact that IHAP information about maternal drug abuse is more complete and specific than that from birth certificates. The total number of IHAP-registered infants who were born to drug abusers in 1986 was 1,959, while birth certificates recorded only 1,161 drug-exposed infants in the 12 IHAP hospitals.

Although IHAP data were used to identify infants born to drug abusers and to specify type of drug abused, information about three birth outcome variables—birthweight, gestational age, Apgar scores— was obtained from birth certificates. Data from IHAP registration forms were used for comparing length of hospitalization after delivery, need for intensive care, and presence of neurologic signs between infants in various drug groups. Information about these three outcome variables was not available from birth certificates. IHAP data about these outcomes were drawn from medical charts and delivery log books.

During the months of June, July, August, and September 1986, 613 IHAP registration forms were available for infants born to drug abusing mothers. Of the total 613 drug-exposed infants, 28 were eliminated from our analysis because they were twins, or were born to mothers who abused only alcohol or marijuana. Infants who died before discharge were not included in this study.*

We attempted to find the birth certificates for the remaining 585 infants and were successful in 571 cases. Once birth certificates were available, we attempted to match these 571 infants to drug-free controls born within an interval two weeks before or after the IHAP-registered infants on the basis of infant's sex; hospital of birth; age, race, and ethnicity (Hispanic or non-Hispanic) of mother; and method of payment for delivery (Medicaid, private insurance, self-payment). Five hundred and sixty-six of the infants were matched, although in 198 cases, one or more of the following criteria had to be relaxed: Time of birth of control infants was extended from four weeks surrounding the case infant's birth to a period spanning two weeks before to nine weeks after (111 cases). The age range for control mothers was expanded to within two years of the age of case mothers for teenagers and within five years for older mothers (114 cases). A different method of payment for delivery was chosen for control mothers (five cases); a different institution of birth was also chosen for control mothers (17 cases). Although it would have been desirable to have matched cases and controls for maternal use of tobacco and alcohol, it was impossible to do so because of insufficient data on the birth certificates.

TABLE I. Distribution of Infants in Individual Drug Groups

Drug	Frequency	Percent
Cocaine (powder, intravenous, unspecified form; might include crack)	224	38.3
Crack	33	5.6
Crack/cocaine (both recorded; unspecified combination)	55	9.4
Cocaine (powder, intravenous, unspecified form) + opiates and sometimes alcohol, marijuana, barbiturates, and miscellaneous drugs	81	13.8
Crack or crack/cocaine + opiates and sometimes alcohol, marijuana, barbiturates, and miscellaneous drugs	15	2.6
Cocaine (powder, intravenous, unspecified form) and alcohol, marijuana, barbiturates, and miscellaneous drugs (but not opiates)	52	8.9
Crack or crack/cocaine and alcohol, marijuana, barbiturates, and miscellaneous drugs (but not opiates)	18	3.1
Heroin (no cocaine)	15	2.6
Methadone (no cocaine)	38	6.5
Heroin and methadone (no cocaine)	17	2.9
Any opiate and alcohol, marijuana, barbiturates, and miscellaneous drugs (but no cocaine)	9	1.5
Unspecified drug abuse (but known abuse)	28	4.8
Total	585	100.0

The 585 IHAP-registered infants were grouped into 12 "maternal drug abuse" categories using information contained in the IHAP registration forms (Table I). IHAP's information about the presence of drug abuse during pregnancy and the type of substance involved was obtained from several sources: the delivery log book, the medical chart of the mother or infant, a social worker's report, or, rarely, from the interview between the IHAP Public Health Advisor and the mother. Only one hospital in this study routinely screened every newborn for maternal drug abuse, using results from infant urine toxicology testing.

For a better understanding of the data, infants in the first 11 categories were combined into five larger groups, as described in Table II. In this report, results are presented and discussed in terms of these larger groups, although we initially calculated and reviewed the findings for the smaller categories. In an effort to distinguish the effects of opiates from those of cocaine, we defined the following three mutually exclusive groups:

I. opiates without cocaine (categories 8–11): users of opiates, with or without additional drugs (except cocaine).

II. cocaine without opiates (categories 1, 2, 3, 6, 7): users of cocaine, with or without additional drugs (except opiates).

III. cocaine + opiates (categories 4 and 5): users of both cocaine and opiates, with or without other drugs.

To differentiate the effects of crack from those of other forms of cocaine, two subdivisions of group II were created:

IIA. crack without opiates (categories 2, 3, 7): users of crack, with or without additional drugs (except opiates).

IIB. other cocaine without opiates (categories 1, 6): users of non-crack cocaine, with or without additional drugs (except opiates).

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* Among all 1,071 IHAP infants registered from June through September 1986, there were 69 deaths which occurred before hospital discharge; at least six of these deaths were among infants born to drug abusers.

TABLE II. Distribution of Infants in Larger, Combined Drug Groups (N = 557)

Larger Drug Group (and Constituent Subcategories)	Frequency	Percent
I. Opiates without cocaine (8–11)	79	14
II. Cocaine without opiates (1, 2, 3, 6, 7)	382	69
III. Cocaine + opiates (4, 5)	96	17
IIA. Crack without opiates (2, 3, 7)	106	19
IIB. Other cocaine without opiates (1, 6)	276	50

Since differentiation of "crack" from "other forms of cocaine" on IHAP registration forms was occasionally ambiguous, there may be some misclassification between the "crack" and "other cocaine" subdivisions.

Methods of Analysis: ***Analysis of Outcomes Derived from Birth Certificates: Birthweight, Gestational Age, and Apgar Scores.*** Birthweights, gestational ages, and one- and five-minute Apgar scores of infants in the larger groups were compared to those of matched drug-free controls by means of t tests. In order to control for additional possible confounders such as maternal educational level, marital status, parity, and prenatal care, as well as for the variables on which drug-exposed and drug-free infants had previously been matched, comparisons for birthweight and gestational age were repeated using an analysis of covariance (GLM SAS).[16] When birthweight was examined as the outcome variable, we controlled for gestation to determine if there was a drug effect on birthweight independent of gestational age. A finding that birthweight is related to drug exposure after controlling for gestational age would indicate that drug use may lead to a decrease in birthweight for gestational age. The model with birthweight as an outcome variable was also tested without controlling for gestation, since a finding of lower birthweight among infants of drug abusers regardless of gestational age would be considered a significant finding.

Using the analysis of covariance, the five drug groups were compared to their controls as well as to each other with respect to birth outcomes. First, a model was constructed to detect any differences between "opiates without cocaine" (I), "cocaine without opiates" (II), and "cocaine + opiates" (III). Second, "crack without opiates" (IIA) was compared to "other forms of cocaine without opiates" (IIB).

Analysis of Outcomes Derived from IHAP Registration Forms: Presence of Neurologic Signs, Need for Intensive Care, and Length of Hospitalization After Delivery. IHAP forms for infants of drug abusers were examined for presence of neurologic signs, need for intensive care, and length of hospitalization after delivery. Drug groups could not be compared to controls on these variables because controls were not IHAP registrants and birth certificates do not contain the information of interest here.

Information about the presence of adverse neurologic signs (ie, tremors, increased tone, seizures) was abstracted from clinical records and entered on IHAP registration forms by the public health advisors; the majority of infants with recorded neurologic signs were also described on registration forms as having a withdrawal syndrome. "Need for intensive care" refers to care more specialized than that routinely given in the regular nursery, which is provided in either a neonatal intensive care unit or a special care nursery.

Mean lengths of hospital stay for infants in the five drug groups were calculated; lengths of stay for infants in the five groups were compared using t tests. Proportions of infants requiring intensive care and demonstrating neurologic signs were calculated and compared using χ^2 tests.

While a nominal significance level of 5% was used for testing individual hypotheses, it should be borne in mind that, as the number of tests increases, the probability of a type I error at the overall study level also increases. However, in this observational study with moderate sized groups, it was felt that making the significance level too stringent could obscure some potentially important effects, which ought to be studied further.

RESULTS

General Description of Sample. The various types of drug abusers ("opiates without cocaine" (I), "cocaine without opiates" (II), "cocaine + opiates (III) and "crack without opiates" (IIA)) may be described in terms of the variables that were used for matching (Table III). Mothers who abused opiates, either alone or with cocaine, were approximately 2.5 years older than those who abused various forms of cocaine without opiates. The groups that included opiate abusers had larger percentages of Hispanics and smaller percentages of blacks than did groups consisting only of cocaine abusers. With regard to the financial status of this sample, the proportions of deliveries covered by Medicaid were approximately the same in all drug groups. However, it should be noted that deliveries that were not covered by Medicaid were not necessarily to patients with any form of private insurance or other means of payment; because this sample was drawn mainly from municipal hospitals, the patients giving birth are often medically indigent. The male to female ratio of infants approached the normal 51:49 ratio in the largest groups; differences from this ratio in some groups may have reflected smaller sample sizes.

The four types of drug abusers are also described in terms of the variables that were controlled in the linear model (but that were not involved in matching). Drug abusing mothers received less prenatal care than did controls, with between 55% and 66% having late or no prenatal care as compared to 40% of all controls; the large proportion of control mothers with inadequate prenatal care is not unusual for women who deliver at municipal hospitals. Cocaine and crack abusing mothers in groups II and IIA had a lower parity (1.4) than the somewhat older opiate and polydrug abusers (1.9 and 2.0, respectively). The proportions of unmarried mothers were approximately the same in all drug groups (89% to 94%) and were higher than the proportion of unmarried women in the control group (69%). There was no difference in educational levels between control mothers and drug abusers, as all had an average of 11 years of schooling.

Analysis of Outcomes Derived from Birth Certificates. *Birthweight.* Examination of the larger drug groups using t tests showed that all differences in birthweight between drug-exposed and drug-free infants were significant ($p < 0.001$) (Table IV). The mean birthweight of infants born to all drug abusers was 2,669 g, compared to a mean control birthweight of 3,092 g. When the analysis of covariance was used to compare infants in the drug groups to their controls, differences in birthweight between drug-exposed infants and their controls remained highly significant. Thus, controlling for maternal educational level, marital status, parity, and prenatal care, as well as for the variables on which drug-exposed and drug-free infants had previously been matched, did not change the nature of these results. Differences in birthweight for cases and controls continued to appear even when we controlled for gestational age, indicating that these differences arose from small size for gestational age as well as preterm birth.

In comparison to infants born to abusers of "opiates without cocaine" (I) or "cocaine without opiates" (II), those of "cocaine + opiate" (III) abusers differed most from their controls (mean difference = 653 g) and had the lowest mean birthweight; those born to abusers of "cocaine without opiates" (II) differed least, though still significantly, from their matched controls (mean difference = 376 g). It should be noted that the use of the analysis of covariance to compare groups I, II, and III to one another showed that the apparent differences between these groups were not significant at the 5% level.

Infants of crack abusers (IIA) differed more from their controls and had a lower mean birthweight than did those born to abusers of other cocaine forms (IIB); the mean difference in birthweight between crack-exposed infants and their controls was 507 g, compared to a difference of 324 g between infants exposed to other forms of cocaine and their controls. When mean birthweights of subdivisions IIA and IIB were compared using analysis of covariance, the difference was significant at the 5% level.

Gestational age. Examination of the larger drug groups using t

TABLE III. General Description of Sample

Maternal and Infant Characteristics (Variables used in matching)	Larger Combined Drug Groups				
	I (Opiates Without Cocaine)	III (Cocaine + Opiates)	II (Cocaine Without Opiates)	IIA (Crack Without Opiates)	Controls
Mean maternal age in years ±SD	28.6 ± 5.1	27.2 ± 4.8	24.7 ± 4.6	24.5 ± 4.6	—
	(N = 77)	(N = 93)	(N = 369)	(N = 103)	
Ethnicity					
% Hispanic	60%	39%	23%	19%	—
% non-Hispanic	40%	61%	77%	81%	—
	(N = 77)	(N = 93)	(N = 369)	(N = 103)	
Race					
% black	34%	53%	78%	82%	—
% white	66%	47%	22%	18%	—
	(N = 77)	(N = 93)	(N = 369)	(N = 103)	
Payment					
% Medicaid	69%	63%	73%	66%	—
% non-Medicaid	31%	37%	27%	34%	—
	(N = 77)	(N = 93)	(N = 369)	(N = 103)	
Infant sex					
% male	57%	45%	53%	50%	—
% female	43%	55%	47%	50%	—
	(N = 77)	(N = 93)	(N = 369)	(N = 103)	
(Variables controlled for in linear model)					
Prenatal care					
% late or no	58%	66%	58%	55%	40%
% early	39%	18%	26%	19%	49%
	(N = 77)	(N = 93)	(N = 369)	(N = 103)	(N = 566)
Maternal education (mean years of schooling)	11.0 ± 1.4	11.0 ± 1.6	11.1 ± 1.8	11.1 ± 1.9	11.0 ± 2.4
	(N = 77)	(N = 91)	(N = 360)	(N = 102)	(N = 558)
Parity (mean)	1.9 ± 1.5	2.0 ± 1.6	1.4 ± 1.4	1.4 ± 1.2	1.3 ± 1.5
	(N = 74)	(N = 88)	(N = 349)	(N = 100)	(N = 531)
Marital status					
% married	9%	6%	11%	8%	31%
% unmarried	91%	94%	89%	92%	69%
	(N = 77)	(N = 93)	(N = 369)	(N = 103)	(N = 566)

tests showed that all except one of the differences in gestational age between drug-exposed infants and their controls were significant ($p < 0.05$) (Table V). The mean gestational age for infants born to all drug abusers combined was 268 days, compared to a mean control gestational age of 274 days. However, the significance of these differences is called into question by the results we obtained when we compared gestational ages of drug-exposed and drug-free infants in the larger groups using analysis of covariance. After controlling for maternal educational level, marital status, parity, and prenatal care in the analysis of covariance, only infants in the crack subdivision (IIA) still had significantly shorter ($p < 0.05$) gestational ages than did their controls.

In comparison to infants born to abusers of "opiates without cocaine" (I) or "cocaine without opiates" (II), those of "cocaine + opiate" (III) abusers again differed most from their controls (mean difference = 10.2 days) and had the shortest mean gestation. Infants born to abusers of "opiates without cocaine" (I) and "cocaine without opiates" (II) had similar gestational ages. Here, too, examination of the cocaine subdivisions showed that infants of crack abusers differed more from their controls and had shorter gestations than those born to abusers of other cocaine forms; the mean difference in gestation between crack-exposed infants (IIA) and their controls was 8.1 days, compared to a difference of 3.9 days between infants exposed to other forms of cocaine (IIB) and their controls. However,

TABLE IV. Birthweights of Infants in Larger, Combined Drug Groups

Drug Group (Number of Infants in Group With Known Weight)	Mean Birthweight of Drug-Exposed Infants (g)	Mean Birthweight of Controls (g)	Mean Difference (g)	Degrees of Freedom	t	Probability
All drugs combined (562)	2,669	3,092	423	561	11.34	<0.001
I. Opiates without cocaine (77)	2,777	3,259	482	76	5.75	<0.001
II. Cocaine without opiates (366)	2,663	3,038	376	365	8.21	<0.001
III. Cocaine + opiates (92)	2,522	3,175	653	91	6.56	<0.001
IIA. Crack without opiates (103)	2,558	3,065	507	102	6.25	<0.001
IIB. Other cocaine without opiates (263)	2,704	3,028	324	262	5.90	<0.001

TABLE V. Gestational Ages of Infants in Larger, Combined Drug Groups

Drug Group (Number of Infants in Group With Known Gestational Age)	Mean Gestational Age of Drug-exposed Infants (days)	Mean Gestational Age of Controls (days)	Mean Difference (days)	Degrees of Freedom	t	Probability
All drugs combined (562)	267.6	273.8	6.2	561	4.18	<0.001
I. Opiates without cocaine (77)	267.8	274.4	6.6	76	2.01	<0.05
II. Cocaine without opiates (366)	267.8	272.8	5.1	365	2.62	<0.01
III. Cocaine + opiates (92)	265.6	275.8	10.2	91	2.74	<0.01
IIA. Crack without opiates (103)	265.8	273.9	8.1	102	2.54	<0.05
IIB. Other cocaine without opiates (263)	268.6	272.4	3.9	262	1.63	>0.10

all apparent differences in gestational age between the five drug groups were not significant when they were examined using the analysis of covariance.

The importance of differences in gestational age of only a few days, whether statistically significant or not, may be argued. However, it must be remembered that the weight gain that occurs in the last two weeks of pregnancy is considerable and, therefore, that differences in gestational age of no more than a week may contribute to reductions in birthweight.

One-minute and five-minute Apgar scores. There was no consistent direction for the differences in one- and five-minute Apgar scores that were observed for drug-exposed infants and their controls; none of the differences were significant at the 5% level. When one- and five-minute Apgar scores were compared between larger drug groups using analysis of covariance, no significant differences emerged.

Analysis of Outcomes Derived from IHAP Data. Since information about outcomes that are analyzed in this section could not be obtained from birth certificates, we were unable to compare drug-exposed infants to matched drug-free controls. Thus, comparisons in this section are only between drug groups.

Presence of neurologic signs. The groups "opiates without cocaine" (I) and "cocaine + opiates" (II) had the highest proportions of infants with adverse neurologic signs, 46% and 44%, respectively (Table VI). These two groups were similar to each other, but χ^2 tests showed them to have a significantly higher ($p < 0.05$) proportion of infants with neurologic signs than the "cocaine without opiates" group (II), in which 22% of infants showed neurologic signs.

Thirty percent of infants in the "crack without opiates" group (IIA) showed adverse neurologic signs, as compared to 19% of infants in the "other cocaine without opiates" group (IIB); a χ^2 test showed that crack-exposed infants differed significantly from those exposed to other forms of cocaine with respect to presence of adverse neurologic signs.

Need for intensive care. The highest proportions of infants needing intensive care were found in groups I and III, "opiates without cocaine" and "cocaine + opiates," which had 39% and 41%, respectively, of their infants needing intensive care (Table VI). Chi-square tests showed that infants in these groups were significantly more likely ($p < 0.05$) to need intensive care than were those in group II, "cocaine without opiates," of whom 21% required intensive care.

Infants of crack abusers did not differ significantly from those of "other cocaine" abusers with respect to need for intensive care.

Length of hospital stay. T tests showed that infants in group III, "cocaine + opiates," had a significantly longer ($p < 0.05$) mean post-delivery hospital stay than infants in either group I, "opiates without cocaine," or group II, "cocaine without opiates" (31.1 days vs 23.1 and 20.1 days) (Table VI). The length of hospital stay for infants of crack abusers was not significantly different from that for infants of "other cocaine" abusers.

DISCUSSION

Infants born to both cocaine and opiate abusers had significantly lower birthweights than controls and, more equivocally, shorter gestational ages as well. Although we did not try to determine incidence of fetal loss among drug-exposed infants, the adverse outcomes that we found for children of cocaine abusers appeared to fall somewhere within the continuum defined by previous studies (which ranges from no effects of maternal cocaine abuse[11] all the way to an increased incidence of abruptio placentae[3,9]).

Most importantly, the association demonstrated here between maternal drug abuse during pregnancy and poor birth outcome persisted even when we attempted to control for confounding socioeconomic variables. Thus, these findings contradict the conclusion of Lifschitz et al[15] regarding opiate abusers, that "...adjustment for confounding variables abolished the difference in birth measurements between infants of drug abusers and controls." However, because our study is an observational one, we cannot conclude that the association between maternal drug abuse and poor birth outcome is causal.

Our findings of decreased birthweights for infants of cocaine and opiate abusers agree at least in part with those reported in earlier studies. The reduction in birthweight that we observed for infants exposed only to cocaine is less

TABLE VI. Birth Outcomes Derived from IHAP Data for Infants in Larger, Combined Drug Groups: Proportions of Infants with Neurologic Signs and Needing Intensive Care; Length of Hospital Stay

	Infants with Neurologic Signs		Infants Needing Intensive Care		Mean Length of Hospital Stay (in days) ± Standard Deviation (Number in Group With Known Length of Stay)
Drug Group	Number/Total With Characteristic Known	%	Number/Total With Characteristic Known	%	
I. Opiates without cocaine	34/74	46%	30/78	39%	23.1 ± 19.3 (72)
II. Cocaine without opiates	78/353	22%	77/371	21%	20.1 ± 23.4 (362)
III.Cocaine + opiates	40/90	44%	38/93	41%	31.1 ± 28.1 (90)
IIA. Crack without opiates	29/97	30%	25/102	25%	23.6 ± 27.2 (103)
IIB. Other cocaine without opiates	49/256	19%	52/269	19%	18.7 ± 21.6 (259)

dramatic than that described elsewhere; in fact, birthweights for cocaine-exposed infants in this study were considerably higher than those reported by Bingol et al.[3] However, the mean birthweight of our polydrug-exposed infants was lower than that reported by Ryan et al[12] for infants who had been exposed to both opiates and cocaine in their study. The significance of the difference in gestational age between drug-exposed infants and controls in our study was more equivocal than that for the difference in birthweight; the gestational ages that we observed for children of heroin and methadone abusers were somewhat higher than those reported by Stimmel et al.[7] It should be noted here that Ryan et al[12] observed no significant differences between the gestational ages of drug-exposed infants and drug-free controls in their study.[12] Lengths of hospitalization after delivery for drug-exposed infants in our study were considerably longer than the routine three-day stay of healthy infants; the lengths of hospitalization that we noted for opiate-exposed infants were in the range of those reported earlier.[7] The proportion of infants with neurologic signs was higher among those exposed to opiates than among those exposed to cocaine alone. In this study, the proportion of cocaine-exposed infants who displayed symptoms that might be consistent with withdrawal was higher than the 10% of cocaine-exposed infants who were identified by Bingol et al[3] as demonstrating withdrawal. The proportion of opiate-exposed infants who displayed signs of withdrawal was higher in our study than in that described by Rosen and Johnson.[6] Our results differ from those reported by Ryan et al,[12] in that we observed no significant difference between the Apgar scores of the drug-exposed infants and those of drug-free controls.

Although infants of various types of drug abusers could not be considered a homogeneous group, for most of the outcomes we observed no statistically significant differences between infants of cocaine and opiate abusers. Only with respect to proportions showing neurologic signs and requiring intensive care did opiate-exposed infants fare significantly worse than those born to cocaine abusers. There may be some bias towards recording neurologic signs in opiate-exposed infants in whom a well-defined withdrawal syndrome has been identified. The findings of a greater need for intensive care among opiate-exposed infants may be more important, since intensive care probably represents a more objective way of assessing infant health status than evaluation of neurologic signs.

The findings for infants of polydrug (cocaine + opiate) abusers certainly run counter to the suggestion that simultaneous exposure to central nervous system depressants and stimulants produces an interaction in which each drug antagonizes the effects of the other.[14] Infants of polydrug abusers had significantly longer hospital stays than did the infants of women who abused either cocaine or opiates alone; birthweight and gestational age were also worse for infants of polydrug abusers, although for these outcomes, differences did not attain statistical significance. As far as presence of neurologic signs and need for intensive care were concerned, infants of polydrug abusers resembled those of opiate addicts. Although infants who were exposed to cocaine as well as opiates in the study by Ryan et al[12] generally fared worse than those exposed to opiates alone, the two groups of drug-exposed infants did not appear to differ significantly from one another; certainly prenatal exposure to cocaine in addition to opiates did not worsen what these authors defined as neonatal abstinence symptoms.

Given the theoretically greater potency of crack compared to other types of cocaine,[2] it was noteworthy that birth outcomes for infants of crack abusers were worse than those observed for infants of mothers who abused other forms of cocaine with respect to birthweight and presence of adverse neurologic signs. Differences in length of hospital stay, though not of statistical significance, also pointed toward worse outcomes for infants of crack abusers.

The fact that drug-exposed infants had greater need for intensive care and longer hospital stays suggests that their treatment will be a costly drain on the financial resources of society, and suggests that successful health educational programs designed to prevent drug abuse during pregnancy will be cost effective. However, additional levels of prevention must also be implemented. These results suggest the need for a surveillance system to monitor postnatal developmental delay among drug-exposed infants, as well as for services designed to minimize developmental problems. Addicted mothers of preterm, low birthweight infants are reportedly less likely to participate in drug treatment programs than are mothers of full-term, normal birthweight infants.[17] The findings of adverse birth outcomes for drug-exposed infants suggest that special outreach and follow-up efforts may be necessary to encourage and facilitate mothers' participation in drug treatment and pediatric programs.

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Wood burning related injuries

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ABSTRACT. During the past two years, 80 patients were seen in the emergency department of The Mary Imogene Bassett Hospital (Cooperstown, NY) for injuries related to the use of wood burning stoves. The types of injuries included 25 lacerations, 19 crush injuries, 10 fractures, 7 eye injuries and 7 burns. Seven of these patients required hospitalization, and five required operative procedures. There was no mortality. Physician and patient education about the potential dangers of wood stove use may help prevent these injuries.
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The use of wood as a fuel source is a common alternative to fossil fuels, especially in rural areas. During the late 1970s and early 1980s, with rising prices of oil and gas, many people returned to burning wood either for primary heating or as a supplementary heating source. An increase in injuries related to burning wood was observed at this institution, including two deaths, which prompted a prospective review of all wood stove related injuries seen at this hospital. A review of the literature revealed various anecdotal experiences, but aside from a review of burns in children by Yanofsky,[1] we found little in the way of cataloging of the variety of injuries associated with the use of wood burning stoves.

Reported here is the experience of The Mary Imogene Bassett Hospital, a rural teaching hospital, with wood stove related injuries over the period 1984–1986. The risks involved in the use of this type of burner and fuel are discussed in an effort to improve preventive efforts and patient education.

METHODS

Emergency department records were kept prospectively for the autumn and winter months of 1984–1985 and 1985–1986 on all patients who presented with chief complaints related to the procurement, preparation, or burning of wood as a fuel source. These records were examined for injuries including burns, lacerations, fractures, puncture wounds, soft tissue injury, and eye injuries. Patients were identified and their records reviewed with regard to type of injury, age, severity, morbidity, and subsequent complications.

RESULTS

There were 81 visits to the emergency department that fulfilled the criteria. These represented 1.2% of all visits during the study period. The injuries are listed in Table I.

Lacerations were the most frequent injuries and accounted for 31% of all cases. The average age of patients presenting with lacerations was 38 years. The most frequent mechanism of injury was related to use of the chain saw (13 cases) or wood splitter (seven cases). These injuries predominantly occurred in people who were harvesting wood from their own property for personal use, or for sale to others to supplement their income. Many were farmers who harvest wood in autumn after crops are in. The lacerations ranged from 4 cm to 15 cm in length. There were five complicated lacerations, including digit amputation, nerve injury, tendon injury, cellulitis, skin loss requiring grafting, or arterial injury. Two patients required hospitalization lasting two days each. The mean length of time lost from work was one day, with a range of from zero days to six months.

Crush injuries were the second most common form of trauma observed. They accounted for 23% of patients. The average patient age was 37 years, and most patients were male, again reflecting the fact that these injuries occur among people harvesting wood. The usual mechanism of injury was dropping a log on an extremity or being hit by a falling branch. Two of these patients required hospitalization, one for six days, the other for two. One of these patients fell from a tree, incurring significant hemorrhage—the hematocrit fell from 44% to 34%—which resolved without a source being identified. The other was crushed between a log and a tree and subsequently developed cellulitis from an abrasion. The morbidity from these injuries averaged seven days of lost work.

The next most common injuries were fractures, occurring in 12% of patients. The average patient age was 35 years. Eight patients had fractured phalanges, one had a trimalleolar ankle fracture, and one had a radial fracture with median nerve damage. Three patients required open reduction and were hospitalized for an average of two days. The mean time lost from work was seven days, with a range of three days to six months.

Burns occurred in seven patients, with an average age of 16 years, making this the most common pediatric injury related to wood stove use. All were first degree and second degree burns covering less than 1–3% of the body. On average, these burns disabled the patients for two weeks.

Eye injuries occurred in seven patients, with an average age of 28

TABLE I. Wood Burning Related Injuries Treated at The Mary Imogene Bassett Hospital (Cooperstown, NY), 1984–1986

Type of Injury	Number	Percent of Total
Lacerations	25	31
Crush	19	23
Fractures	10	12
Burns	7	9
Eye	7	9
Muscle strain	7	9
Punctures	3	4
Smoke inhalation	2	2
Bursitis	1	1
Total	81	100

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years. Five of these patients had corneal abrasions, one a foreign body in the eye, and one conjunctivitis from smoke exposure. All these injuries resolved within two days.

Muscle strain was the chief complaint of seven patients, with an average age of 33 years. Five complained of lumbosacral spine strain, one abdominal muscle strain, and one neck strain. These all resulted from chopping and carrying wood. Most patients with these injuries experienced a resolution of symptoms within a week.

Three patients were seen for puncture wounds, one of which was complicated by infection. Two of these were caused by wood splinters, but one was caused by a piece of steel from a wedge. Two patients were seen for smoke inhalation, and one required hospitalization for a day. One patient was seen for bursitis of his shoulder after chopping wood.

DISCUSSION

The principal health risk from the use of wood stoves is death by structural fire which, according to the National Injury Information Clearing House (NIICH), accounts for about 350 deaths per year.[2] Several deaths that occurred as a result of domestic fire caused by a wood stove were the impetus for our study. No such deaths were seen at this institution during the period included in the study. There was one death of a professional logger who was crushed by a tree during the summer months. This case was not included in this series.

The second major health risk seen in this study was chain saw and wood splitter accidents, and injuries related to residential firewood harvesting and preparation. The NIICH receives reports of 29,000 such injuries each year.[2] We suspect many more go unreported.

Burns from wood stoves, which reach surface temperatures of 300–600°C, are the third major health peril. Children who play in the vicinity of unenclosed wood stoves are especially at risk, as described by Yanofsky.[1] This type of injury can be avoided by properly enclosing the wood stove with a child protective screen.

There are an estimated 10–15 million wood stoves in use across the country, according to the New York State Department of Environmental Conservation (personal communication, Albany office, October 1986), making them a common fixture in approximately 10% of American households and a ready source of injury. Although the current prices of oil and gas are making wood a less attractive alternative, the United States has a relative underutilization of wood. Given the variability of fossil fuel prices, wood is likely to remain a significant source of heating, particularly in rural areas where wood is easily obtainable. It is estimated by the Department of Environmental Conservation that 20% of homes in New York State burn wood as a source of fuel, and that over half these households harvest their own wood.[3] A large number of these harvesters are not professional loggers, which makes this a particularly dangerous undertaking for these people. Chain saw and wood splitter injuries have dramatically increased over the past ten years as these tools have become more common among residential users.[4] The use of protective clothing and eye shields, and the use of properly maintained saws by informed individuals can all reduce risk of injury. We recommend continued efforts at improved safety in the design of tools, and education in their correct use.

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Transfusion reactions

Case Presentation

David Rube, MD (Medical Intern): A 75-year-old man was admitted with a complaint of progressive weakness of one week's duration. The patient had had atherosclerotic heart disease and stable angina pectoris for a period of ten years. Two months prior to admission, he began to experience angina at rest. A stress test at that time was positive, and he was promptly referred for bypass grafting of three coronary arteries. During the surgical procedure he received four units of packed red blood cells; prior to the transfusion, his direct and indirect Coombs tests were negative. The patient's postoperative clinical course was complicated only by atrial and ventricular arrhythmias which were controlled with procainamide hydrochloride, propranolol, and digoxin. He was discharged from the medical center in stable condition with a hematocrit of 29%. Follow-up hematocrit at his physician's office three weeks after the surgery was 30%.

Approximately one week prior to the present admission, the patient began to feel progressively weak and tired on minimal exertion. He denied any chest pain, shortness of breath, nausea, vomiting, fever, palpitations, bright red blood per rectum, or melena. A complete blood count performed in the physician's office revealed a hematocrit of 18%, and the patient was referred for admission to the hospital.

The patient's medical history included a right hernia repair, and a transurethral prostatectomy four years before, at which time he had received several blood transfusions. His medications at the time of the present admission included enteric-coated acetylsalicylic acid, one tablet per day; dipyridamole, 25 mg three times daily; propranolol, 10 mg every six hours; procainamide hydrochloride, 500 mg four times daily; digoxin, 0.25 mg/day; and ferrous sulfate, 324 mg/day. The patient worked as an accountant; he did not use alcohol to excess; he had smoked five cigars per day up until ten years previously, at which time he discontinued the habit.

Physical examination disclosed a well developed, well nourished elderly man in no distress. His blood pressure in the supine position was 115/60 mm Hg, with a pulse rate of 68/min; blood pressure in the upright position was 105/50 mm Hg, with a pulse rate of 76/min; the respiratory rate was 18/min, and temperature 99°F. The skin showed no rashes; no lymph nodes were palpable. Examination of the head revealed only pale conjunctivae and anicteric sclerae. The neck was supple with a full range of motion and with no audible carotid bruits. The chest showed only a sternotomy scar; the lungs were clear on percussion and auscultation. The first and second heart sounds were normal; a grade II/VI systolic ejection murmur was audible at the left sternal border. Abdominal examination was negative; the liver and spleen were not palpable. There was no costovertebral angle tenderness. The rectal examination revealed a black stool which was a trace positive on guaiac test for blood. The genitalia were those of a normal adult male. The extremities showed no cyanosis, clubbing, or edema. The neurologic examination was within normal limits.

Urinalysis yielded entirely normal findings. The hemoglobin (Hb) was 5.7 g/dL; hematocrit (Hct), 16.1%; white blood cell count (WBC), 3,300/μL, with 32% mature neutrophilic polymorphonuclear leukocytes, 21% immature forms, 45% lymphocytes, and 2% monocytes. The reticulocyte count was 6.6 per 100 red blood cells (RBCs). Examination of the peripheral blood smear showed prominent polychromasia, numerous spherocytes, normal appearance of platelets, and no fragmented RBCs. The platelet count was 278,888/μL; prothrombin time (PT), 11.4 sec (control, 12.2 sec); and partial thromboplastin time (PTT), 24.4 sec (control, 26.5 sec). The direct Coombs test was negative; the indirect Coombs test was initially reported as negative but later as weakly positive on retest. Bone marrow aspiration on admission revealed normal cellularity with normal RBC and WBC maturation.

Chemical tests indicated a blood urea nitrogen (BUN) of 21 mg/dL, and serum levels of creatinine, 1.5 mg/dL; glucose, 157 mg/dL; alkaline phosphatase, 92 U/L; creatine phosphokinase, 28 U/L; haptoglobin, 160 mg/dL (normal, 80–300 mg/dL); total protein, 5.9 g/dL (albumin, 3.4 g/dL; globulin, 2.5 g/dL); total bilirubin, 0.9 mg/dL; alanine aminotransferase (ALT/SGPT), 29 U/L; aspartate aminotransferase (AST/SGOT), 26 U/L; lactate dehydrogenase (LDH), 247 U/L; and gamma-glutamyl transferase, 38 U/L. The serum electrolyte levels were as follows: sodium, 138 mEq/L; potassium, 4.6 mEq/L; chloride, 107 mEq/L; bicarbonate, 25 mEq/L; calcium, 7.8 mg/dL; and phosphate, 3.6 mg/L. The serum digoxin level was 1.1 ng/μL, and the procaine level was 3.6 μg/μL.

The electrocardiogram (ECG) revealed normal sinus rhythm, first degree atrioventricular block, elevated ST segments in leads I, II, aV_L, and aV_F; and depressed ST segments in leads V_4 through V_6.

The chest film revealed a normal heart size, with no evidence of pulmonary infiltrations or effusions.

Based on a clinical conference held on New York Infirmary-Beekman Downtown Hospital, New York, NY, on May 13, 1988.

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After transfusion with one unit of packed RBCs, the hematocrit rose to 20% and the hemoglobin to 6.7 g/dL.

In view of the elevated reticulocyte count and the previous marked fall in the hematocrit over a two-week period without an obvious source of bleeding, a series of guaiac tests for blood in the stool were performed, all of which were negative. An adverse drug reaction to procainamide hydrochloride (Procan) was considered; the medication was discontinued, and treatment with prednisone was begun.

During the patient's week in the hospital, his serum LDH level rose to 634 U/L, and thereafter fell to 516 U/L. The direct Coombs test never became positive. It was believed that a hemolytic reaction to procainamide was unlikely, and that the cause of the fall in hemoglobin and hematocrit was a delayed transfusion reaction. Blood samples were submitted to the New York Blood Center for study.

While in the hospital, the patient's bone marrow responded well, with a continued elevation of the reticulocyte count and a rise in the hematocrit to 29%. Therapy with prednisone was rapidly reduced and finally discontinued, and the patient was discharged in good condition.

HISTORICAL PERSPECTIVE

Norbert Brau, MD (Medical Resident): In 1628, William Harvey revealed the nature of the circulation; a few decades later, in 1666, Richard Lower[1] in London reported the first successful transfusion of blood between dogs. Shortly thereafter, Jean-Baptiste Denis[2] from Paris reported preliminary transfusion experiments in which nine out of 19 dogs died. He subsequently secured blood by phlebotomy for the first blood transfusions into two paid healthy human volunteers. Both developed transfusion reactions, and a third volunteer died after a transfusion. Blood transfusions were abandoned over the next 150 years.

In 1824, James Blundell,[3] an obstetrician in London, approached the problem of postpartum hemorrhage with the idea of blood transfusions. In his animal experiments, Blundell had found that interspecies transfusions were incompatible; dogs receiving human blood died immediately. In 1828, he reported the first human blood transfusion, into a woman hemorrhaging with sufficient severity that she would otherwise have died.[3] For the procedure, a volunteer donated blood at the bedside which was then directly infused into the patient's vein. Blundell warned against the possibilities of circulatory overload and air embolism.

In 1901, Landsteiner[4] in Vienna first identified the agglutination phenomenon and subsequently the ABO blood typing system. Subsequently, the administration of blood transfusions was systematized, especially in World War I; but difficulties arose in techniques of storage and the occurrence of clotting. In 1914, A. Hustin[5] reported that citrate and dextrose prevented the donor blood from clotting. The next two decades witnessed the improved organization of blood banking.

In 1943, Beeson[6] described the occurrence of jaundice many months after transfusions. Twenty-five years later, the causative agent, the Australia antigen, was identified. More recently, we have been faced with the problem of transmission of the human immunodeficiency virus (HIV) by transfusion. Today, blood transfusions have a complication rate of between 1% and 5%—some reactions being minor, others much more serious.

Complications of blood transfusions can be categorized as immediate or delayed. The immediate reactions are of immune and non-immune types. The immune-mediated reactions include hemolysis, which Dr Rothman will discuss. Febrile reactions occur in between 1% and 3% of recipients, mostly due to so-called leukoagglutinins or lymphocytotoxin factors, which are antibodies directed against granulocytes rather than against lymphocytes. Sensitivity to platelets may also cause febrile reactions. Uncomplicated febrile reactions typically begin between 30 minutes and 80 minutes after the beginning of the transfusion, and resolve completely within 24 hours.

A rare reaction to white cells can give rise to so-called pulmonary hypersensitivity, which is manifested by dyspnea, fever, and infiltrates on chest film. This complication resolves within 24 hours, once the transfusion is stopped and the patient receives supportive care. However, in patients with already impaired pulmonary function, the outcome can be fatal.

Allergic reactions to blood transfusions usually present with pruritus, urticaria, and, in more severe cases, with bronchospasm. These are thought to be a sensitivity reaction of the recipient to plasma proteins in the transfused blood. Occasionally, there have been reports in which at the time of donation the donor was taking antibiotics, such as ampicillin, or other medications to which the recipient of the transfused blood is sensitive and consequently develops a reaction.

A more severe hypersensitivity reaction to infused plasma proteins can occur in the form of anaphylaxis, most often in individuals in whom the serum levels of IgA are very low or absent. This occurs in about one per 850 persons in the general population. Throughout most of their lives such individuals are asymptomatic; however, they have developed class-specific antibodies against IgA, so that when they are infused with plasma or whole blood or packed red blood cells with as little as 20% of plasma, they develop an anaphylactic reaction. Another immediate reaction is that of circulatory overload in the cardiac-impaired patient, which can lead to pulmonary edema, or in milder cases only to some increase in congestive heart failure.

Massive transfusions are required most often in surgical patients who are experiencing severe acute hemorrhage and in whom as a consequence an almost complete replacement of blood may result. In this situation, citrate intoxication with decrease of serum calcium may have to be corrected with injections of calcium. Hyperkalemia in the recipient can result from the loss of potassium from the donor red cells into the donor plasma during the mild hemolysis that normally occurs during the storage of the blood units. Hypothermia may result from rapid transfusion of cooled blood units, especially in accident victims; usually these blood units can be warmed prior to administration. If only packed RBCs are transfused and replace about half of the recipient's blood, the latter's coagulation factors become diluted; in addition, donor platelets usually do not survive more than two or three days in the stored red blood cell units.

Instances of bacterial contamination of stored blood products were more frequent when glass bottles were formerly used for the storage of transfusion products. With the present closed system of plastic containers, such contamination is very rare. However, once the unit is attached to the transfusion set, it becomes an open system, so that transfusion of any single unit of blood should not continue at room temperature for more than four hours. Bacterial contamination of donor blood can occur, although rarely. Organisms such as *Pseudomonas* may cause septic shock, which is fatal in 50% to 80% of cases. For the sake of completeness, we should mention the transmission by blood transfusion of infectious diseases such as hepatitis, acquired immunodeficiency syndrome (AIDS), cytomegalovirus (CMV) infection, malaria, and syphilis.

Delayed complications of blood transfusions include delayed hemolysis, which we believe that this patient had, and which Dr Rothman will discuss.

In patients with hematologic disorders who require multiple blood transfusions, the long term complications of iron overload with hemochromatosis may develop. When more than 20 to 50 grams of iron have been transfused, the body has no normal mechanism to excrete the excess iron, and hemochromatosis develops, usually after a total of 50 to 100 units of transfused blood.

When transfusion reactions such as fever, urticaria, and hypotension develop, it is difficult to determine initially from these symptoms what kind of reaction is occurring. Fever is nonspecific and can occur as an isolated mild reaction or can accompany acute hemolysis or allergic reactions. Whenever there is doubt, the transfusion should be stopped, and an intravenous line should be maintained in the event that hypotension develops and the patient needs intravenous medications.

The routine procedure following a transfusion reaction includes examination of the donor unit for any signs of bacterial contamination, such as discoloration or formation of clots; culture of the unit for bacterial growth; and typing and cross-matching of the donor unit again with pre- and post-transfusion blood samples from the patient. By this means, an acute ABO hemolytic reaction can be excluded. When acute hemolysis and bacterial contamination have been eliminated as the cause of a febrile reaction, the reaction is attributed by exclusion to an antileukocyte reaction. Because of its expense, antileukoagglutinin testing is not routinely available in most blood bank laboratories.

Precautions against transfusion reactions and their treatment depend on their causes. Febrile reactions against white blood cells can be treated with further observation and antipyretics; the condition usually improves spontaneously within 24 hours. In the event of single isolated febrile reactions, it is usually not recommended that the patient be subsequently transfused with leukocyte-poor blood, prepared by removal of buffy coat after centrifugation. However, such a recommendation might be appropriate for a recipient who had already experienced two or three such febrile reactions. In the rare instances of pulmonary hypersensitivity, the treatment is supportive with careful observation; subsequent blood donations should not be taken from multiparous donors. Women who have had several pregnancies can often develop anti-white blood cell antibodies consequent to fetal-maternal transmission of blood. Donor units from such women have been reported to cause pulmonary hypersensitivity reactions against the white blood cells of the recipient. To avoid this type of complication, such multiparous women should be excluded from donating blood, as should other donors who have received multiple transfusions themselves in the past.

The patient in whom a mild allergic reaction develops with urticaria or a rash can be treated with antihistamines alone and observed further. Subsequent blood transfusions should be administered with as little plasma as possible, as would be the case with washed or frozen red blood cells. If the patient requires platelets, which cannot be transfused without plasma, then the patient should be pretreated with antihistamines.

The more serious allergic reactions, such as anaphylaxis, may require the use of steroids and epinephrine if the status of the circulation deteriorates. The need for subsequent blood transfusions presents a difficult situation. If red blood cells are required, they can be washed prior to transfusion so as to remove most of the plasma. An even better alternative is transfusion of frozen red blood cells; prior to the freezing process the plasma is replaced by glycerol, and prior to transfusion the glycerol is replaced by normal saline. By whatever means, subsequent transfusions of red cells must be given with the least amount of plasma.

Platelets or white blood cells may be required by transfusion for patients who have previously had anaphylactic reactions to transfusions. Here, one must identify the patients who do not have IgA and who therefore will account for most instances of anaphylactic reactions. These patients have anti-IgA antibodies which will react with IgA from the donor, so these patients should receive blood only from donors who themselves lack IgA. The American Association of Blood Banks maintains a rare-blood donor file for such purposes. In all other individuals who need WBC or platelet transfusions and in whom the cause of such anaphylactic reactions is not known, the patients should be pretreated with steroids and the transfusion procedure monitored carefully.

Jan Rothman, MD (Assistant Chief Medical Resident): I will review immune-mediated hemolytic transfusion reactions, which can be immediate or delayed.[7]

The surface of the red blood cell is the site of from three hundred to four hundred antigenic determinants. The immediate immune hemolytic transfusion reactions are related to the presence on the red cell of certain antigenic determinants, for example, antigen A and B. We all produce alloantibodies in response to certain antigens that are not present on the surface of our own red blood cells. For example, if we belong to blood group A—ie, have antigen A on our own red blood cells, we produce anti-B IgM antibodies. If an individual receives donor red blood cells that have foreign antigenic determinants on their surfaces, these will react with antibodies in the recipient's serum (either IgG or IgM) which may have the ability to fix complement.

This adherence of antigen and antibody can result in immediate hemolysis and a febrile reaction within the first few minutes of starting a transfusion, or up to four to six hours after the patient has first received the blood. Fortunately, the incidence of this type of incompatible transfusion reaction is only one in 6,000 to one in 7,000 transfusions. More than 75% to 80% of such reactions are due to either technical or human error, and could be avoided.[8]

The symptoms precipitated by these reactions are variable—from mild to very severe, ranging from nausea and vomiting with febrile reactions, tachycardia, and tachypnea, to hypotension, disseminated intravascular coagulation, and acute renal failure with renal shutdown. The patient described here did not manifest any of these symptoms.

The delayed type of immune-mediated hemolytic transfusion reaction appears to be more relevant to this patient. Delayed transfusion reactions almost invariably occur in patients who have been previously sensitized, either through previous blood transfusions, or by fetal-maternal transmission of red cells during pregnancy.[9–12] This patient had previously received four units of blood several years earlier in association with prostate surgery.

Delayed hemolytic reactions characteristically occur within three to four days, or up to two to three weeks at most, after a transfusion. The symptoms, in contrast to those of an acute hemolytic reaction, are usually less severe, ranging from mild anemia, weakness, and mild jaundice, to more severe anemia with a fall in the hematocrit and with symptoms that correspond to the degree of anemia. Most of these reactions are undetected, so that the reported incidence of about one in 7,000 to 8,000 transfusions is probably a gross underestimation.

For a better understanding of the delayed hemolytic reaction, one needs to review the fundamental principles that underlie direct Coombs testing, which is commonly used to detect the potential for hemolysis. Human plasma contains certain proteins, in particular immunoglobulin G (IgG) or immunoglobulin M (IgM), which bind the third component of complement (C3), and, when directed against the host's own red cells, can sensitize them to subsequent hemolysis. Antisera against these immunoglobulin or complement components are obtained by injecting rabbits with human serum. The antiserum from the animal, also known as the Coombs reagent, is then purified. The red blood cells to be tested are washed to remove any impurities or contaminants on their surfaces, and are then incubated with the Coombs antiserum. Shortly thereafter, if the red cell surfaces are coated with IgG, C3, or both, then these protein substances will combine with their corresponding antibodies in the serum, causing a physical bridge between cells, with resulting agglutination of the red cells.

The IgG or IgM or both may not necessarily be found on the surface of one's own red blood cells but rather in the serum. Human serum suspected of containing IgG or IgM or both directed against a minor RBC antigen can be incubated with ABO- and Rh-compatible red blood cells. Subsequently, these red cells are exposed to the Coombs antiserum; if agglutination occurs, this indirect Coombs test is said to be positive.

A false negative direct Coombs reaction can ordinarily occur in three situations:

1. The IgG or C3 in question may not be present on the red blood cell in significant amounts; or there may be clumping of the IgG on the surface of the red blood cell.
2. Infrequently, if the washing of the red blood cells has not been adequate, certain contaminants, that is, other immunoglobulins, may prevent the antibodies in the animal antiserum from binding to the IgG or C3 on the surface of the red blood cell and agglutination will not occur.
3. A possible mechanism could be a steadily diminishing immune response by the recipient against the donor's red blood cells. Antibodies directed against the donor's red blood cells may be detected by the Coombs antiserum for the first several days after the transfusion, so that the direct Coombs test will be positive. However, as the donor's cells are progressively removed either by intravascular or extravascular hemolysis, the direct Coombs test will change from positive to negative.

The mechanism of delayed transfusion reactions appears to be the following. The patient has either been given a blood transfusion previously or has had a previous pregnancy. As a result, the recipient may develop an antibody in the serum to a certain antigenic determinant on the foreign red cells, but at such a low concentration as to escape detection during usual blood banking procedures. During subsequent transfusions, if the surfaces of the new donor red blood cells happen also to have the same antigenic determinant, then the recipient's plasma cells may respond by raising the level of antibodies against them. As a result, during the first several days to weeks after the transfusion, these antibodies will initiate a delayed hemolysis of the donor red blood cells. The direct Coombs test will become positive; but as the donor red blood cells are progressively removed from the recipient's serum by hemolysis or by the reticuloendothelial system, the direct Coombs test will revert to negative.

One would expect that in such a situation the indirect Coombs test would be significantly positive, that is, the antibody should be detected in the serum, if not in the first two or three weeks, then in the next several months, and thereafter would remain positive indefinitely.

A few reported cases suggest the possibility of a rare type of primary sensitization which may produce a transfusion reaction. A patient who has not received any prior transfusions may develop so-called alloantibodies against the donor's red blood cells, with a resulting hemolytic reaction seen anywhere from three to six weeks post-transfusion. This was roughly the time sequence in this patient. But again, one would expect that both the direct and the indirect Coombs test reactions would be significantly positive.

There are three basic mechanisms by which red blood cells are destroyed:

1. The donor's red blood cells are coated with IgG of various classes and IgM. In the case of acute hemolytic reactions due to ABO incompatibility, the antibody present in the recipient's serum has the ability to fix complement and therefore results in rapid hemolysis of the donor's red cells.

2. The donor's red blood cells are coated with IgG class I, II, or III, and are hemolyzed by extravascular means in the reticuloendothelial system (RES). Complement is present but is not the means by which the hemolysis is produced. IgG FC receptors in the RES system, particularly on the macrophages in the spleen, recognize the donor red blood cells and facilitate their removal from the circulation by the macrophages. The red cells thus acted on will appear either as microspherocytes, or will be completely disintegrated. The production of any one of these three results is a function of the type of antibody present, its quantity, and/or the presence of complement.
3. The so-called antibody-dependent cell cytotoxicity (ADCC) reaction is due to the coating of donor red blood cells with antibodies, normally IgG, together with the presence of T-lymphocytes in the recipient that have not been exposed or sensitized to these antibodies. These cells are able to act directly on the antibody-coated donor red blood cells to produce hemolysis.

The clinical detection of hemolysis is a function of four variables: the rate of red blood cell breakdown; the quantity of red blood cells broken down; the ability of the body, particularly the bone marrow, to compensate for the fall in hematocrit and hemoglobin; and the underlying condition of the patient. Rapid hemolysis is detected usually with ease, as the patients commonly develop impressive clinical symptoms. However, quite often there are more subtle manifestations, in which case one must rely on the laboratory to supply the essential diagnostic information.

During intravascular hemolysis, the red blood cells are rapidly broken down, and hemoglobin is liberated into the plasma. Initially, most of this hemoglobin is picked up by α_2-globulins, which include haptoglobin and hemopexin. These rapidly bind to hemoglobin, pass through the hepatic circulation, and are excreted by the liver. However, once the liver's capacity to synthesize these globulins is exceeded, there will be a fall in haptoglobin and hemopexin in the serum; in this circumstance, the excess hemoglobin in the plasma is not bound to them but is metabolized, initially to methemoglobin and finally to methemalbumin. Circulating hemoglobin in the plasma can be detected by centrifuging a sample of blood and looking for a change in color of the serum. Also, the urine can be tested for the presence of hemoglobin, methemoglobin, and hemosiderin.

Reliance on a fall in haptoglobin, though detectable by laboratory methods, is fraught with one difficulty. Haptoglobin is an acute-phase reactant, that is, it is synthesized in the liver in increased amounts as part of the acute response to inflammation, as with burns and infections. Thus, a normal or elevated level of haptoglobin certainly does not rule out hemolysis.

Extravascular hemolysis often overlaps with the intravascular type. As hemoglobin is metabolized in the reticuloendothelial system, it will be converted to biliverdin and to unconjugated bilirubin. The latter will be transported to the liver, where most of it will be converted to conjugated bilirubin, and then excreted in the bile. Once the converting capacity of the liver is overwhelmed by an excessive load of unconjugated bilirubin, the serum bilirubin level will rise—one of the earliest manifestations of the delayed transfusion reaction or of extravascular hemolysis. Also, in the presence of excess urobilinogen in the plasma, the liver will not be able to excrete it completely, and the excess will appear in the urine. Another relatively simple way to identify hemolysis is by review of the peripheral blood smear for the presence of spherocytes, reticulocytes, and polychromasia. The bone marrow biopsy may demonstrate erythroid hyperplasia.

Over the past several years, a heterogenous group of so-called innocent antibodies, identified in patients who have received previous transfusions, have been found quite often to interfere with the determination as to whether a transfusion reaction with hemolysis has taken place. These antibodies are IgG noncomplement-binding, normally of very low avidity, and usually of no clinical significance; certainly in this patient, I do not believe that they are the reason for the delayed hemolysis.

In order to prove definitively whether an antibody is implicated in producing hemolysis, the gold standard would be the identification of the antigen against which the antibody is directed. The method consists of obtaining red blood cells that present this antigen, labelling the antigen with radioisotope, injecting the red cells into the patient, and then determining their half-life. Their rapid removal from the patient's serum is fairly good evidence that the hemolysis is due to this antibody.

It is reasonable to conclude that the patient presented here did undergo hemolysis, because of the presence of spherocytes, the fall in the hematocrit, and the elevated serum lactate dehydrogenase. There are four possible reasons for hemolysis in this patient. Some are very remote and others more relevant:

1. *Congenital spherocytosis.* This condition usually declares itself at an early age and is normally accompanied by a family history of anemia and jaundice. The spherocytosis is persistent, as is splenomegaly. We should have been able to obtain such a history and to observe the clinical findings mentioned, if this patient had such a disorder.
2. *Drug reaction.* As we have mentioned, the patient was taking an anti-arrhythmic medication, procainamide, which is quite often associated with drug-induced autoimmune hemolytic anemia. Generally speaking, however, patients with this condition have a positive direct Coombs test, and the antibody in question can usually be easily identified as a panagglutinin, as opposed to an alloantibody. A drug-induced hemolytic anemia is usually accompanied by a gradual drop in the hematocrit; the drop seen in this patient was not gradual.
3. *Delayed transfusion reaction.* In spite of the likelihood of this cause, some compelling opposing evidence in this case must be mentioned. We would expect the hemolysis and the fall in hematocrit to occur within a period of two or three days post-transfusion, up to the first two weeks; this patient's drop in hematocrit did not take place until four or five weeks after transfusion. Similarly, we should have seen a negative direct Coombs test because of the rapid re-

moval of the affected red blood cells; but we might have anticipated a significantly positive indirect Coombs test, with the presence of an alloantibody which should be identifiable. The patient's indirect Coombs test was positive, perhaps due to one of these nuisance antibodies which we have already mentioned, but which I do not think was the reason for his hemolysis.

4. *Primary sensitization.* This is seen four to six weeks after an initial transfusion; but again the possibility is remote because one would expect, if not a direct positive Coombs test, at least an indirect positive one.

So, in the face of these possibilities, I will let the experts decide as to which one is the most likely.

Bruce Raphael, MD (Director of Hematology/Oncology): Whenever a patient develops an abnormality of his or her blood count during hospitalization, shortly after discharge, or following contact with a physician, we must immediately consider the possibility that some event in the hospital or some medical activity may have contributed to the abnormality. It is always helpful to have evidence that the patient did not have the abnormality previously, and then to review any events that occurred between that time and the appearance of the abnormality.

We had such evidence in this case; that is, prior to the cardiac surgery the patient's hemoglobin level was normal; drugs were added and he received transfusions during cardiac surgery; following the operation and for two weeks after discharge, his hematocrit was 30%. Then suddenly his hematocrit dropped. The examination of this patient does not indicate the cause of the hemolysis, so it is necessary to depend on laboratory data and intuition to determine a reasonable cause.

The only drug that this patient received for the first time that could have been responsible for the hemolysis was procainamide. Yet at the time of admission, his direct Coombs test was negative, and the indications of continuing hemolysis at that point were minimal, with an LDH of 250 U/L. The drug could be responsible, but one would not expect the hemolysis to diminish so markedly during a time when he was still continuing to take it. Therefore, we did not consider the drug to be the likely cause.

This patient had been given blood at the time of cardiac surgery. Blood transfusions are not always benign procedures; both immediate and delayed reactions can occur, such as delayed hemolysis as well as reactions of nonhemolytic varieties, ie, infection transmitted by the donor blood. There are antigens other than the ABO and Rh varieties on the surface of red blood cells. We do not cross-match for this host of minor antigens because they are not strongly antigenic and do not stimulate antibodies in most people. Prior transfusions, especially multiple transfusions, in a recipient who is an immune-responder will tend to generate such antibodies, and these in turn will later destroy any red blood cells that carry such antigens. Later cross-matchings may be made more difficult; the indirect Coombs test will tend to be positive. The blood from a donor who has such antigens should not be used for transfusion for such a patient.

Four weeks after his transfusions for bypass surgery, this patient's level of hemoglobin dropped; he reacted with a good bone marrow response and a good reticulocyte count. The marrow was morphologically normal. His reaction was not likely to be due to procainamide, and therefore we accepted the diagnosis of a delayed transfusion reaction. The comforting awareness that that diagnosis generates is that the patient will hemolyze only the donor red blood cells with which he was transfused. The process will be self-limited, and he will not hemolyze his own red cells. Consequently we waited. As expected, the process of hemolysis gradually terminated, and his marrow replaced the destroyed donor cells with his own.

John T. Flynn, MD (Associate Chief of Medicine): If this patient were to need a transfusion in the future, what would you expect to happen?

Dr Raphael: Ordinarily there would be no problem, because the indirect positive Coombs test would tell us which antibody and which antigen were involved. As long as the antibody is one that reacts with only one of various antigens which are well distributed among the white, black, and Oriental populations, the antigen could thus be identified and the patient transfused with blood that does not contain it.

This patient is unique in that we never detected what the antibody was against, so we do not know the corresponding antigen. Thus there is an excellent chance that his level of this same antibody will again rise following transfusions in the future, and once again this patient will hemolyze the transfused cells.

In following up on this patient, we should repeat the Coombs test in a month, hoping that the titer of the antibody will have increased in the serum and that we will be able to identify it and consequently its corresponding antigen. We can thus avoid giving him inappropriate donor blood in the future. But until we make such an identification, I would be very worried about transfusing him. And that is why we did not consider giving him more transfusions during his hospital stay here, in spite of his coronary artery disease and his hematocrit of 20%. We kept him on a regimen of bed rest and hoped he would replace the hemolyzed blood with his own, rather than run the risks of additional transfusions, which he might well hemolyze subsequently.

Harold Coppersmith, MD (Attending Physician): How good is a cross-match in a case like this?

Dr Raphael: A cross-match is actually an indirect Coombs test specifically against the donor's blood; yet an antibody against the donor cells may be present in the recipient's plasma but not detectable. Obviously, if you do not detect the antibody, you are not going to identify it.

Dr Flynn: Is there any chance that this patient would have a worse hemolytic reaction the next time?

Dr Raphael: Most IgG antibodies are removed by the recipient's reticuloendothelial system, and the reaction is not complement-mediated. Therefore, the patient in this situation is not likely to develop rapid hemolysis with renal failure and disseminated intravascular coagulation (DIC), such as would characterize a mismatch. It is likely that a second reaction after a future transfusion would be of the same kind as this one, and that he would hemolyze only the donor red blood cells. Such a reaction might be undetected clinically, except for the appearance of mild jaundice or angina pectoris because of the decrease in his hemoglobin. It is unlikely that he would develop a high fever, discolored urine, or other more severe symptoms.

Ann S-Y. Chan, MD (Director of Blood Bank): On the first blood specimen which we received from this patient, the blood was typed as group B Rh positive; the direct and indirect Coombs tests were negative. Once alerted to the possibility of a delayed hemolytic reaction, we repeated our tests, and found that the patient had a very weakly positive indirect Coombs test. This meant that some type of antibody was present in his serum, but was not coating his own red blood cells.

The one unit of blood that the patient received in this hospital was cross-matched as compatible; the patient received that unit, he had no reaction, and his hemoglobin level rose 1 g/dL. This suggests that the unit of blood that was given to him during his hospital admission here survived normally, at least temporarily. If he had hemolyzed that unit of blood subsequently, he should have had a decrease in his hematocrit and increase in his bilirubin and serum LDH levels.

A specimen was sent to the New York Blood Center for special studies. One of the items in which we were interested was whether there were any surviving donor cells from the blood transfusions given during coronary bypass surgery. This was determined by a method called differential agglutination. The principle is based on the fact that when the patient is typed for the minor blood groups (usually the M N system), one will see more than one population of donor cells present. Only one homogenous population will be identified if only the patient's own cells are circulating. This test showed that there were no donor cells still in the patient's circulation when he was admitted here. So essentially he had hemolyzed all the four units of blood that he received at the medical center, during the period when his hematocrit fell from 30% to 18%. When he entered the hospital here, the hemolytic process had ended. That explains why the direct Coombs test was negative at the time of his admission here, because there were no more donor cells left. However, it is very unfortunate that we are not able to identify the antibody that caused the hemolysis. Whatever it was, it must have appeared and disappeared very rapidly, which is unusual. Rare instances have been reported in which the patient had evidence of hemolysis as shown by chromium-tagged red cell survival studies, yet an antibody could not be detected in the patient's serum.

The time sequence of the hemolysis in this patient is very unusual. This might be thought to be an anamnestic response, initiated originally by the blood given at the time of prostate surgery and subsequently restimulated by the blood given at the time of cardiac surgery. But if that were so, then the hemolysis should have occurred much earlier, probably within one or two weeks after the surgery, rather than three to four weeks later. The possibility of a hemolytic transfusion reaction due to a primary antibody response has been considered, but so far there have been no such cases reported that are clear-cut. Usually when the patient has a primary response to donor red blood cell antigens, it seems to occur four to six weeks after the transfusion. The donor cells give evidence of diminished survival, but this condition is not manifest clinically.

The most potent blood group antigen besides the ABO system is Rh O (D). We have transfused Rh-negative patients with Rh-positive blood when the demand for Rh-negative blood exceeds the supply, and this procedure has not been followed by frank delayed hemolytic transfusion reactions. Anti-D antibody may appear two to five months after the transfusion, and the direct Coombs test may become positive, but there is no frank hemolysis.

With this patient, we were not able to identify the antibody; the one which we found proved to be a red herring. This antibody belonged to the high-titre low-avidity group, most probably anti-McCoy. Anti-McCoy antibodies have been shown by various authors not to cause hemolytic transfusion reactions, and not to affect red blood cell survival as evidenced by chromium-tagged red cell studies. Patients who already have anti-McCoy antibodies have been given McCoy-positive blood transfusions, and the donor red cells survived. So I would not consider this class of antibodies as the cause of this patient's hemolysis. We really cannot predict this patient's future as a transfusion recipient. Perhaps in a few weeks, the antibody responsible may appear in the patient's serum; but it may never appear. Some time in the future, he may have another delayed reaction to a transfusion, if the specific antigen is involved and if he mounts an anamnestic response. We will need a fresh blood specimen from him before every future transfusion.

CLINICAL SUMMARY

A 75-year-old man, who had been given transfusions in connection with prostate surgery four years earlier, underwent recent coronary bypass surgery during which he received blood transfusions; three to four weeks later, hemolytic anemia developed.

In this conference, the varieties of reactions to blood transfusions are reviewed; it is believed that this patient's hemolytic anemia was due to a delayed immune reaction against the donor's red blood cells.

FINAL CLINICAL DIAGNOSES

1. Delayed hemolytic transfusion reaction, with destruction of donor red blood cells following coronary bypass surgery.
2. Atherosclerotic cardiovascular disease. Coronary artery disease. Angina pectoris. Status-post coronary bypass surgery (three vessels).
3. Status-post transurethral prostatectomy four years earlier, involving blood transfusion.

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A FURTHER CONTRIBUTION TO THE SUBJECT OF BLOOD TRANSFUSION: DIRECT METHOD

The crude study of anatomy and surgery by [Galen and Vesalius and Paré and others] laid deep and wide the foundation for future investigation, and upon this foundation rested the masterly work of Harvey, who explained to the world more fully the nature of the circulation. That was in 1628. About fifty years more was now consumed in experimental work upon animals. Then came the first transfusion upon the human subject. From time to time many more attempts at blood transfusion were made with asserted good results; but our present understanding of the composite character of the blood would lead us to doubt the accomplishment of anything definite by these early experimenters. Nevertheless, their work was an inspiration to the generations which followed them. They tried to exclude air from the vessels, and their technique was intended to prevent clotting of the blood on its way from the donor to the recipient. All their methods, however, required an intermediate structure, usually some form of tubing. Death was not uncommon from emboli, or the entrance of air into a vein. A careful search of the literature, therefore, would reveal many failures. Two hundred years after the first transfusion there was still no improvement in technique the apparatus of Aveling being the one of choice. With this instrument and its application we are all familiar. Transfusion was done from vein to vein. Referring to the American Textbook of Surgery, about this time, we find the subject dismissed in very few words, preference being given to the use of the normal salt solution. Ten years more brings us down to the present time. During the last half of this decade, blood transfusion has acquired a new impetus, so that more importance is attached to the efficacy of blood to-day than ever before in the history of surgery, and the manner of transfusing it has been completely revolutionized. Blood is now carried from artery to vein, instead of from vein to vein; and if carefully done, under proper indications, transfusion is no longer a doubtful procedure. . . .

We have already come to know a great deal about the effect of one blood upon another, and the tendency toward incompatibility and haemolysis. Before transfusion, the percentage of haemoglobin in the patient is usually low and the number of red blood cells is diminished. The whites are variable. After transfusion, the erythrocytes and the percentage of haemoglobin are both augmented but not always to a readily perceptible degree. The patient seems to be vitalized from the start, but the actual blood findings are not commensurate with the favorable clinical manifestations. A little later, however, there is evidence of greater improvement in the blood constituents than could have been realized, had the ordinary treatment of anaemia been carried out. Eight to twelve ounces of blood is a safe quantity to be transfused at any one time. The donor is not in any particular danger, but the donee may be overcharged to the extent of causing dilatation of the right ventricle. He should be kept under constant surveillance. Unusual irritability, or respiratory embarrassment, or marked sudden change in the character of the pulse, are indications of approaching danger. When we consider the rate at which the blood circulates, it is evident that the newly transfused blood must immediately fall into line with the rapidly moving current, and deliver to the tissues its portion of nutrient material. If this does not occur there is danger of incompatibility and the transfused blood becomes a foreign body. That haemolysis does result in a small number of cases should remind us of the desirability of making the necessary preliminary tests to determine whether the blood to be transfused is physiologically acceptable. . . .

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(*NY State J Med* 1911; 11:313-316)

Health and health care in Nepal

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Nepal's system of health delivery can be traced to 1951 when the country was liberated and opened to the outside. Nepal is illustrative of the classic case study of a developing nation with limited financial resources to correct a multitude of major health problems including widespread malnutrition, low life expectancy, high mortality of children under the age of five, and limited access to clean water. While Nepal's health statistics have shown a gradual improvement, they still rank among the poorest in the world. In addition to financial exigencies, the problem is compounded by understaffed and inadequately trained personnel, limited resources, poor transportation, poor communication networks, widespread illiteracy, a high population growth rate, and geographical constraints (including an agrarian base). This paper describes some of the problems and presents an analysis of Nepal's attempts to deliver "Health for All by the Year 2000."

BACKGROUND

The travelogue image of Nepal is of an Asian Switzerland; the reality is quite different. With an infant mortality rate of 111.53/1,000 live births, maternal mortality of 8.53/1,000 live births, and life expectancy of 52.88 years for males and 50.10 years for females,[1] Nepal presents a far from serene and sanitary alpine landscape.

In a developing nation how does one begin to address the health problems that exist? How does one manufacture resources to bring health services to a population living in the ninth century in 1951? These questions faced health planners in Nepal's Ministry of Health (MOH) and have not yet been resolved.

Nepal is not a typical developing nation; it has had neither the burdens nor the benefits of a colonial experience. It has had no recent wars, is the only Hindu monarchy in the world, and is a landlocked country with geographical extremes. Nepal has also attracted a disproportionate amount of Western donor attention. For example, the United States Agency for International Development (USAID) over an eight-year period has spent $37 million in direct government-to-government aid (personal communication, David Calder, Director of USAID, Kathmandu, Nepal, November 19, 1987). The reasons for this large amount of Western donor aid are multiple and include political factors as well as the attractiveness of the land, climate, and people.

Like other developing nations, Nepal's statistics reveal the extent of the problems it faces. The per capita gross national product in 1984 was $160 compared to an average gross national product per capita of $258 for South Asia and $844 for Southeast Asia.[2] According to Nepal's National Planning Commission's estimates, 40% of Nepalese live below the poverty line.[3]

More than 90% of the Nepalese live in rural areas and about 93% of the people earn their livelihood from the land.[3] The overall literacy rate in 1981 was 23.5%—34.9% for males and 11.5% for females[4]; however, in rural areas, the discrepancy is even greater.[5] Nepal's heavy investment in primary schools should begin to address both the low literacy rate and the male/female inequity in ensuing years.

Nepal is ruled by King Birendra—a direct descendant of the Shah family dynasty. It was his grandfather, King Tribhuvan, who regained power in 1951 from the Rana family (who although only serving as prime ministers actually ruled Nepal for almost two centuries).[6] When the Shah family regained control, they "opened" Nepal to foreigners. Although the system of government remains a constitutional monarchy, the king introduced a *Panchayat* democracy in 1962 which was reaffirmed by a national referendum on May 2, 1980. The *National Panchayat* is a nonpolitical (one-party system) organization, with a pyramidal structure from the village and town *panchayats* at the base, to the *Rashtriya Panchayat* at the apex.[6]

For administrative convenience, Nepal is divided into five development regions, 14 administrative zones, 75 districts, and 3,866 village *panchayats*, each comprising several to many village communities. A majority of the 75 districts have less than 50 village *panchayats* and only six districts have more than 100 village *panchayats*. There is an average of 11 village *panchayats* to a district in the Inner (trans) Himalayan region, 33 in the Terai, and 55 in the heavily populated midland.[6]

NEPAL'S VITAL STATISTICS

The key to understanding the immensity of Nepal's health problems lies in its vital statistics, which reveal that major intervention is necessary in order to effect any change in the Nepalese health picture.

Countries of Southeast Asia have an average of 69 deaths per 1,000 live births, compared with a conservative estimate for Nepal of 206 deaths per 1,000,[7] one of the

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world's highest. It is worth noting that health indices figures supplied by His Majesty's Government (HMG) usually are unreliable as precise indicators. When they err, it is on the side of underreporting.[8] For example, the statistical problem relative to accurate data on infant and maternal mortality is compounded by the fact that in Nepal fewer than 5% of total deliveries occur in health institutions,[3] making data collection difficult.

Nepal's population is estimated to be about 16 million and is increasing at an annual rate of 2.66%. In 1984–1985 the crude birth rate was 41.57/1,000,[1] with "no evidence of a decreasing trend in fertility."[7] The fertility rate has been estimated at 6.1–6.3 (personal communication, David Calder, Kathmandu, Nepal, November 19, 1987). The life expectancy at birth for 1984 was 52.8 for males and 50.10 for females, down from 1981 expectancy rates of 54.98 and 51.20 for males and females, respectively.[1,5] These data reveal two rare phenomena: a lower life expectancy for females—which most likely reflects the astounding burden of work females assume and their excess fertility and subsequent mortality; and a decreasing rate of life expectancy—a reversal of worldwide trends.

Nepal had a crude death rate in 1984–1985 of 16.57/1,000 population with communicable diseases as the main causes of mortality (Table I). The main causes of morbidity after complications of pregnancy are also the communicable diseases.[9]

In 1985–1986 a range of immunizations were given covering approximately 27% of the population for oral polio vaccine 3 (OPV3) to 87% for tetanus toxoid 1 (TT1), with other immunizations (bacillus Calmette-Guerin, diphtheria, diphtheria 3, oral polio vaccine 1, measles, tetanus toxoid 2) ranging in between.[9] Deaths from diarrhea among children less than five years of age accounted for 46% of the deaths.[10] That figure is not surprising when one considers that until 1980 only 10.9% of the population had a piped water supply, and in rural areas only 5.2% had access to potable drinking water.[3] Therefore the majority of Nepalese were deprived of safe drinking water. Additionally, there was almost no system of sanitation; estimates in 1985 revealed that only 17.2% of urban and 0.2% of rural dwellers were served by sanitation facilities, accounting for a total of 1.7% of Nepal's population.[11]

Another priority health problem for Nepal is the widespread undernutrition and malnutrition of the population. In 1980 Nepalese were getting only 86% of their caloric intake requirements and the per capita protein intake expressed in grams per day was 45% of requirements.[12] It was estimated that the proportion of children under five years of age who were adequately nourished was only 7% in 1985.[9] For the country as a whole, only 29% qualified as being at a normal nutritional level in 1975.[13]

TABLE I. Leading Causes of Death in Nepal, 1984–1985,[9] in Descending Order of Prevalence*

Other symptoms and ill-defined conditions
Tuberculosis, all types
Bronchitis, emphysema, and asthma
Fever of unknown origin
Gastritis and duodenitis
Enteritis and other diarrheal diseases
Pneumonia
Other injuries
Cirrhosis of the liver
Bacillary dysentery and amoebiasis

* Ten leading cause groups of mortality reported by 15 health posts, Kaski District, 1984–1985, which accounted for 72% of the total number of deaths reported.

These health indicators present the extent of the problems facing Nepal's Ministry of Health (MOH). The major problems requiring intervention fall into several areas: maternal and child health, including reduction of infant mortality, total fertility, and maternal deaths due to pregnancy; communicable disease, including reduction in the incidence of tuberculosis, Hansen disease, malaria, measles, tetanus, diphtheria, and childhood diarrhea; and malnutrition, including reduction of protein-energy deficiency.

However, intervention in those targeted areas will not be sufficient unless it is integrated with improvements in water and sanitation, improvements which will necessitate massive education of the population in order to be effective. For example, in most villages located in the hill country, houses are built near the bottom of the hill. The water supply is located further up the hill. Above that is the pit latrine. These practices have existed for generations because of longstanding ignorance of the realities of sanitation.

NEPAL'S SYSTEM OF HEALTH SERVICE

Nepal's health delivery system basically started from nothing, with weak local resource mobilization, in the 1950s. Nepalese health officials created a series of five-year plans beginning in 1956; they are now in the seventh, 1985–1990. The thrust of each of the five-year plans can be seen in Table II.

The provision of basic health services was identified as one of the minimum needs of the Nepalese. The MOH, in its early five-year plans, designed an implementation plan

TABLE II. Summary of Nepal's Five-Year Plans

First (1956–1961) and Second (1963–1965) Five-Year Plans
- Attention focused on programs related to preventive health, eg, malaria eradication, smallpox vaccination, leprosy and tuberculosis project.
- Hospital construction emphasized.

Third (1965–1970) Five-Year Plan
- Beginning attempts towards a comprehensive health plan with proper categorization of different activities as curative, preventative, training, and miscellaneous.
- Introduced family planning and maternal and child health projects.

Fourth (1970–1975) Five-Year Plan
- Priority extended to preventive services.
- Strengthening of existing curative services.
- Introduction of middle level health workers.
- Community Health and Integration Division set up in the Department of Health Services.

Fifth (1975–1980) Five-Year Plan
- Clearly defined objectives, policies, priorities, targets and details regarding the development of the health sector.
- Targeted the expansion of facilities for the rural population and an emphasis on village level basic health care activities.

Sixth (1980–1985) Five-Year Plan
- Set higher goals including increasing life expectancy by reducing the death rate, preventing infection through preventive services, controlling birth rates, improving nutritional level and environmental hygiene, and developing basic health services at village and community level.

which included models of operation for the 75 districts, approximately 4,000 *panchayats*, and the tens of thousands of village communities (as well as for nongovernmental organizations (NGOs) and the private sector).

The early plans called for the establishment of three levels for the delivery of service: physician-staffed hospitals and health clinics located in districts, health posts in the *panchayats*, and individual health workers at the community village level. In addition, separate projects were begun which aimed at vaccination, malaria eradication, leprosy and tuberculosis control, family planning, and maternal and child health (begun in the third plan, 1965). Some of these projects were run under the auspices of the MOH, and some were set up and run by various NGOs (for example, USAID, Japanese Organization for International Co-operation in Family Planning, Save the Children).

The major development of the heretofore limited medical infrastructure began after initiation of the first five-year plan. Table III presents a comparison of facilities prior to 1956 and in 1987.

Although the first two five-year plans included primary health care targets, it soon became apparent that primary health care in a vacuum could not solve Nepal's health problems. Thus the third five-year plan marked the beginning of attempts towards comprehensive health planning which integrated many previously isolated health oriented projects. It was aptly named the Integrated Community Health Services Development Project (ICHSDP).

These efforts were unfortunately insufficient to change Nepal's depressing health statistics. Despite significant improvement, Nepal's health statistics continue to remain among the world's worst.

Nepal has committed itself to trying to achieve the goals of the World Health Organization's (WHO) "Health for All by the Year 2000" (HFA/2000). In 1979, WHO issued guiding principles for formulating strategies for achieving health for all by the year 2000, which stressed primary health care and focused on the development of the country's health system's infrastructure in order to deliver programs that reached the entire population.

Nepal presented its first plan, "Planning for Meeting the Basic Minimum Needs of the People Between 1980–2000," to WHO in December 1979, and made subsequent revisions in March 1981 and May 1987. Policy and strategy for meeting WHO's basic minimum needs were also reflected and elaborated in the documents *The Long Term Health Plan* (*1975–1990*) and *The Seventh* (*Five-Year*) *Plan* (*1985–1990*).

The key strategies for the health sector in the seventh plan include stressing primary health care, providing appropriate manpower, developing and integrating Ayervedic (herbal medicine) services, improving nutrition, encouraging private sector growth of health services, and intensifying family planning programs to reduce the birth rate.

Severely hampering achievement have been the constraints of geography and population distribution, shortages of MOH manpower and trained personnel, lack of effective coordination between and within the MOH, inadequate infrastructure, and limited financial resources. However, it is important to remember that prior to the mid-1950s, traditional healers (spiritualists and herbal medicine practitioners) provided all the medical care in the country.

TABLE III. Health Resources in Nepal

Resource	Time Period Prior to 1956[3]	 April 1987*
Hospitals	34 (with 625 beds)	91 (with 3,842 beds†)
Dispensaries/health posts	24	814
Ayervedic clinics	63	145
Physicians	50	600–850‡

* Source: His Majesty's Government/Ministry of Health, Health Planning Division, Health Information Bulletin 2 (3), 1987.

† 50% of health services are confined to Kathmandu Valley. Includes hospitals under the Ministry of Health (66) and under other government agencies and nongovernment agencies.

‡ Personal communication, Dr B. B. Karki, Kathmandu, Nepal, November 19, 20, 1987.

HEALTH SERVICE INFRASTRUCTURE

Ninety-two percent of the population of Nepal live in rural areas, many of which are not serviced by roads; in 1984, there were only 5,717 kilometers of roads in all of Nepal. Recognizing the problem of population distribution, the MOH based its pyramidal infrastructure on volunteers termed "community health leaders." Use of volunteers as the first line of service began in 1980, as part of a project established by ICHSDP. One person per ward, preferably someone able to read and a stable community member, is selected as the community health leader, and given a minimal training program of approximately 24 days. The community health leaders work at the ward level of the village *panchayat* on a voluntary, nonremunerative basis to help local people improve their health conditions through mutual help. These Nepalese are mobile workers who travel to individual village homes to provide preventive health education, eg, how to build pit latrines. They also provide some basic primary health care. A few medications, (malaria kits, aspirin, cough syrup, etc) are also provided to enhance the community health leader's motivation and status within the "pill oriented" community (personal communication, Richard Coffey, Peace Corps health worker, Nepal, 1984–1987, New Paltz, March 8, 1988). A job description of the community health leader is presented in Table IV. The goal of the MOH for the year 2000 is to have one health volunteer per 500 population.

The next tier is filled by salaried government employees called village health workers (or *panchayat*-based health workers), who travel to the villages and also work out of health posts. Their job requirements involve spending four to six days per month at the health post doing paperwork and the remainder of the month in the field visiting every house in their *vek* (an administrative subdivision of the district). This latter requirement can be an unreasonable expectation in certain *veks* given geographical constraints (personal communication, Richard Coffey, Peace Corps health worker, Nepal, 1984–1987, New Paltz, March 8, 1988). The MOH hopes to provide each of the 3,866 *panchayats* with a *panchayat*-based health worker (eighth grade education plus three months of training), who will,

TABLE IV. Job Description of the Community Health Leader, Nepal*

1. Enlists cooperation of the ward people in all the health activities carried out by the health post to make them more effective.
2. Provides treatment in case of minor ailments with medicines available with him.
3. Educates people and especially pregnant and lactating women about the health care necessary for such women.
4. Assists in the conduct of any immunization program and educates people about immunization.
5. Teaches the people ways and means to prevent diarrhea.
6. Teaches the villagers the importance of a mixed diet (food prepared from various ingredients) and also teaches them preparation techniques.
7. Educates the people of the ward about family planning methods.
8. Continues clean-up campaigns in the ward.
9. Educates the people about preventive and containment measures for communicable diseases. In case of an outbreak, takes necessary containment measures and helps those affected by the disease to obtain necessary treatment.
10. Tries to bring about radical behavioral changes in people to improve their health status.
11. Makes people aware of the traditional home remedies which are proven to be beneficial.

* Source: *Community Health Leader Training-Cum-Working Manual.* Nepal, Community Health and Integration Project, 1982.

among other duties, provide family planning advice, information on nutrition, and immunization services for children (personal communication, Dr B. B. Karki, Public Health Officer, Nepal Ministry of Health, Kathmandu, Nepal, November 19, 20, 1987). This far reaching system will continue to be essentially free to all Nepalese.

The health posts are the most peripheral units and they provide both static and mobile basic health services. Although there are currently 744 health posts, only approximately 450 are delivering integrated community health services and operating at full efficiency.[9] It is the aim of the MOH to have nine fully staffed health posts in every one of Nepal's 75 districts (for a total of 675 health posts throughout Nepal), thus reducing the distance between the most peripheral health institution and the community.

The health posts will be managed by a health assistant who has received a certificate from the Institute of Medicine, indicating three and a half years of study past the tenth grade. This includes one year of basic science, plus one and a half years of specialization. Four auxiliary workers assigned to each post will include two auxiliary nurse midwives and two auxiliary health workers, the last requiring a one-year course of study past tenth grade at the Institute of Medicine.

Health clinics, which were established in the first five-year plan and which are distinguished from health posts because they are manned by physicians, are being phased out. This reflects the limited number of doctors in Nepal.

District hospitals are the next level of patient contact and referral. The MOH's goal is to have one hospital for each of the 75 districts. Currently there are 18 districts without any hospitals, and 26 districts without hospitals under MOH jurisdiction. There are four types of hospitals under the MOH: district, zonal, regional, and central hospitals. Hospitals under other governmental agencies and NGOs are located in 15 districts and include one hospital of the Institute of Medicine (the teaching hospital) located in Kathmandu, eight under the Royal Army Police, one private hospital located in Kathmandu, six nongovernmental, and nine mission hospitals. The total number of beds for all hospitals in Nepal is 3,842, serving a population of 16 million people. Services at hospitals under the MOH are free, but patients must pay for drugs and pay for or provide their own food.

Within the MOH there is an interface with one set of traditional healers, the Ayervedic practitioners, who constitute a parallel health infrastructure. Ayervedic practitioners follow a theory of medicine based on the Gyu Shi, four ancient treatises. They are viewed as operating midway between modern 20th century medicine and the *Jhankris*, Nepalese spiritualists. Ayervedic practitioners believe that illness is caused by an imbalance of the three humors (phlegm, wind, and bile) and work to restore the lost equilibrium, mainly with herbal medicines. The group of traditional healers not represented within the MOH are the *Jhankris*. It has been conservatively estimated that they number at least 400,000 practitioners,[14] or an average of 7.5 *Jhankris* per village (approximately one practitioner for every 40 people). Their fees range from one cent (25 paisa) to ten cents (2 rupees) per contact, although a large cash gift is customary for a "complete cure."

The remaining pyramidal tiers are occupied by the central MOH bureaucracy which provides the logistical support for the in-field health services, as well as acting as a planning and coordinating resource.

Over the years since the inception of the first five-year plan, several fiefdoms have developed within the MOH. These were the outgrowth of specific targeted projects and included ICHSDP, Family Planning and Maternal and Child Health Project (FP/MCH), Leprosy Control Project, Nepal Malaria Eradication Organization, Expanded Immunization Project, Prevention of Blindness Project, and the Tuberculosis Control Project. Most of these projects received large amounts of outside donor funding, amounting to twice the funds allocated by the government,[9] which tended to make the projects separate polities, and gave those outside agencies a disproportionate amount of influence in the project's operation. Because each sector acted independently, there was a lack of coordination between and within the different sectors[9] and also intense competition for limited resources.

A major recent reorganization of the MOH has occurred in order to meet the HFA/2000 goals (July 16, 1987). As a result of the reorganization, ICHSDP no longer exists. Instead there is now a Public Health Division, which is responsible for all primary health care activity as well as having authority over many of the heretofore separate projects. Ultimately this reorganization should achieve one system wherein all districts will be treated equally, and there will no longer be separate special projects operating partially outside the Ministry of Health and receiving special attention and financial resources.

In a recent interview, one high official within the MOH made ominous statements to the author about the reorganization. He suggested that it represented merely a bureaucratic "sleight of the hand," a paper shuffle reorganization in which the old system was essentially left intact. "The same people are still doing the same jobs," was his caustic comment.

FINANCING NEPAL'S GOAL OF HEALTH FOR ALL BY THE YEAR 2000

The Nepalese government has set very ambitious levels of health status indicators (Table V) that it must meet and has committed itself to attaining the goals of HFA/2000 through primary health care. The program initiated by the MOH has as its aim the delivery of free basic health services to Nepalese throughout the country. However, there have been many studies indicating that Nepal does not have the financial resources for achieving these goals. Understandably there is concern about the long term financial base to sustain health services in Nepal,[15,16] which are proving very expensive.

Currently, health services are financed through public revenues, household expenditures, and donor funding. As mentioned earlier, non-WHO external donor funding amounted to about twice the funds allocated by the government.[9] More than 55% of the MOH's development budget is from foreign assistance.[15] Reliance on this sort of funding is problematic and provides an unstable base upon which to make major future health decisions.

As for household expenditures, in a survey conducted in 1976 in four districts of Nepal, it was estimated that Nepalese spent out-of-pocket between 15 and 40 rupees per person per year for health care services and supplies, representing at that time about 3% of the average annual per capita income.[17] *Jhankris* account for one significant household expenditure, with fees either in cash or in-kind, while retail purchases of pharmaceuticals (many of limited therapeutic value) account for another.

It is foreseen that in the future, a convergence of rising incomes, increased urbanization, and successful public health information campaigns will result in higher expectations for more and better health care from the government. This will, in turn, require even more of a financial commitment on the part of the government.

At this time, the per capita public expenditure for health is lower in Nepal ($1.63) than in the other nations of Southeast Asia ($4.12). Government allotments to the health sector account for slightly over 5% (5.16% in 1985–1986) of the total government budget.[9]

TABLE V. Targets for Health for All Nepalese by the Year 2000*

Reduction of the infant mortality rate from the present level of 111.50 per 1,000 live births to 45 per 1,000 by the year 2000.
Reduction of the population growth rate to less than 2% per year by the year 2000.
Life expectancy to be raised from 51 years to 65 years by 2000.
Reduction in the incidence of communicable diseases (tuberculosis, leprosy, malaria, measles, tetanus, and diphtheria) by the year 2000.
Reduction of the number of people suffering from protein-energy deficiency to 1–2 per 1,000 by the year 2000.
Reduction of deaths due to diarrhea, especially among children.
Increased coverage of the population by government health services and nongovernmental community health services to include the following:
  ratio of doctors and other trained personnel (health assistants, auxiliary health workers, etc) to population will be on average 1/3000;
  one health volunteer per 500 population;
  gradually reduce the distance between the most peripheral health institution and the community.

* Source: His Majesty's Government/National Planning Commission, Central Bureau of Statistics, Basic Minimum Needs, May 1987.[9]

TABLE VI. Nepalese Health Sector Allocations by Functions,[15] Fiscal Year 1986–1987

Function	1986–1987 Amount	Percent
Primary health care*	$404,622,000	64.43
Secondary and tertiary care†	125,313,700	19.95
Central administration	49,861,000	7.94
Total Ministry of Health budget	579,796,700	
Ministry of Education allotments of health training‡	45,085,000	7.18
Royal Drug Ltd.§	3,118,000	0.50
Total	$628,000,200	100.00

* Includes health post services and project level activities such as EPI, ICHSDP, FP/MCH, leprosy, tuberculosis, nutrition, goiter, rabies, malaria control program, and health education.

† Includes all hospitals, health centers, pharmacies, and health laboratories.

‡ Ministry of Education budget allocations to health-related teaching facilities.

§ Ministry of Forestry budget allocation to pharmaceutical production.

The MOH allocates more than 65% of its public funds to primary health care services (Table VI). However, less than 40% of these funds are aimed at field-level primary health care services.[15] This amount might actually be lower because a substantial amount of primary health care funds (42.7%) is spent on central administration support tasks, and there is a difference between planned and actual expenditures. (There is a consistent pattern of underspending of the health budget because of lack of manpower or materials.[18]) More than 16% of the MOH's budget is used to support recurrent costs (fuel, drugs, vehicle and building maintenance) of the health posts. In a recently published report, Daly[15] addressed the problem of health financing and made several major recommendations to improve health services with the limited funds available. Whether they will be implemented is questionable. In summary, it seems apparent that the MOH is operating with a very limited budget which is being thinly spread and is not reaching a large proportion of the population.

ISSUES

There is an unbalanced distribution of health services between urban and rural areas, which mitigates against access. For example, at least 60% of Nepal's physicians are in the Bagmati zone (which includes Kathmandu, the capital), but only 13% of the population live in this area.[18] Dr B. B. Karki has estimated the number of physicians in the Kathmandu area to be about 600 to 860 of the approximately 1,000 doctors in Nepal (personal communication, Kathmandu, Nepal, November 19, 20, 1987).

The ambitious plan to have 675 fully staffed health posts throughout Nepal is laudable until one analyzes what is meant by "fully staffed." The senior administrator of the health post is the health assistant. His one and a half years of specialized training beyond the first year of basic science education consists of one half year of field work and six courses in health education, including introduction to health and disease, specific health problems and health services, foundations of health education, applied health education, introduction to sanitation, and special sanitation problems (personal communication, Dr Yogendra Pradhananga, Department of Community Medicine, Institute of Medicine, Kathmandu, Nepal, November 27, 1987). This training does not adequately prepare health workers to handle health problems. It is precisely this lack of training which results in the leading cause of death being reported as "other symptoms, signs, and ill defined conditions" (Table I).

It must be remembered, however, that 80% of health problems are minor, self-limiting, and easily treatable by simple intervention (personal communication, Dr B. B. Karki, Kathmandu, Nepal, November 19, 20, 1987). For example, giving oral rehydration to children with diarrheal problems is a function that can be performed by village *Panchayat*-based health workers who can also facilitate vaccinations, encourage family planning, and teach sanitary improvements. Further, they can recognize diseases which need to be referred to a physician and identify people for such functions as initial tuberculosis screening.

Because Nepalese are so poor, they have a limited capacity to pay for health services and thus are reluctant to spend time or money on preventive health care, especially when it involves travel time to a distant health post. Additionally, since Nepalese do not perceive diarrhea, tetanus, measles, or frequent pregnancy to be serious problems, they delay seeking treatment to a point where preventive intervention is no longer effective (personal communication, Richard Coffey, New Paltz, March 8, 1988).

Access to convenient medical facilities and trained personnel alone will not remedy Nepal's health problems. What is needed is a simultaneous assault on environmental and social issues. While family planning or fertility control is very important, it will not be a major factor in reducing the birth rate by itself. Blum[19] states that fertility control will follow "particularly when there is clear cut evidence of improving survival of children and infants." To improve survival requires improvements in mass sanitation and assuring a safe water supply.

Time will tell how successful the Nepalese will be in fulfilling ambitious goals against difficult odds.

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Trends in American gynecology, 1800–1910: A short history

JOHN S. HALLER, JR, PHD

American gynecological surgery began in 1809 when Ephraim McDowell, MD (1771–1830), of Danville, Kentucky, removed a 22-pound ovarian tumor without anesthetic. His 47-year-old patient, Jane Todd Crawford of Greene County, had ridden on horseback 60 miles to Danville for the operation, which was performed while the patient sang hymns and a mob of irate neighbors threatened to storm McDowell's home for attempting such surgery. McDowell's successful ovariotomy was followed by 13 other operations, with eight recoveries. Subsequent ovariotomies were performed by Nathan Smith, MD, of Yale, in 1821, Alban G. Smith, MD, of Kentucky, in 1823, David L. Rogers, MD, of New York, in 1829, and J. Bellinger, MD, in 1835. By the 1840s, ovariotomies were frequently performed although sepsis caused certain risks. Washington L. Atlee, MD, of Lancaster, Pennsylvania, reported in a paper in 1855 that 17 of the first 30 patients had recovered. By the end of the century, the mortality for ovariotomies had decreased to 10–12%.[1-7(pp 421-423),8,9]

Landmarks in the history of gynecology included the establishment of the first gynecological clinic by G. S. Bedford, MD, in 1841 in New York; the founding of the Woman's Hospital of New York in 1855 by J. Marion Sims; and the contributions of Addis Emmet, MD, whose operations for lacerated cervix, rectocele, cystocele, rectovaginal fistula, and laceration and prolapse of the urethra placed him second only to Sims in American gynecology. Other significant developments included the founding of the *Journal of the Gynecological Society of Boston* and the *American Journal of Obstetrics and Diseases of Women and Children* in 1869; the organization of the American Gynecological Society; T. Gaillard Thomas' removal of an ovarian cyst per vaginam in 1870, the work of Emil Noeggarath on gonorrhea, and Lawson Tait's removal of suppurating oviducts in 1872. From the 1870s onward, antisepsis via Lister's carbolic method had reduced the risk of abdominal operations and, by the end of the century, vaginal hysterectomies (introduced by Lane, Fenger, and Henrotin) and abdominal hysterectomies (introduced by Baer of Philadelphia) had become common. In the early 1890s, physicians began treating cases of lymphangitis, salpingitis, ovaritis, and peritonitis by curretting the endometrium, a process which eventually became a popular technique among gynecologists both in the United States and abroad. By 1900, there were some 101 obstetrical and gynecological journals in existence: 50 in Germany; 16 in France; 10 in the United States; 8 in Great Britain; 5 in Spain; 4 in Belgium; 3 each in Holland and Italy; 1 each in Russia, Denmark, and Switzerland.[10-15]

The specialty of gynecology can properly be attributed to J. Marion Sims of Alabama, the "Moses of Gynecology," who introduced the duck-bill speculum into general use. This device, along with Recamier's revival of the tubular speculum in 1801, enabled physicians to report injuries to the parturient canal and learn more of the parts of the pelvis than from centuries of previous medical speculation. Prior to the use of the speculum, diseases such as cystocele, rectocele, carcinoma, corroding ulcer, polypus, and cauliflower excrescence were recognizable. On the other hand, inflammations of the uterus were unknown and vesicovaginal fistulas were considered incurable. With the speculum, which he fashioned from a bent pewter spoon, Sims experimented on slaves suffering from vesicovaginal fistula in an infirmary constructed in his backyard. The speculum enabled him to see the fistula of the slave Anarcha, who passed both urine and feces through the vagina. Sims operated some 30 times on Anarcha between 1845 and 1849 before finally succeeding in correcting the condition with the use of silver wire sutures. Soon, visual examination via the speculum gave new impetus to the study of the cervix and os uteri, the state of the vaginal walls, and discharges from the uterine canal.[16-19]

INFLAMMATION AND ULCERATION PERIOD

Although the speculum marked a turning point in the treatment of female diseases, its widespread use also afforded physicians the opportunity to attribute almost all of woman's illnesses to the inflammation or ulceration of the cervix, which they treated with surgery or local applications of silver nitrate. For 20 years after Sims' initial success, the inflammation and ulceration theory influenced doctors treating nausea, uterine inertia, sexual dysfunction, disorders of the liver and kidneys, hysteria, and the irritable uterus.[7(pp 442-453),20]

Although the speculum was a catalyst in the development of gynecology as a specialty, many critics described it as a diabolical instrument designed to undermine women's chastity. Doctors opposing the specialty condemned gynecology as a quackish effort to interpret all of women's ills as somehow connected to the sexual system. They dubiously titled the speculum "the spoiler." As J. A. Mowris in his *Expose of Surgical Seduction! As Practiced by*

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Physicians who Specialize on Female Diseases (1871) remarked, "Let every true woman shrink instinctively from the physician who, for paltry gain, would subordinate her individuality to sexualism. Let the father and husband protect the domestic circle from the intrusion of the spoiler." [21(p 4),22] A review of medical journals during this period indicates that many of their editors as well as authors objected to the use of the speculum, since it required doctors to make visual observation, thus "blunting virtuous sensibility" and "breaking down the barriers which hedge in chastity." [23,24] Its application was thought to wreck women's physical and moral nature, leading to "a shock whose climax in this case is a spasm or fit" which shattered the nervous system and made permanent invalids of many women.[21(p 1)] One physician depicted young women "reduced, by the constant use of the speculum, to the mental condition of prostitutes; seeking to give themselves the same indulgences by the practice of solitary vice; and asking every practitioner, under whose care they fell, to institute an examination of the sexual organs." [25,26]

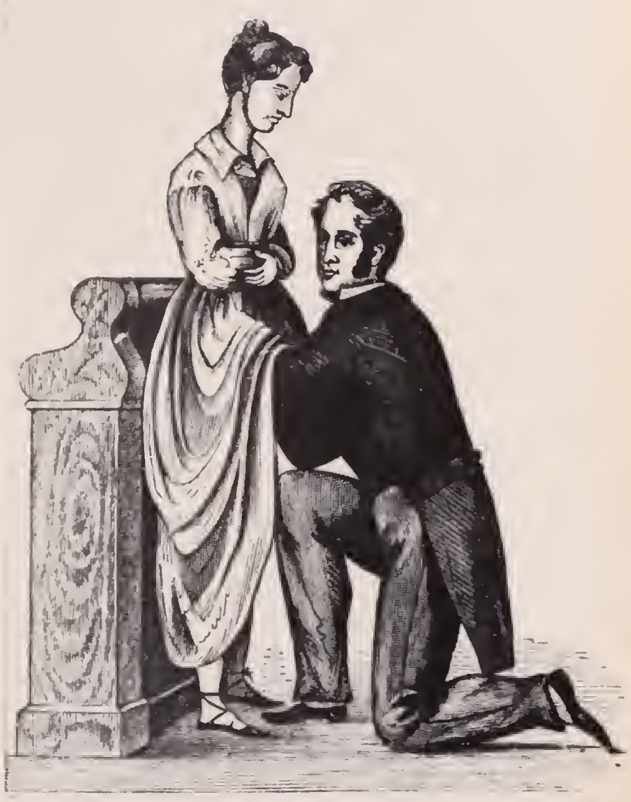
Even at the turn of the century, many within the medical profession criticized their colleagues for subjecting women, particularly unmarried ones, to thorough pelvic examinations. These opponents believed that such examinations were little more than "moral rape," and should be made only as a last resort to protect life. Physicians soon found it prudent, when examining young unmarried women, "to insist upon the mother or some other discreet married friend being present." Those physicians, however, who accepted the responsibilities of preventive medicine insisted that the advantages of the pelvic examination far surpassed its perceived affront to modesty. "The truth is," observed Sheldon Leavitt, MD, in 1909, "there would be fewer physical wrecks among our girls if careful examinations were more common, for there are many remediable ailments which, when neglected, work disaster to physical health and vigor." [27,28]

Mechanical Period

The development of the uterine sound and sponge tent by Sir James Simpson in the 1840s resulted in the gradual overthrow of theories of inflammation and ulceration of the cervix and gave rise in the 1860s to a new mechanical theory of uterine pathology. Displacements of the uterus (versions and flexions) now held major pathological importance and advocates of the new mechanical theory became authorities on exercise, bicycling, walking, sitting, social etiquette, and sexual relations. Womb displacement became an important medical and social concern of Victorians as physicians used it to explain everything from menstrual disorders to constipation and hysteria. Believing that women's health demanded proper mechanical supports, doctors became structural engineers, straightening the uterus with an assortment of props. Despite the lack of systematic investigations regarding the correct position of the uterus in healthy women, flexions were given grave pathological importance, even those which presented no difficulty. Proponents of the mechanical theory could not believe that the healthy uterus could encompass so wide a range of movement and variation of shape.[29]

During this second period of gynecological treatment, Hugh L. Hodge, MD (1796–1873), professor of diseases of women at the University of Pennsylvania from 1835 to 1863, introduced a hard rubber ring pessary (1830), suggesting that the uterus characterized by enlargement, dislocation, congestion, hypersecretion, or tenderness could best be treated through a truss-like support, designed to hold the cervix well back and up in the pelvis. His model became the basis for nearly all other pessaries, including those of Albert Smith and T. Gaillard Thomas.

MEDICAL MORALS. 21



The above Plate, copied from "Magrier's Midwifery Illustrated"—edition of Dr. Doane, New York—represents some of the indelicate duties of male physicians.

Fifty thousand men, composing the medical profession in the United States, are engaged in such an occupation!

"The present practice of medicine, especially obstetrics, must be set down not only as having an immoral tendency, but as, in itself, a gross, abusive, and shameless immorality."—Rev. Wm. Hosmer, *in the Young Lady's Book*.

FIGURE 1. Illustration with original caption from Gregory G: *Medical Morals, Illustrated with Plates and Extracts from Medical Works; Designed to Show the Pernicious Social and Moral Influence of the Present System of Medical Practice, and the Importance of Establishing Female Medical Colleges, and Educating and Employing Female Physicians for Their Own Sex.* New York, George Gregory, 1853.

Within a short time, however, imitators attempting to capture the gynecological market flooded medical and popular journals—including the *Sear's Catalog*—with devices almost as numerous as patent medicines. In many ways, the Victorian age represented itself as a period of mechanical contrivances, and gynecology, no less than the other professional specialties, became enamored of its possibilities. For the empiric, gynecology offered a lucrative opportunity to develop the ultimate panacea for women's troubles. Gynecologists who had become overly monistic in their focus on the genitalia of the woman turned the vagina into a "perpetual tool chest or toy box for pessaries" (known popularly as "pokers" or "jockey caps") and contrivances too numerous to catalog—from vaginal injections of iodine and carbolic acid, sponge tents, caustics, and cotton tampons, to electricity, ovarian pressure belts, corsets, and inflated rings.[30-34] The pelvic organs remained the locus of most of woman's ills as physicans and empirics found that her uterus, "like her nose, is a little on one side; or, again, that the organ is running a little, or it is as flabby as her biceps; so that the unhappy viscus is impaled upon a stem, or perched upon a prop, or is painted with carbolic acid every week in the year except during the long vacation when the gynecologist is grouse-shooting." [35]

One conservative innovation, massage, was introduced into gynecology by Thure Brandt (1819–1895) in the 1890s. Although many vigorously protested its therapeutic value as unscientific and warned that its application was morally dangerous to both doctor and patient, prominent gynecologists including A. W. Alvord argued that massage had been successfully used to treat chronic and sub-acute perimetritis and oophoritis, displacements, relaxation of the sphincters, hematocele, and rectal prolapse. Admitting that "unscrupulous and designing persons" might possibly produce harm, Alvord nevertheless believed the technique preferable to the surgical knife, electricity, or addictive drugs. Because of moral implications if male physicians administered massage, Alvord suggested the training of women assistants in the technique.[11,36] Disregarding Alvord's conciliatory remarks, critics continued to insist on the moral dangers of the treatment, suggesting the possibility that a genito-urinary surgeon might advise massage of the penis for "inflammatory deposits" or "pathological congestions." [37]

Despite notable achievements, the early years of gynecology suffered because too many physicians turned to the specialty without first learning from general practice, a situation which, according to medical critics,[38-40] left many physicians tempted "to overleap the bounds of prudence and humanity in subjecting their patients to unwarranted mutilation" from narrow pathological reasonings.[38] While the introduction of aseptic surgery rendered gynecological operations safer, it also increased the number of instances in which physicians, without sufficient justification, were only too eager to wield the knife when other, less radical techniques would have sufficed.

Radical Surgery Period

The mechanical theory of the 1860s and 1870s, which replaced the inflammation theory of the 1840s, gave way in the 1880s to a period of radical surgery, with doctors often excising the entire female generative system. So pervasive did this tendency become that noted neurologist Silas Weir Mitchell (1829–1914) of Philadelphia complained that pelvic surgery had fallen into the hands of inexperienced physicians who thought that destroying the function would bring an end to the disease. "In no case seen by me," he wrote, "has ablation of ovaries and termination of menstruation cured an epilepsy. I have never sanctioned such operations where the appendages were sound." [41]

Critics complained that total extirpation of the generative organs was done for trifling as well as serious problems. As one physician remarked in 1896, "the value of the operations was too highly placed, the relief obtained from them was unduly magnified, while the evil results and suffering following them were minimized or ignored." In effect, remarked those opposing the tendencies toward radical surgery, surgeons without sufficient knowledge of the pathology and natural history of diseases chose unusually severe operations as the rule rather than as the exception. Failing to trace a diseased condition from cause to effect, they indiscriminately unsexed women with the promise of cure. Women with menstrual problems, epilepsy, masturbatory habits, hysteria, or routine nervous disorders had their ovaries removed with little thought of alternative treatment. Surgeons "as well might conclude that a man should be castrated for mental derangement accompanied by some abnormal condition of the generative tract," remarked R. R. Kime, MD, in 1892. So common had these operations become, he added, that he would rather take his chances as "a moonshiner in the mountains of East Tennessee than to be a uterus in New York." [37]

The professional gravity with which doctors exercised their diagnostic skills on woman's illnesses only thinly disguised their mental reservations respecting woman's place in nature. In discussing the female of the species, physicians tended to indulge in a romantic exultation of her beauty while choosing a very narrow explanation of her ills. Hugh Hodge was typical of many of his colleagues. In his *Diseases Peculiar to Woman Including Displacements of the Uterus* (1860), Hodge gave an intellectualized testament to woman's refined nature which relegated her to a self-centered invalidism.

> Sex modifies irritability in a remarkable manner. Women, compared with men, are impressible and sensitive. This is true of their organic, but especially of their animal life. The action of their heart and arteries is rapid, their secretions and excretions are usually abundant and very readily increased or diminished. They generate animal heat rapidly; and this function, connected with the ever varying fluctuations of the circulation, is also easily disturbed in the various portions of the economy. Hence arise sudden determinations of blood to the head, with the development of sensations of heat, as in the phenomena of blushing, crying; and, at the same time, a corresponding diminution of excitement, a sedation, in other tissues or organs, as in the hands and feet. The sensibility and versatility of the organs of animal life are most remarkable in the female sex. Their sensations are all delicate and acute, making powerful impressions on the "sensorium commune," with a corresponding influence on the muscular system, dependent, as we all know, on the nervous system. Hence arises the ready disturbance of the heart, stomach, bowels, uterus, etc., from certain cerebral excitements; hence the rapidity and delicacy of their muscular movements, giving rise to the beauty and grace which shed a charm over the motions of women.[42]

A physician writing in the *Buffalo Medical and Surgical Journal* in 1894 described a young woman, aged 23, whose fiance broke off their engagement three days before the wedding. The woman took to her bed with nervous prostration marked by dysuria and an irritable bladder. After diagnosing these "uterine sympathies" as signs of hysteria, her family physician treated her with external and internal electricity and repeated flushing of the bladder. When her health continued to deteriorate she sought the advice of a specialist who diagnosed her symptoms as resulting from anteflexion of the womb, cervical erosion, and a fissure at the neck of the bladder. For these problems, he dilated the urethra, washed out the bladder and treated the womb *secundum artem*. When the symptoms continued, the woman went to a third physician who dilated the urethra, washed out the bladder, and applied internal electrodes and cold douches to the bladder. Failing to obtain relief, she sought the services of yet another physician who continued the same form of treatment, to no avail. A fifth physician treated her for anteversion of the womb and a rectal ulcer, but again, the symptoms remained. At length she turned back to an earlier physician who dilated the urethra so frequently that in examination his finger entered the external opening, and not recognizing his mistake, he then tried an artificial vesicovaginal fistula. When no benefit accrued from this, the physician removed her ovaries. Before the results could be observed, the patient died of peritonitis. The significance of this case was not so much the endless quest for recovery as it was the unanimity among the physicians treating her that she suffered diseases of the bladder and ovaries rather than what one medical skeptic more aptly diagnosed as a "sore brain" and "shocked pride." [43]

Operations were not without risk and countless women suffered the aftermath of unnecessary surgery. At the same time Abraham Flexner was urging an overhaul of the medical educational system in the United States, physicians concerned with the excessive surgical tendencies in gynecology began calling for an end to the ovariotomies, curettage, and wholesale sacrificing of women's sexual organs. In criticizing the tendency of gynecologists to operate on women for symptoms which were "neurasthenic," J. Thomas Kelly, MD, of the District of Columbia, complained that "there was nearly a wholesale wiping out of gynecological therapeutics." [44] As another medical critic observed, there had been "a clean sweep of the pelvic organs—a mutilated, unsexed woman—what a tribute to the skill of a surgeon!" [45] Charles Lee Carroll, MD, of the Woman's Hospital in New York, one of the first gynecologists to warn of the dangers of over-enthusiasm in operative procedures, reminded his colleagues in the profession that "women had all the usual organs outside their pelvis any of which might be the subject of disease!"[12(vol 1,pp 204–205),46]

REASSESSMENT

Although modern techniques made it possible for the surgeon to invade nearly every part of the body, those very skills tended to transform patients into a "case of this or that disease, the woman with the tumor, the woman with a tube," rather than as a creature of intellect with concerns and emotions. Moreover, critics of radical surgery from the early decades of the 20th century onward were quick to point out that in addition to the known function of ovulation, the ovaries played a subtle and complex role in the woman's body.[47,48] "In saving tubes and ovaries," remarked C. M. Rakestrow,[49] MD, in 1909, "our object is something more than merely to save the sexual delights of life or the office of procreation; in saving a uterus we are attempting more than the preservation of the menstrual function or the prevention of a premature menopause. We are saving the intellectual life, the emotional life, and are regulating the sensory nervous system so that the various gland functions will not be interfered with."

Though not all general practitioners opposed gynecology, many felt that efforts to define the sexual functions apart from the general system complicated successful diagnosis and treatment. To be proficient in the field of gynecology, argued the generalist, a doctor should first broaden rather than narrow the frame of reference. "The differential diagnosis between local troubles of the various organs and reflex irritations due to abnormal conditions of the sexual system," remarked G. E. Cogswell,[50] MD, in 1894, "is not always clear, and to be able to tell at a glance, one must be familiar not only with the local manifestations, but with the reflex irritations as well." For specialists to refer every disorder to the confines of their own particular branch without understanding its relation to the whole was dangerous. All too often, medicine became inundated with specialists whose tendency was to view their own precinct as the source of all ills and overlook the rest. "What we need is not less knowledge of the whole. When we can take the entire system in at a glance, we shall be better able to assist in restoring the body to a state of health by making it every bit whole." [51] A successful gynecologist, argued the new school of conservative therapeutics, must necessarily look to the entire system.

The true art and science of gynecology remained lost amid an assortment of surgical and scientific triumphs in the 19th century—triumphs which were often achieved at the expense of an enlightened understanding of patient needs. The muddled banalities used to define women, the awkward caricatures and cultural prejudices which managed to outlast the eras which spawned them, finally culminated in more recent times in deep moral stirrings that significantly modified both cultural attitudes and institutional barriers. Partly in response to these changes, surgeons have increasingly chosen to retain the sexual function during repairs of prolapse and lesions of the cervix. Disorders which they previously "cured" with radical surgery are now treated with less drastic therapeutic procedures. Nevertheless, while change has become apparent, the removal of the uterus, dilation and curettage, mastotomy, and mastectomy remain among the more frequently performed operations.[52] Perhaps the advice of one doctor in 1888 best illuminated the historical and cultural influences on the practice of gynecology—and may still serve to remind us today. "No men in medicine are worthy of more honor than the true gynecologist," he wrote, "but to be a gynecologist one must be broader than a woman's pelvis, deeper than her vagina—in his range of mental vision." [30]

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Asthma in Timisoara

Diary of a glimpse at the Romanian medical system

RICHARD SELTZER

We just returned from a trip to Romania, where our son Bobby played in the World Under 14 Chess Championship as the representative from the United States. There, unexpectedly, we got a glimpse of the Romanian medical system and became friends with a young Romanian doctor who aspires to become a surgeon.

July 25. The eight tournaments (World Under 10, Under 12, Under 14, and Under 16 Championships for boys and girls) are being held in Timisoara, an industrial city of 300,000 in southwestern Romania, near Yugoslavia and Hungary. To one arriving by plane from Frankfurt, the land looks flat and fertile, large fields of corn and wheat are interspersed with stretches of forest. On the ground, it looks like Indiana.

Recent emigres advised us not to drink tap water and not to eat the meat (for fear of hepatitis as well as intestinal disorders). Meat was likely to be stale, poorly refrigerated, and handled without Western concern for cleanliness. They told us to bring canned meat, chocolate, and coffee. They said we would not be able to obtain milk.

In a fertile land, which is now bursting with ripe grapes, plums, and watermelons, fruit is very hard to find here. We are surprised that there are no street vendors in the parks, and the stores have no fruit.

Today, we saw bakeries with idle sales people and no baked goods, not even bread, for sale. Fruit and vegetable stores have nothing but cabbage for sale. A milk store had nothing but cobwebs on its shelves. A nearby meat store was empty the first time we passed. Later, we saw lines stretching outside and around the block.

It turns out that for almost everything that is worth buying, one needs dollars, not *lei*, the local currency. The hotel bill has to be paid in dollars. And the shops in the hotel, that sell Romanian-bottled Pepsi Cola (with a strange bitter-sweet taste) and bottled water, only accept Western currency. On the flourishing black market, a dollar is worth 80–90 lei, in contrast to the official 8–9 lei exchange rate. The chances of a foreigner's running into trouble with the law are great, and hardly worth the risk.

Because of the underground economy and the relative uselessness of the local currency, Kent cigarettes have become an unofficial medium of exchange. A package of Kents makes an excellent tip. Fortunately, we had heard that before and brought along a couple of cartons.

Outside the hotel, there is very little to be purchased. The department store next door has unappealing, machine-made goods, displayed on dark shelves. Because of a perpetual shortage of electricity, there are no lights on, and the escalators have not operated for years.

At every other street corner, someone huddles up against the wall and opens a canvas bag, and passersby swarm to buy black market merchandise, typically cigarettes and foreign goods.

July 30. After breakfast, standing in the lobby by the stairs, I was talking to the mother of Hedinn Steingrimsson from Iceland. Hedinn is sick. He collapsed and had to be taken to the hospital. He's in their room now, resting. She doesn't know if he'll be strong enough to play when the tournament resumes tomorrow.

Darshan Kumaran's father came along. (Darshan is the representative from England in the Under 14. I hadn't known that his father was a physician.) Concerned about Hedinn, he inquired about the symptoms and what the Romanian doctors had done.

Hedinn had had stomach troubles and diarrhea. He had severe headaches and felt very weak. When he fainted, his mother was afraid it had something to do with the heart. "No," she answered Dr Kumaran's question, "he never had trouble with the heart before." They gave him calcium. As a dietitian, she understood that was because of the lack of calcium due to the absence of milk in the diet here in Romania. "How did they give it to him?" Dr Kumaran asked, with considerable concern. "Tablet or injection?" "Injection." "My God, that's dangerous. He's all right now. That's fortunate. No need to worry now. But such an injection could have stopped his heart. It's normal to be weak from diarrhea. There is nothing wrong with his heart. They should never give an injection of calcium in those circumstances." "But I didn't know." "If he is sick again, call me. Talk to me. Let me talk to the Romanian doctors."

At lunch, the head of the British delegation told me that seven of their players are sick, with stomach troubles.

July 31. Last night, while we were playing cards in our room, Bobby started sniffling. He had had a slight head cold for days. How could he catch a cold when it's

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100 degrees outside and there's no air conditioning? Maybe he's allergic to something, and we shouldn't have opened the window.

About midnight, when the card game ended, Bobby went running up the stairs to the sixth floor with the other kids and came running back. When he returned, he was breathing heavily, coughing, and wheezing. We tried to calm him.

He had previously had some asthmatic symptoms associated with bronchitis, most recently a year ago. So we had brought along his Ventolin "inhaler." And on several nights, he had wheezed a bit and taken medicine through that inhaler. Tonight that medicine had no effect.

For an hour and a half, he struggled to find the right position or achieve the right state of mind for the problem to subside. But it was becoming increasingly difficult and painful to breathe. By 1 AM, he was desperate. He needed to see a doctor.

I went to the front desk at the hotel. At first the woman at the desk could not find the doctor who was assigned to the hotel for the chess tournaments. She called another hotel. The doctor there wasn't in. She got the number of another doctor. I asked her to please call Dr Kumaran. She did not know what room Dr Kumaran was in and couldn't find his name in the hotel register. Eventually, she located the doctor assigned to our hotel.

At 1:20, the hotel doctor came to our room. He looked at Bobby, who was leaning over the sink trying to breathe and said, "It seems he breathes fine." I tried to explain in more detail Bobby's history of asthmatic bronchitis. Without examining Bobby, without even getting within three feet of him, the doctor recommended taking him to the hospital for "an injection." He explained that he was "not a doctor for children" and could not "be responsible" for treating Bobby. Bobby had to go to the children's hospital for treatment.

I was reluctant. I wanted to talk to Dr Kumaran first. I went back to the front desk. For an interminable time, the very patient and helpful woman at the desk scanned the hotel's two handwritten registers and could not find Dr Kumaran listed. I asked her to call someone else from the English delegation. This person gave her Kumaran's room number, 511. She dialed his room, and I explained the problem. He did not know me by name (at least not at 2 AM, having just awakened). But he did come within five minutes.

Dr Kumaran agreed that Bobby probably needed a hydrocortisone shot, but said a slower working pill might bring relief in about 45 minutes. We decided to try the pill, which Kumaran fetched from his room. Both doctors left.

For over half an hour, Bobby struggled again to calm down and achieve normal breathing. There was no improvement.

The Romanian doctor returned. We asked him to please call an ambulance. Dr Kumaran returned and gave additional advice.

The ambulance was called at 3 AM, and arrived at 3:05. I rode with Bobby to the hospital (about half a mile away), where he was was under observation by 3:15.

A team of medical people briefly examined him and asked me questions, with the doctor from the hotel acting as interpreter. They asked my permission to give Bobby a hydrocortisone shot. (It helped my sanity that Kumaran had already advised that.) They then took him upstairs to another room. There they prepared the injection and give it to him while I sat and watched.

The white metal table beside me had a slightly open drawer. While I watched them treat Bobby, a cockroach scampered in and out of that drawer.

There was a blood stain on the sheet Bobby was lying on. At the time, I thought it was there before they put Bobby into bed, which fed my fears of unsanitary conditions. (Afterwards, I realized it was blood from Bobby, when they gave him the injection.)

The syringes were not disposable. They appeared to be made of glass and had been used many times. The needle was inserted into the patient's arm and then the glass container with the medicine was screwed onto it.

Along with the hydrocortisone, they gave Bobby something to help him sleep. Very soon, his breathing returned to normal, and he drifted off to sleep.

Every ten minutes or so the head doctor (a woman who spoke no English) returned to listen to his breathing.

The doctor from the hotel stayed.

The first time he had come to our room, back when I wishfully thought that the pill from Dr Kumaran would do the trick, I had offered him a package of Kent cigarettes as a thank you. He had refused and seemed professionally offended by the gesture. Now I explained that I appreciated his help and simply wanted to thank him. Was there anything, such as books, that I could send him from the United States as thanks?

That led to an hour-long conversation, during which he enjoyed practicing his English and I got some insight into the Romanian medical profession and came to respect his good intentions and competence.

Students in Romania attend high school until the age of 20, then take tests for the university. Only about one in eight passes these tests. University is free, but one can only enter if one passes these tests. If one wishes to become a doctor, one must attend the university for six years, then have three years of residency, after which another test must be passed. But for several years now, that test has not been given, so this student is stuck in limbo. After passing that test, he will have three more years of residency in his specialty (surgery). His "chief" had the extraordinary good fortune of being able to study at Johns Hopkins on a US fellowship.

Doctors in Romania are paid about 100 lei per day. In US currency, at the official rate, that would amount to $12.50. At the black market rate, it is only $1.25. (Although he didn't say so, this is almost certainly supplemented with black market dealings.)

Foreign medical books are almost impossible to obtain. Standard texts like *The Textbook of Internal Medicine* by Harrison and *Textbook of Surgery* by Schwartz sell for $85 in the US. He would have to work about 68 days and save every penny to get enough US currency to buy one such book.

The doctor gave Bobby an assortment of pills—prednisone, enfilin, calcium, and something to help him sleep. Now Bobby cannot eat anything with salt because the salt would interact with the medication. That means he cannot eat the canned meat and crackers that we brought with us.

The restaurant in the hotel will prepare special salt-free meals for him.

August 1. Bobby had another bout with asthma this morning. Again he went to the hospital. Again they gave him a hydrocortisone injection (this time, three vials full).

Seeing the children's hospital in the daylight, I could see that the building must date back to before 1900. It looked like an old monastery complex in bad repair. Stone, in need of a new coat of whitewash. An exterior wall, also needing a whitewash. Inside, plaster peeling from the walls. Even the upper stories have the look of a basement. To conserve electricity, there are almost no lights on in the halls, very dark at noontime.

They also provided enough enfilin and prednisone pills to last for the rest of our stay. When our supply of calcium pills ran out, we were given calcium in liquid form in glass tubes that could only be opened by cracking the glass (which is very hard to do without getting particles of broken glass in the medicine).

Bobby has played very well while sick. The weaker he is, the more determined he gets. Yesterday, after staying up most of the night with asthma, he made a mistake on move 9 that should have cost him the game. But he played confidently, as if his mistake were a deliberate sacrifice; and his opponent, Eliet from France, proceeded cautiously and didn't find any of several possible winning plans. Bobby got the upper hand, and at move 20, Eliet resigned, in a hopeless position. Then, today, even more exhausted from his second bout with asthma, Bobby beat Faybisch from Belgium in 22 moves.

August 2. The cold symptoms have returned and become far worse. Bobby is constantly coughing and sneezing. The doctor has given him penicillin pills, along with the other medication.

Today, he played one of the finest games he has ever played—not for flashes of brilliance, but rather for firm control of the position—carefully analyzing his own and his opponent's options many moves in advance, finding the best plan, and executing it well.

By evaluating his opponent's threats precisely and providing no more defense than necessary, he could gain tempo or add a piece to the attack. It was like the performance of a race car driver who lets his vehicle go on two wheels around a difficult turn, a well-calculated risk that provided a winning advantage.

August 3. Round 9 was a battle of the walking wounded, Bobby versus Hedinn. Hedinn looked like an Arab, with a wet cloth wrapped turban-style around his head. Bobby was well-armed with handkerchiefs for his runny nose. They had played last year in Puerto Rico. Bobby had lost then, but theirs was one of the best games in the Under 12 championship tournament.

This year, after a slow start in the first few rounds, both Hedinn and Bobby needed a win to climb back on top. Actually, in a closely matched tournament like this, it looked like eight points might be enough to tie for first—so they both needed wins in the last three rounds.

Hedinn, as white, played a line that offered many traps and quickly led away from familiar positions. Underestimating the dangers of this line, Bobby accepted a pawn sacrifice and quickly found himself in a stranglehold position—unable to castle or develop, while Hedinn relentlessly added pieces to the attack. It was a painful experience, probably far more painful than his worst attack of asthma.

August 6. In round 10, Bobby beat Zhu from China. And today, in the last round, he was paired with Chatalbashev from Bulgaria. While the game was in progress, I took a walk around the block. When I returned, there was an ambulance at the door. A crowd had gathered near the stairs. They were carrying a child out. I struggled to get closer. No, it wasn't Bobby, It was another player, a young girl.

When we said goodbye to our doctor friend tonight, he told us that two players in the tournaments had had appendicitis and had had to be operated on immediately. We were quite fortunate that Bobby's was only a minor problem.

August 7. Despite the shortages, we saw no evident discontent, no signs of imminent rebellion. We saw armed guards at the airport, but nowhere else. This was simply a different way of life that people were used to.

In fact, the shortages, the rationing, and the black market that they give rise to are part of the system of government. For instance, each family is rationed just 30 liters of gasoline per month (not enough to fill the tank once); the only way to get enough to meet ordinary needs is to resort to the black market. The same is true for many other essential commodities. There is no milk, so to help their children grow properly and stay healthy, parents have to obtain calcium pills on the black market. Everybody uses the black market, and everyone knows the secret police are everywhere, watching everything—taking notes and keeping files, but only prosecuting when it suits their purpose, always having enough evidence to put anyone in jail or to intimidate and manipulate whomever they wish.

Machiavelli would have admired this system of government by corruption. It seems to be very effective. It seems to perpetuate itself and to require only a modicum of central direction and control.

In a nation where everyone feels compromised, guilty, and at risk, it must be difficult to generate righteous indignation against the powers that be. Abstract notions of justice and human rights ring false here. An Oliver North might learn to cope here, but a Robespierre would be totally out of place. The survivors are those who can work with and around the bureaucracy, who know when and how to bend and break the rules.

Distal colonic tuberculosis with tuberculous enteritis, peritonitis, and pericarditis

JOSEPH C. SCIAMMARELLA, JR, MD, ALEX H. BRUCKSTEIN, MD

Gastrointestinal tuberculosis has declined in frequency since the introduction of anti-tuberculous chemotherapy. The 36 cases reported by Sherman et al[1] comprise the largest recent North American series published. Of the 38 cases of extrapulmonary tuberculosis reported by Weir and Thornton,[2] only four had intra-abdominal involvement. The disease remains endemic in Asian countries, particularly India, where it continues to be a major health problem.[3]

We report a case of gastrointestinal tuberculosis involving the transverse and descending colon without clinical pulmonary involvement. The patient had associated tuberculous enteritis, pericarditis, and peritonitis, all documented antemortem. At postmortem examination, a basilar pulmonary granuloma was discovered, from which *Mycobacterium tuberculosis* was recovered.

CASE REPORT

A 66-year-old woman was admitted to St Vincent's Medical Center of Richmond for evaluation of a 40-pound weight loss over a six-month period. The patient was born in Jamaica, British West Indies, and had emigrated to the United States four years prior to admission. There was no clinical history of tuberculosis, and chest radiographs taken at the time of immigration were reported to be negative.

Although experiencing general malaise, the patient denied pulmonary or cardiovascular symptoms. Physical examination revealed a cachectic, elderly woman. Her temperature was 101.6°F; blood pressure, pulse, and respirations were within normal limits. Bilateral anterior cervical and inguinal lymph nodes were palpable, as was a 4-cm left axillary lymph node. No evidence of increased central venous pressure was noted, and the cardiopulmonary examination was unremarkable. The abdomen was soft and slightly distended. There was no ascites appreciable and no hepatosplenomegaly. Rectal examination was unremarkable. Stool was negative for occult blood.

Initial laboratory data included hemoglobin, 7.3 g/dL; hematocrit, 23.5%; leukocyte count, 12,600 cells/mm^3, with a differential count of 76% neutrophils, 16% bands, 6% lymphocytes, and 2% monocytes; platelet count, 617,000/mm^3. Serum electrolytes were within normal limits. The serum aspartate aminotransferase (AST) was 77 IU/L; lactic dehydrogenase (LDH), 291 IU/L; and alkaline phosphatase, 134 IU/L.

A chest radiograph revealed cardiomegaly, but no pulmonary infiltrate or effusion was seen. An echocardiogram subsequently documented the cardiomegaly to be due to a pericardial effusion. Skin testing showed the patient to be anergic. Computed tomography of the abdomen demonstrated ascitic fluid.

The initial impression was one of occult malignancy. A barium enema revealed a filling defect in the transverse colon, with colo-colo and colo-enteric fistulae (Fig 1). Colonoscopy revealed several small ulcerations of the transverse and descending colon.

The preliminary diagnosis by the colonoscopist was inflammatory bowel disease. Biopsy of these lesions revealed acute and chronic granuloma formation. Acid-fast bacilli were seen on stains of the biopsied specimens and on stains of ascitic fluid obtained at paracentesis. Cultures subsequently identified *Mycobacterium tuberculosis.* Multiple blood and sputum samples were negative for acid-fast bacilli.

The patient was begun on triple anti-tuberculous therapy. Several days later, sudden cardiac arrest developed. The presence of electromechanical dissociation in the setting of a documented pericardial effusion prompted a therapeutic pericardiocentesis. The pericardial fluid yielded acid-fast bacilli.

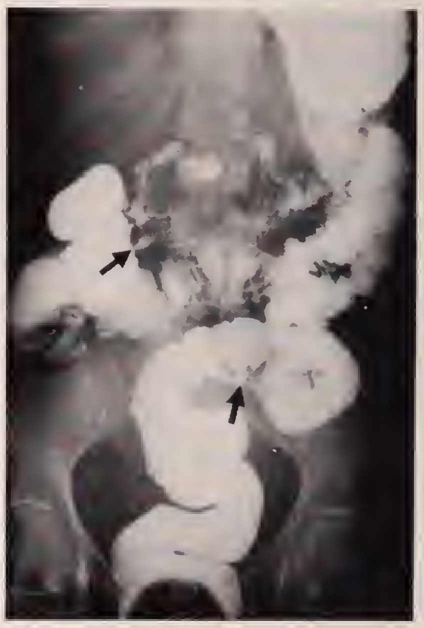

FIGURE 1. Barium enema, demonstrating colo-colo and colo-enteric fistulae (arrows).

The patient was resuscitated, but died several days later after a clinical course of septic shock. Postmortem examination determined the cause of death to be septic peritonitis secondary to multiple bowel perforations. Extensive caseating granulomas of the mesenteric lymph nodes were present, as was a clinically undetected 2.5-cm caseating granuloma of the left posterior lung base.

DISCUSSION

This case is very unusual, not only because involvement of the transverse and left colon with tuberculosis is rarely seen, but also because of the clinical evidence of pericardial, peritoneal, and enteric disease.[1]

The ileocecal area is the most common location for gastrointestinal tuberculosis, while the transverse and left colon are very rarely affected. In the most recent review from a United States in-

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stitution,[2] of 38 patients with extrapulmonary tuberculosis, there were no cases of involvement of the large bowel. In the most recent review of gastrointestinal tuberculosis outside the United States, Klimach and Ormerod[3] found only eight of 109 patients with involvement of the large bowel.

Prior to the introduction of anti-tuberculous drugs, 70% of advanced pulmonary tuberculosis cases had coexisting gastrointestinal tuberculosis.[4] Advanced, cavitary pulmonary disease, with or without tuberculous laryngitis, allowed large enough numbers of organisms to be swallowed that the defense mechanism of a low gastric pH was not sufficient to prevent infection of the small bowel. Currently, gastrointestinal tuberculosis is very rare in the western world. In a study of 4,222 patients with tuberculosis hospitalized at the University of Michigan affiliated hospitals over a 21-year period, only 36 cases of intestinal involvement (0.85%) were noted.[1] Of these, 11 (0.26%) had tuberculous enteritis, 21 (0.50%) had tuberculous peritonitis, and 4 (0.09%) had both.

Because of the rarity of intestinal tuberculosis, such a diagnosis is seldom considered, often causing a delay between presentation and diagnosis. This is especially true when pulmonary involvement is absent or when the sites of intestinal involvement are unusual. Eighty-five percent of all cases of intestinal tuberculosis involve the ileocecal area, perhaps because of its abundant lymphoid tissue. As of 1980, only 100 cases of tuberculosis occurring distal to the cecum had been reported in the English literature.[5] The frequency of involvement decreases segmentally as one proceeds distally from the ascending colon, with only 28 cases of involvement of the transverse colon reported in the world literature as of 1979.[6]

Concomitant tuberculous enteritis and tuberculous peritonitis is uncommon, occurring in four of 36 cases in one study,[2] two of 31 cases in another,[7] zero of 47 cases in another,[8] and in five of 30 cases in an additional study.[9]

If intrinsic intestinal tuberculosis is present, the peritoneum can be affected either by direct extension from the intestinal wall or through hematogenous or lymphatic spread of the infection.

Tuberculosis was once the most common cause of pericarditis. A 1980 study that evaluated a ten-year period found that the incidence of tuberculous pericarditis had decreased to 12 of 1,194 cases (1%).[10] Based on our review of the English literature, there are no reports of documentation of concomitant tuberculous colitis and tuberculous pericarditis.

Unfortunately, no pathognomonic syndrome exists for intestinal tuberculosis. The classic "doughy" abdomen is rarely encountered[11] as are the findings of ascites[12] and intestinal fistulae.[1] Constitutional symptoms are present in more than 50% of cases, with fever and abdominal distention being the most common.[9] The erythrocyte sedimentation rate is often elevated, but this is nonspecific. Pulmonary involvement is frequently absent, with minimal or no radiographic evidence of pulmonary lesions.[3] Because of the severity of the disease, advanced age, or malnutrition, the purified protein derivative (PPD) skin test can be falsely negative due to anergy.

The yield of abdominal paracentesis in the diagnosis of tuberculous peritonitis is poor.[9,13] Smears of ascitic fluid are usually negative, but the yield increases as the volume of fluid examined increases. Bacteriologic cultures are often negative, although one study had a positive culture rate of 83% by utilizing a liter of ascitic fluid.[8] Peritoneal biopsy and stool cultures, for suspected tuberculous peritonitis and enteritis, respectively, may help confirm the diagnosis.

Making a diagnosis of pericardial tuberculosis is difficult. Diagnostic pericardiocentesis for bacteriologic examination yields positive smears in only 6% of cases. Pericardial biopsy may increase the yield.[14] Colonoscopy with biopsy has proven a useful tool in the diagnosis of colonic tuberculosis.[15] Because accurate differentiation from malignancy or inflammatory bowel disease is difficult, colonoscopy remains an important diagnostic modality. Despite the lack of direct evidence of acid-fast bacilli on smear or positive bacteriologic culture, some studies have reported a presumptive diagnosis of gastrointestinal tuberculosis based on a positive response to anti-tuberculous chemotherapy.[12]

Prior to the use of anti-tuberculous drugs, intestinal perforation due to tuberculosis was rare, occurring in only ten of 734 cases (1%) in one study.[16] Perforation was prevented by early reinforcement of the ulcer bases by adherent mesentery. Reports have since documented the prevalence of intestinal perforations during anti-tuberculous chemotherapy.[17,18] The presumed mechanism of perforation is a significant decrease in the inflammatory response before enough time has elapsed to allow the formation of adequate amounts of fibrous tissue.[4]

This patient presented with several unusual features, including the atypical location of the lesions in the transverse and descending colon, the presence of intestinal fistulae, and tuberculous enteritis, peritonitis, and pericarditis. Also unusual was the recovery and subsequent identification of acid-fast bacilli as *Mycobacterium tuberculosis* from ascitic and pericardial fluid.

Features consistent with intestinal tuberculosis were the presence of constitutional symptoms, a normal chest film, anergy, and the development of bowel perforations (perhaps related to the initiation of anti-tuberculous therapy). One must be alert to the possibility of bowel perforation following the administration of anti-tuberculous drugs. The usefulness of colonoscopy for diagnostic biopsy was corroborated in this case.

A high index of suspicion, along with an awareness of the possibility of intestinal tuberculosis, especially in a person who has emigrated from an endemic area such as Asia or the West Indies, despite the rarity of the disease in the western world, is the key to a prompt diagnosis.

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See also pp 251-254.

Mammary tuberculosis

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Since tuberculosis of the breast was first described by Sir Astley Cooper in 1829 approximately 500 cases have appeared in the world literature.[1] A 1924 necropsy study of 10,000 patients who died of tuberculosis found mammary involvement in only seven cases.[2] The majority of cases reported in the past 20 years come from either the surgical literature or from countries such as India, where tuberculous mastitis represents 3% to 4.5% of all surgically treated breast disease.[3] The disease has not been reviewed in the American medical literature since 1977.[3] We present a patient recently seen and treated at the Bellevue Hospital Center in whom a mammary tuberculoma was apparently refractory to treatment with isoniazid and rifampin despite in vitro susceptibility to these drugs. A review of the English language literature failed to show a prior report of treatment failure with this combination of drugs.

CASE REPORT

A 45-year-old woman from Ecuador, who had lived in the United States for 20 years, noted a painless mass in her right breast in June 1986.

She underwent mammography, which revealed a large noncalcified mass. Her medical history was unremarkable. Needle aspiration showed caseating granuloma with acid-fast bacilli present on smears. Cultures grew *Mycobacterium tuberculosis* susceptible to isoniazid, rifampin, ethambutol, streptomycin, pyrazinamide, and ethionamide. She was treated with isoniazid, 300 mg, rifampin, 600 mg, and ethambutol, 1,200 mg per day.

During the next four months, however, the mass continued to enlarge, as documented by serial sonographic examinations. The patient claimed to have taken her medications appropriately, and was noted to have orange colored urine. She was admitted to Bellevue Hospital for surgical excision of the mass. When seen by the infectious disease consultant, she reported no knowledge of a prior history of tuberculosis, and had had no known contact with tuberculosis, yet was found to have a positive purified protein derivative test (PPD) with 15 mm of induration.

On physical examination, she was noted to be afebrile and had no complaints of fever, weight loss, or night sweats. The only remarkable finding was a 6 × 6-cm nontender, freely movable mass in the lateral aspect of the right breast. There was no skin or nipple retraction, and no sinus tracts were present over the mass. A chest film was normal, as was a chemistry profile, including liver function tests and urinalysis. The complete blood cell count showed a hemoglobin of 12.3 g/dL and a white cell count of $5.9 \times 10^3/\mu L$ with a differential of 58% polymorphonuclear leukocytes, 34% lymphocytes, 5% monocytes, and 3% eosinophils.

The patient underwent partial mastectomy for removal of the mass, which on gross examination revealed fibrofatty breast tissue punctuated by areas of caseous necrosis. Multiple sections of the specimen showed diffuse necrotizing granulomatous inflammation. The granulomas were characterized by collections of lymphocytes, plasma cells, epithelioid histiocytes, and multinucleated giant cells with extensive necrosis. Where the breast tissue was not replaced by inflammation, it showed the features of mild fibrocystic disease. No evidence of carcinoma was seen. Three of five lymph nodes submitted for microscopic evaluation also showed necrotizing granulomatous inflammation. While rare fluorescent bacilli were seen on auramine stain of one of the lymph nodes, no acid-fast bacilli or fungi were seen on special stains. Repeat cultures of surgical materials were negative for mycobacteria. It is likely the rare bacilli in the auramine stain were nonviable.

DISCUSSION

The vast majority of tuberculous mastitis is seen in women and occurs primarily during the reproductive years, although cases have been reported from adolescence to age 77.[4] The initial diagnosis entertained is usually breast abscess or cancer, as the disease often involves the lateral aspect of the breast with fixation to the chest wall and nipple retraction. The disease is usually unilateral and, in more than 60% of cases, no other sites of active infection are found.[5] There appears to be a correlation between pregnancy and the development of mammary tuberculosis, as three of five cases reported by Wilson and MacGregor[6] involved lactating women. The reasons for this remain unclear, but changes in hormonal balances and breast anatomy have been suggested as possible explanations. There is no convincing evidence that trauma or breast cancer predispose to the development of tuberculosis.

Three main patterns of clinical presentation of mammary tuberculosis have been recognized.[3] The nodular form is the most common, presenting as a slow growing, well circumscribed mass which is usually painless on examination. Left untreated, this will eventually erode through the skin to form a draining sinus. The second form is disseminated tuberculous mastitis characterized by multiple foci and discharging sinuses. The sclerosing form of tuberculous mastitis is seen mainly in elderly

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females; it is the least common form and leads to chronic fibrotic changes with breast shrinkage and nipple retraction.

Tuberculous mastitis can occur as secondary spread from a pulmonary focus via hematogenous or lymphatic routes, and there have been cases of direct spread via erosion through the chest wall. Several other conditions can give a similar histopathologic picture including traumatic fat necrosis and mammary duct ectasia; therefore, one should have a positive acid-fast culture before making the diagnosis of tuberculous mastitis.[7]

Prior data on therapy for tuberculous mastitis have shown variable results.[8-11] The majority of patients have been treated with either surgical excision alone or excision followed by a course of antituberculous medications. Algaratnam et al[9] reported 16 cases of tuberculous mastitis, 12 of which were cured via excision of a tuberculoma or wedge resection of an abscess cavity plus overlying skin sinuses, followed by antituberculous chemotherapy for an 18-month period. Some reports have shown a cure with medical therapy alone. Dent et al[8] reported success in 20 patients treated with *p*-aminosalicyclic acid, isoniazid, and streptomycin in whom surgery was confined to a diagnostic biopsy procedure. Others have reported failures of medical therapy when used alone. Ikard et al[3] reported six patients with tuberculous mastitis, one of whom showed no improvement after a ten-week course of isoniazid and ethambutol and required a simple mastectomy. For the noncompliant patient, a simple mastectomy has been suggested as the treatment of choice.[6] Our literature search failed to show prior reports of therapy involving isoniazid in combination with rifampin.

In summary, we report a case of tuberculous mastitis in a middle-aged woman, which did not respond to antituberculous medications despite the presence of a susceptible organism. Cases have been reported of central nervous system tuberculomas developing in patients taking antituberculous medication with organisms found to be susceptible to these drugs.[12] It is possible a similar situation occurred in the patient reported here. Possible mechanisms for this may include poor drug diffusion into the infected area, as well as defects of cell mediated immunity.

While this disease is rare in the United States, it should be considered in the differential diagnosis of breast masses, particularly in immigrants from areas where the incidence of tuberculosis is high. Despite this patient's apparent failure on medical therapy alone, a trial of antituberculous medications should be attempted in a patient who is able to cooperate. The patient must be frequently re-evaluated, however, and it should be acknowledged that surgical excision may be necessary.

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Congenital splenic cysts presenting after blunt abdominal trauma

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Congenital splenic cysts are rare lesions found predominantly in children and young adults. Excision of these cysts by total splenectomy has been the accepted treatment until recently. We describe our experience with two adolescents who presented with abdominal complaints following blunt trauma and were found to have congenital splenic cysts. Included is a review of the pertinent literature. Reports of successful splenic salvage in patients with congenital splenic cysts are discussed.

CASE REPORTS

Case 1. A 15-year-old girl presented with vague abdominal discomfort and a palpable mass in the left upper quadrant following a fall from her bicycle five months earlier. An upper gastrointestinal series revealed the stomach displaced anteriorly and to the right by a large mass with a partially calcified circumferential rim (Fig 1). A liver-spleen scan identified filling defects in the upper and lower poles of the spleen. At surgery, the spleen was essentially replaced by a bilobed cyst. Total excision was performed. The specimen measured 22 × 14 × 11 cm and weighed 2.1 kg. Bisection revealed two noncommunicating cysts (Fig 2), the larger being 14 cm in diameter and containing 1.5 L of a brown fluid. The cysts were lined by stratified squamous epithelium with intercellular bridges. Microscopic examination revealed the cysts to be epidermoid in nature.

Case 2. A 17-year-old boy presented with left pleuritic chest pain and postprandial, left-sided abdominal pain. Three months earlier he had been involved in a collision while skiing, receiving a severe blow to his left side. He sought no medical attention. Physical examination revealed a palpable spleen. A computed tomographic (CT) scan (Fig 3) identified a 12-cm splenic cyst. A re-

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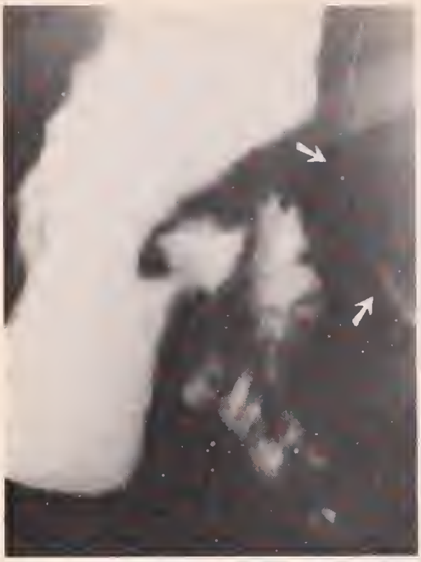

FIGURE 1. **Case 1:** Upper gastrointestinal series (lateral view) showing anterior displacement of the stomach by a mass with a partially calcified circumferential rim (arrows).

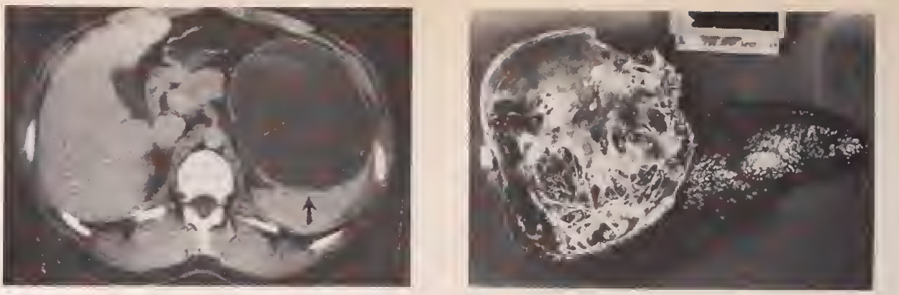

FIGURE 3. **Case 1:** Left, CT scan revealing a splenic mass (arrow) of water density, 12 cm in diameter; Right, large epidermoid splenic cyst replacing the superior pole.

peat CT scan two months later showed no decrease in the size of the cyst, and the patient was referred for surgical management. At exploration, the spleen was enlarged, with the upper pole and hilum replaced by a large cyst adherent to the diaphragm. The spleen and cyst were removed. On gross pathologic examination, the specimen (Fig 3) measured 20 × 14 × 13 cm, weighed 1,480 g, and consisted of a unilocular 13.5 × 8.0-cm cyst containing brown fluid. Sections of the cyst showed a lining of squamous epithelium (Fig 4). The lining was focally replaced by a resolving inflammatory reaction, presumably to blood. Microscopic examination revealed the cyst to be epidermoid in nature.

DISCUSSION

Association with Trauma. Splenic cysts associated with trauma usually result from blunt injury to the spleen causing parenchymal damage with an intact splenic capsule.[1] The resultant subcapsular hematoma liquefies, forming a false cyst without a cellular lining.

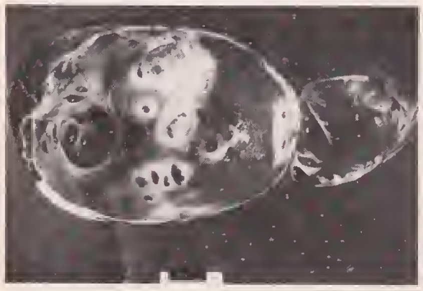

FIGURE 2. **Case 1:** Internal surfaces of the two noncommunicating epidermoid splenic cysts.

Congenital splenic cysts that were previously undetected can become symptomatic as a result of abdominal trauma. Stylianos et al[2] found that 30 (24%) of 125 patients with congenital splenic cysts presented following an episode of blunt abdominal trauma. The majority (83%) were children who had relatively minor blunt trauma.[2] The interval from trauma to presentation with abdominal findings ranged from several hours to two years. Most patients (63%) presented within six months with an abdominal mass (60%) and abdominal or pleuritic pain (57%).[2] It is likely that the intraluminal or intramural pressure of the preexisting congenital splenic cysts is increased due to hemorrhage following abdominal trauma. The blood and inflammatory exudate, as a response to injury, cause a localized irritation of the diaphragm and peritoneum and an increase in the size of the cyst, exerting pressure on adjacent structures. Three patients presented acutely with peritonitis following trauma secondary to rupture or infection of their congenital splenic cysts.[2-4] The hemorrhagic nature of cyst contents is best explained by the presence of focal subepithelial ectatic and engorged veins which are vulnerable to even minor trauma or increased luminal pressure. The trabeculated appearance of the luminal surface is due to the organization of the intramural hemorrhage. In the patients presented here, the congenital splenic cysts showed evidence of both intraluminal hemorrhage and subepithelial fibrous reaction with hemosiderin deposits.

Treatment. Since Peán performed the first recorded splenectomy for a splenic cyst in 1867,[5] splenectomy has been the standard treatment for such lesions. Simple evacuation, intraoperative or percutaneous, or catheter drainage via the transabdominal or retroperitoneal approaches have been unsuccessful in preventing recurrences. Subsequent splenectomy was required after these failed attempts.[2] Splenectomy has been advocated as it relieves symptoms, alleviates the disfiguring abdominal protuberance, confirms the nature of the mass, and removes the possibility of rupture.[6] However, removal of the cyst alone can also accomplish these goals without the need for total splenectomy. Furthermore, the hazards and severity of overwhelming postsplenectomy sepsis in children following splenectomy for trauma is well documented, and the value of splenic conservation when possible must be emphasized.[7] With a better understanding of the segmental blood supply to the spleen and improved hemostatic techniques, splenic salvage has become a major focus in splenic surgery. Successful splenic cystectomy has been reported in eight patients with congenital splenic cysts.[6] These eight reported cases represent only 6% of all patients with congenital splenic cysts who were surgically treated. The remainder had splenectomies.[6]

The current accepted practice of nonoperative management of splenic injuries due to blunt abdominal trauma will likely result in an increased clinical experience with splenic cysts. Although the potential failure of splenic salvage must be recognized,[8] partial splenectomy with cystectomy for splenic cysts not involving the hilum is recommended whenever possible.

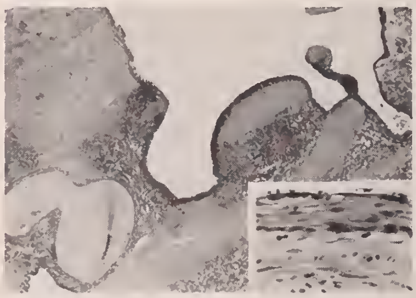

FIGURE 4. **Case 2:** Section of the splenic cyst wall with subepithelial fibrous bands and foci of old blood (hematoxylin-eosin stain, × 25); Insert: squamous epithelial lining with intercellular bridges (hematoxylin-eosin stain, × 400).

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Malignant melanoma of the anal canal

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Malignant melanoma of the anal canal is a very rare entity; it comprises only 0.25% to 1.25% of all malignant tumors of this anatomic region. It is a high-grade invasive cancer, and eventually all patients die of widespread metastasis, usually to the liver and bones. A ten-year review of the records of the Western Reserve Care System tumor registry disclosed only one patient with melanoma among 20 patients with primary malignancies arising in the anal canal. The patient, a 90-year-old woman, was treated with wide surgical excision. There was no recurrence at 28 months. The patient is presented in this report with a review of the literature.

CASE REPORT

A 90-year-old woman presented with rectal bleeding and was found to have a thrombosed hemorrhoid. A large indurated mass, incorporated with the thrombosed hemorrhoid, was excised. Microscopic examination revealed malignant melanoma of the anal region.

The patient's medical history revealed that 20 years previously she had undergone excision of a well differentiated endometrial adenocarcinoma within an endometrial polyp 2.5 × 1.5 × 0.5 cm in size. After a total hysterectomy and bilateral salpingo-oophorectomy, she was followed for 20 years without any recurrent carcinoma being observed.

The patient was admitted for a wide excision of the anal malignancy in September 1985. Physical examination upon admission was essentially within normal limits. There was no evidence of any other primary tumor. The patient tolerated the surgical procedure very well, progressed satisfactorily during her hospital stay, and was discharged five days later. She has been followed for 28 months without any evidence of recurrence.

Routine histologic examination of the primary resected lesion, which measured 2 × 1.5 × 1 cm, revealed large nodules of melanoma present within the anal mucosa (Fig 1). These nodules closely abutted on the anal squamous epidermal surface. Fontana-Masson stain disclosed a highly pigmented cellular tumor (Fig 2). S-100 immunoperoxide stain demonstrated a strong positive result. In most areas, the epithelium over the melanotic nodules was thin, and no intra-epidermal malignant melanocytes were seen. However, in one region a melanotic nodule appeared nearly contiguous with an area in which there is a strong suggestion of junctional activity of the melanocytes at the lower border of the epidermis. The specimen from the wide excision revealed only nonspecific chronic inflammation and subcutaneous edema without any residual melanoma.

DISCUSSION

Anorectal melanomas are very rare. According to Pack and Oropeza,[1] they comprise only 0.25% to 1.25% of all malignant tumors of the anal canal. Perzin et al[2] described a 1.6% incidence of anorectal melanomas among all melanomas. The skin and eye are the more common sites of origin of these tumors. Morson and Volkstadt[3] stated that approximately one malignant melanoma of the anal canal is seen for every eight squamous cell carcinomas of the anus and anal canal, and one for every 250 adenocarcinomas of the rectum. An evaluation of the incidence of anal melanoma by Scotto et al[4] from the National Cancer Institute in 1976 disclosed only 0.016 per 100,000 population per year, which means only 36 new cases would be diagnosed each year in the United States.

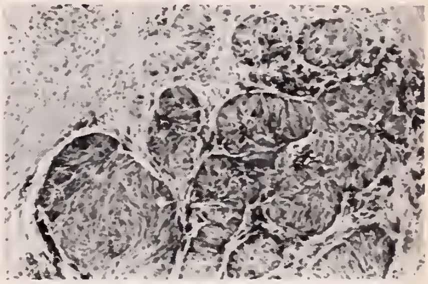
FIGURE 1. Large pigmented nodules of melanoma, consisting mainly of spindle-shaped cells with nuclear pleomorphism, within the anal mucosa (hematoxylin-eosin stain; original magnification ×400).

Mills and Cooper[5] described only four documented sites for primary malignant melanoma in the digestive system: the oral cavity, esophagus, gallbladder, and anal canal. Allen and Spitz[6] suggested that to be interpreted as primary, a mucosal melanoma should ideally have overlying or lateral junctional change. The absence of this important feature should lead to careful evaluation of the patient in order to

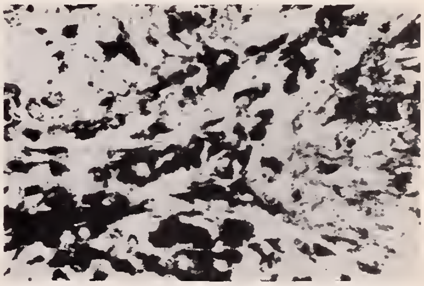
FIGURE 2. Fontana-Masson stain showing highly pigmented cellular tumor containing melanin (original magnification ×400).

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rule out other primary lesions. Only three of the 15 cases reported by Morson and Volkstadt[3] and seven of 17 cases reported by Mason and Helwig[7] were found to have junctional activity.

A review of the records of the Western Reserve Care System tumor registry disclosed only 20 cases of primary malignant tumor arising from the anal canal during the previous ten years. This included ten cases of squamous cell carcinoma, eight cases of basaloid carcinoma, one case of undifferentiated malignant neoplasm which stained faintly with S-100 antigen, and one case of malignant melanoma.

In a study by Mills and Cooper,[5] patients with anal melanoma were found to range in age from 22 to 96 years of age, with an average age of 58 years. Morson and Volkstadt[3] reported 15 patients who varied in age from 14 to 75 years, with an average age of approximately 59 years. This age distribution is much the same as the age distribution for adenocarcinoma of the rectum and squamous cell carcinoma of the anal canal. Anal melanoma affects males and females almost equally, as compared to a preponderance of women for squamous cell carcinoma of the anal canal and a preponderance of men for adenocarcinoma of the rectum.

The tumor arises from the transition zone between skin and mucosa of the anal canal near the dentate line. Mills and Cooper[5] stated that 67% of the 156 cases reported were concentrated in the proximal portion of the pecten and in the transitional zone. The most frequent complaints are rectal bleeding or the presence of a mass in the anal canal. Other symptoms include pain, prolapse, loss of weight, change in bowel habit, and constipation. Hemorrhoids and polyps are the most frequent misdiagnoses.

Melanoma is the most aggressive malignant tumor affecting the anorectum. Rosai[8] described the prognosis of anal melanoma as invariably "hopeless." The average life expectancy after diagnosis is approximately five months. The most frequent sites of metastasis are regional lymph nodes, liver, lungs, and bones. Although there were no survivors after 18 months in the 15 cases reported by Morson and Volkstadt,[3] Freedman[9] reported two advanced cases with mesenteric lymph node involvement in which survival was longer than ten years. The reasons for the poor prognosis described by Pack and Martins[10] include the inadequacy of early treatment, the delay in diagnosis, the rapidity of dissemination of the tumor, and the high degree of malignancy. Friedman and Bauer[11] suggested that, in general, anal melanomas thicker than 2 mm have a poor prognosis, while those less than 2 mm in thickness have a relatively good prognosis.

The treatment of melanomas of the anal canal depends on the stage of the disease and the location of the tumor. Patients with evidence of metastatic disease and those with large tumors should have only local excision of the tumor performed. If there is evidence of obstruction, then a diverting colostomy suffices. The prognosis for these patients is extremely poor.

Patients with disease localized to the anal canal should undergo an abdominoperineal resection; however, a palliative result and an occasional cure is the most one can expect. Encouraging results were reported by Baskies et al[12] using a posterior pelvic exenteration with in-continuity groin and pelvic nodal dissections. They had a 50% five-year survival rate in six patients. Likewise, Wanebo et al[13] reported five-year survival rates in some patients treated in a similar fashion. After observing the lack of uniform results with the different surgical modalities, Quinn and Selah[14] concluded that there is no good evidence to support one form of therapy (surgical resection) over another. Indeed, as Stearns et al[15] have suggested, survival is related more to the stage of the disease and thickness of the tumor than to the mode of treatment.

Gastrointestinal malignant melanoma is a relatively radioresistant neoplasm. Radiation therapy has never been proposed as a primary treatment, but Stearns et al[15] have suggested that it is occasionally useful in metastatic disease to the lung, central nervous system, and bone.

There are some chemotherapeutic protocols described by Stearns et al[15] that have shown some response in late stage melanoma. Boey et al[16] reported a 20% response rate to decarbazine (DTIC) for disseminated melanoma. However, no significant increase in survival was observed. It is a real hope that tumor-specific monoclonal antibodies linked to various toxins, as studied by Oldham et al,[17] may prove useful in the future. For the time being, complete surgical excision remains the treatment of choice for primary malignant melanoma of the anal canal.

Acknowledgments. The authors thank Dr Armin V. Banez, Director of Endoscopy and Proctology, Western Reserve Care System, for permission to use the case, and Mrs Zora L. Kosanyar and Miss Diane E. Booty for their secretarial assistance.

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LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Paget-Schroetter syndrome in a patient on the contraceptive pill

TO THE EDITOR: The paper on deep vein thrombosis of the upper extremity by Cohen and Dubin[1] was interesting, but did not clarify the question of management mainly because of the lack of comparison between the groups. One is not informed as to the degree of anticoagulation achieved, and this could be relevant to the resultant degree of disability, which seemed to be the main issue. In Cohen and Dubin's study, 33% of the patients had effort thrombosis, which is interesting, as it is a rare occurrence among patients with an uncommon type of deep vein thrombosis. This form of thrombosis has also been called the Paget-Schroetter syndrome and mainly occurs in young males.[2] Hence it would have been helpful to know the gender of the patients in the effort thrombosis group. Furthermore, other predisposing factors to thrombosis such as drugs, family history, smoking habit, or hematologic abnormality should have been stated.

We recently treated a 15-year-old girl who was admitted with painful swelling of her right arm for a week. A diagnosis of a right subclavian vein thrombosis was confirmed by phlebography. In the absence of any other physical, biochemical, or hematologic abnormality, the cause was thought to be due to effort. However, the patient had been taking a combination contraceptive pill preparation of 35 μg ethinylestradiol and 500 μg northisterone, and she also smoked 50 cigarettes a day. Her symptoms incompletely resolved on full anticoagulation with 6 mg of warfarin per day.

The patient presented probably developed thrombosis as a result of the factors mentioned, but further examination of the "effort" group is required to identify predictors of outcome, especially as these patients seem to do poorly with treatment.[1] Furthermore, this complication should be borne in mind when prescribing contraceptive pills to younger adolescents.

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1. Cohen JR, Dubin M: Deep venous thrombosis of the upper extremity. *NY State J Med* 1988; 88:177–179.
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Professional medical conduct

TO THE EDITOR: I am responding to the letter from Richard Pearlman, MD, in the November issue in which he expresses his concerns over the changing nature of the practice of medicine.[1] Specifically, he expresses concern over the way in which the Office of Professional Medical Conduct in the Department of Health carries out its legislative mandate to investigate complaints of misconduct and conduct formal disciplinary hearings in that small percentage of cases which warrant a hearing (5% of complaints in 1988).

I would like to correct the record. As I described in my presentation to the Richmond County Medical Society on Staten Island, the office receives several thousand reports or complaints each year. These complaints, as required by law, are all reviewed and resolved simply or investigated by one of the department's regional offices throughout the state. At the conclusion of the review or investigation, the vast majority of complaints are dismissed. The reports are confidential, pursuant to the Public Health Law, and cannot be released under the Freedom of Information Act or by subpoena.

Those cases that are not dismissed upon initial review or investigation are considered by a committee of the New York State Board for Professional Medical Conduct, consisting of two physicians and one lay member of the board, which recommends the disposition of the case—either dismissal, additional investigation, or a formal hearing. All information gathered in the course of our review, including the physician's response to the concerns, is presented to this committee.

If the committee of the board determines that there is sufficient information or evidence of a possible violation, the physician is provided with a formal hearing to defend against these charges before another committee of two physicians and one lay member of the Board for Professional Medical Conduct. Dr Pearlman's assertion that "the usual rules of due process are suspended" is without basis. They are, in fact, closely adhered to. Due process requires that physicians be given adequate notice of the charges against them, an opportunity to defend against these charges, and an opportunity to present their own evidence before impartial decision makers.

Contrary to Dr Pearlman's belief, the accused physician does have adequate notice of the charges (in writing) in advance and full opportunity to confront the charges, on the record, before a committee of the board. Many professional medical conduct hearings go on for days.

It is important to underscore that the critical decisions in the professional medical conduct proceedings—the decision to bring a case to hearing and the findings of fact and recommended penalty in a hearing—are made by the members of the Board for Professional Medical Conduct. These are practicing physicians throughout the state nominated to serve on the board by their county medical society, the state medical society, their specialty society, or the New York Academy of Medicine.

In my experience, the members of the Board for Professional Medical Conduct who serve on these committees give careful thought and consideration to each decision they make.

I regret both the misinformation and tone of Dr Pearlman's letter. My presentation to the Richmond County Medical Society, as to other societies and medical organizations and hospitals, was intended both to shed light on the activities of the office and to encourage members of the society to participate in our work. It is only through the active involvement of the practicing physicians that the profession can truly be effective in identifying those we need to help for problems of substance abuse or winnow out for incompetent or unethical practices.

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1. Pearlman RL: Professional medical conduct. *NY State J Med* 1988; 88:603-604.

In reply. I believe Ms Tanner has missed the point of my letter published in the November 1988 issue of the *Journal.*[1] I do not argue or disagree with the goals of the Office of Professional Medical Conduct. It is the process with which I take issue, and the pervasive attitude that seems to have been promulgated by the bureaucratic structure. To my way of thinking, the review process prior to formal hearings assumes the guilt of physicians rather than their innocence. Physicians under investigation need not be notified prior to the decision for formal hearings. While I am thankful for written notice in advance of formal hearing procedures, the fact that physicians need not be informed of the investigation nor their input necessarily sought disturbs me greatly. This is particularly true of those cases reported and investigated anonymously.

The possibility of punishment is devastating and includes the loss of license and reputation. I do not think it appropriate to leave decisions about going on to formal hearings to individuals who did not have input, information, or clarification from the physician in question. I believe this should be sought from the outset of a complaint and should be a mandated step, not one left to the discretion of a committee or of an investigator.

From the perspective of aiding physicians to practice better, more effective, and competent medicine, one would like to think that the Office of Professional Medical Conduct would notify physicians that an investigation is in process or has taken place. This can only be done in a spirit of cooperation that relies on the acceptance of innocence as primary. "Due process" is based, as I learned it, on the assumption of innocence with the burden of proof on those who intimate guilt. The system as currently described by Ms Tanner does not dispel my belief that this process has been perverted.

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1. Pearlman RL: Professional medical conduct. *NY State J Med* 1988; 88:603-604.

Legislator-physician relationships

TO THE EDITOR: One of the seeming difficulties in our continuing struggle with the government is a mutual inability to understand each other's mission. Physicians are primarily concerned with the health care of their patients. Legislators are responding to political and social mandates. To the physician, the legislative process is complex, often contradictory, and given to excessive regulation. To the legislator, medical care is overly expensive and primarily targeted to the well to do. We both are given to labels and stereotypes. They view us as the greedy, Mercedes-Benz acolytes and we, at times, refer to them as those characters in Albany and Washington.

As a large customer for medical services the government has developed an intense interest in patient care. Like the lawyers, legislators have become an integral part of health care and we must adjust to their presence and their power. We must become students of the philosophy and mechanics of the legislative process. We should build a relationship of credibility and trust which allows them to consult us before their subcommittees have begun to consider a possible piece of pertinent legislation. By acting as an honest resource, we can help to shape the reports. Our input should be received prior to the decision making.

In planning for this undertaking we need allies. Our one set of steadfast allies are the auxiliaries which are often composed of savvy ladies who have been protecting our flanks for years. It has to be understood that beyond that group, coalitions can become tomorrow's adversaries. Given a future congruence of interests we may well work together on other issues. In the same vein our legislators may be adversaries or allies depending on the circumstances.

We must also learn to accept the fact that in large legislative systems as diverse and fractious as those we face, change is often incremental, a series of compromises by a multiplicity of stakeholders. These systems require us to be innovative, patient, and persistent. Trips to Washington and Albany are more effective if they have been preceded by meetings in the local offices of our legislators.

We need to learn more about the staffs of our legislators. Very often these talented individuals are the pivotal players in fashioning a bill. By the nature of the structure, they are often the unsung spear carriers who make their bosses look good. Although their input has to be shaped to fit the philosophy of the incumbent, this window of possibilities is worth exploring.

Nurturing the legislator-physician relationship is a high priority item. We cannot leave it entirely in the hands of our lobbyists if we hope to be successful. The future of our profession is inextricably bound to this relationship. To ignore it would be fatal.

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The *New York Medical Journal* and cigarette smoking

TO THE EDITOR: During the course of research for a book on the history of smoking I came upon an editorial in the *New York Medical Journal* of July 25, 1914, clearly intended to remove the then existing prejudice against cigarette smoking[1]: "Particularly do the uninformed . . . enjoy an attack on the cigarette; it is cheap, it is small, and its patrons, numerous as they are, yet form an insignificant minority in our immense population. Therefore the cigarette and its users are fair game for cheap and silly sneers; sneers which are capable, however, of cowering an entire legislature . . . Yet beyond cavil, it has been proved scientifically that of all methods of using tobacco cigarette smoking is the least harmful."

The editorial goes on to say that although employers are "well within their rights in forbidding cigarette smoking . . . they overstep their bounds when they condemn smoking . . . especially when [it] affords . . . solace and recreation to millions perfectly capable of judging what is and what is not good for them. In Europe, where a good deal of logical thinking still prevails, there is probably not one smoker of distinction in any walk of life who does not include the cigarette in his nicotian armamentarium."

That one of the leading medical journals of its time could be so wrong and so prejudiced in its scientific and sociocultural pronouncements is a humbling reminder of how not so very long ago the medical profession was as much hostage to bad science and prevailing cultural dogma as the contemporaneous lay public.

The *New York Medical Journal* eventually went to its reward (some might say fortunately). The *New York State Journal of Medicine* (which had no relationship with the *New York Medical Journal*) has for decades now consistently editorialized against cigarette smoking, providing proof in the immortal words of the tobacco sloganeers that the medical profession has "come a long way, baby."

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1. The luckless cigarette. *NY Med J* 1914; 188-189.

FROM THE LIBRARY

THE ROLE OF THE PHYSICIAN'S PERSONALITY IN MEDICAL PRACTICE (PSYCHOTHERAPEUTIC MEDICINE)

Physicians are constantly in search of ways and means to improve their professional skill. This is a source of justified pride, and we cherish the tradition. It is in this spirit that the present paper is offered.

Any kind of self-examination is painful. We do not like to find ourselves "less than perfect." This accounts for the doctor's almost exclusive emphasis on the *patient* and what is wrong with *him*. The tacit assumption is made that the doctor is a constant. It is important that physicians become aware of themselves as people. Their personalities are an integral part of their therapeutic armamentarium.

All physicians practice psychotherapy. Some do so with conscious intent. Others are relatively unaware of the effect on their patients of their manner, words, and actions. It is inevitable in a relationship between any two human beings that they will exert some effect upon one another. This is especially true in the case of a doctor-patient relationship. When the physician keeps this in mind, he can use it to therapeutic advantage.

It is well known that patients bring with them to the doctor their individual conscious and unconscious attitudes and biases. For most people he represents some combination of man-of-science, father-confessor, adviser, and even a bit of magician. Just as a sick child looks to his parents, so a sick adult looks to his doctor to make him "all better." The patient's expectations and the physician's response to them play a part in determining the outcome of the illness. His valuation of the prescription given him, his likelihood to carry out therapeutic orders, his sensitivity to any hint of optimism or pessimism on the doctor's part, etc., are all involved. We can scarcely hope to fill all the roles patients assign to us, but we can learn how to use their attitudes and expectations to help them. In so doing we shall be practicing not only psychotherapy but good medicine. . . .

BERNARD V. STRAUSS, MD
(*NY State J Med* 1951; 51:753–757)

The wickedest woman in New York

The Wickedest Woman in New York: Madame Restell, the Abortionist. By Clifford Browder. 217 pp. Hamden, Conn, Archon Books, 1988. $25.00 (hardcover)

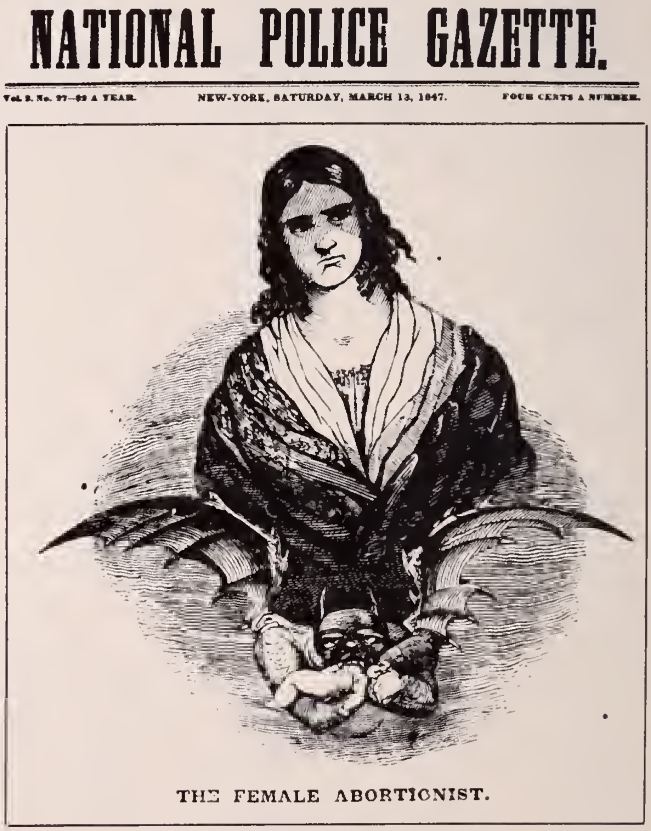
Despite its puerile title, *The Wickedest Woman in New York: Madame Restell, the Abortionist* is fun to read. The author, freelance journalist, editor, and writer Clifford Browder, has dipped with relish into the newspapers and public records of Victorian New York to produce not only a biography, but a fascinating picture of the attitudes of the mid-19th century towards the seamier side of sex. In tracing the lurid yet banal career of one of the foremost abortionists of the day, he manages to teach us a great deal about the social conditions which left the management of unwanted pregnancies to lay persons brave enough to risk all for money.

The writing is first class, an important factor for those not content merely to be entertained or to learn, but truly appreciative of the language. The portrait drawn of the young, maverick, and fearfully ambitious British immigrant is challenging and multidimensional. We see her rise from dire poverty on the Lower East Side of New York to become mistress of a carriage trade, as well as devoted wife and mother of one. When we say farewell to our protagonist as she lies in the bathtub like Marat, exsanguinated and anticlimactic, we are still unsure of her character. Was she a pragmatic helper of women in distress, or a greedy opportunist with enough chutzpa to maintain a 40-year reign of uterine sociopathy?

The story of a woman ever ready to tweak the noses of the staid New York establishment by aborting its wives, mothers, and daughters in dead of night somehow casts the 20th century struggle over abortion in a whole new light. For this reason, I would recommend the book highly to all persons interested in the history of the women's movement, as well as all medical personnel, particularly those for whom abortions constitute a significant portion of their income. I would also suggest the book to devotees of Victoriana, New York City history buffs, journalists and historians, and all persons lacking in confidence. The brazen gall of Madame Restell should be a lesson to us all.

Faithfully and steadily, the author draws upon contemporary news accounts to show us how one woman, aided, abetted, and prodded by her husband, managed to establish a virtual monopoly on the one personal service most highly prized by half the population of the city. Through a canny knowledge of PR which would put most of Madison Avenue to shame, and word of mouth (several million of them, actually), she managed to define her target population, secure it, retain it, milk it of vast riches, and only spend one year in the hoosegow (where, characteristically, she saw to it that she had preferential treatment and all that bribery could buy). She survived not only the mild disapprobation of a once fairly accepting public, but the increasing viciousness of an establishment rapidly Victorianizing in its pursuit of the sexually wicked.

When she was brought down at last, and forced into suicide the night before a trial fomented by a crusader who had devoted his life to her destruction, we are actually feeling sorry for the flamboyant wretch. We regret that Fifth Avenue will no longer see her coach and four, flaunting its gaudy finery in the faces of the bluebloods who financed her mansion at its upper end (next door to the highest church of the day). We no longer look askance at the woman who murdered a few young women, yet probably saved thousands more from a life of misery. We no



NATIONAL POLICE GAZETTE.

Vol. 2. No. 27—$2 A YEAR. NEW-YORK, SATURDAY, MARCH 13, 1847. FOUR CENTS A NUMBER.

THE FEMALE ABORTIONIST.

FIGURE 1. This "purported likeness" of Madame Restell was originally published in the *National Police Gazette* (March 13, 1847), and is reprinted from *The Wickedest Woman in New York.*

longer deplore the quack to end all quackery, who made a fortune on pills, potions, devices, books, and lectures on what every fecund woman should know. We are actually moved at her depression, a woman finally alone, husband dead, child a disappointment, the lust for life fading away. We see another colorful personality lost to our wagging tongues, a real life Belle Starr, no longer around to unmask the hypocrisy of the establishment.

It is a tribute to the author that such a readable and apparently highly reliable character study is produced, given that this real-life woman left behind no notebooks, letters, or memorabilia upon which such studies characteristically draw. We will remember her and feel that she gave us new insights into the feelings and fate of thousands of unfortunate women trapped by biology and offered no way out by the traditional healers of the day. Surely this woman, and her less awesome imitators, shaped the history of our medical profession as much as the giants whose motivations and accomplishments were less equivocal.

NAOMI R. BLUESTONE, MD
Consulting Editor

FROM THE LIBRARY

MADAME RESTELL, AND SOME OF HER DUPES

It is with feelings of regret that we deem ourself called upon to make some disclosures of the infamous practises of this notorious fiend in human form. No one will doubt, that we are surrounded by learned and scientific medical men, whose skill has not only been appreciated by thousands of our fellow creatures, the subjects of pain and sickness, but whose acuteness of perception in the various diseases which our race is heir to, and the deep love for the profession of their choice, which is manifested by their devotion to it,—have been repaid by tears of joy from many a fond mother, devoted father, and anxious husband. Yet! amidst all this skill and devotedness on the part of the Profession, and enlightenment and wholesome laws on the part of the people, we behold Quacks, of every grade, practising their charlatanism upon the unwary, and becoming millionaires from the fruits of their wickedness. We have been among the number, who have thought best hitherto, to let the people discover for themselves, the motives of the various pretenders in the healing art, which, alas! they too frequently do, when it is too late to repair the sad injury, and the silent grave closes over the dupe who has been the victim of quackery. If the modest Physician is heard to raise his voice against the practice of any of the popular mountebanks of the day, he is immediately accused of selfishness, and the plea of persecution is urged on the part of the Charlatan. But the time has arrived, when "forbearance ceases to be a virtue. . . ."

. . . This creature's advertisements are to be seen in our daily papers; there she invites the base and the guilty, the innocent and the unwary, to apply to her. She tells publicly what she can do, and, without the slightest scruple, urges all to call on her who may be anxious to avoid having children. Here, then, is a premium offered for vice, to say nothing of the prodigal destruction of human life, that must necessarily result from the abominations of this mercenary and heartless woman.

With all the vigilance of the police of our city, and with every disposition, I am sure, on the part of the authorities to protect public morals, and bring to merited punishment those who violate the sanctity of the law, this Madame Restell, as she styles herself, has as yet escaped with impunity. . . .

EDITORIAL
(*NY Med Surg Rep* 1846; 1:158–165)

Reprinted from the December 1988 issue of Epidemiology Notes (*Vol. 3, No. 12*), *published by the Division of Epidemiology, New York State Department of Health, Albany, NY.*

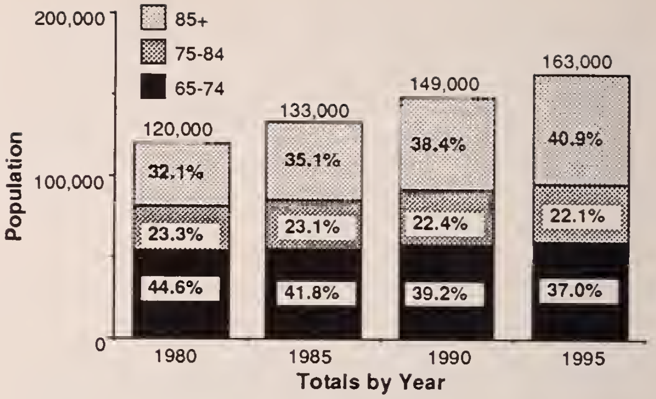


FIGURE 1. Estimated total population diagnosed with severe dementia in New York State. (Note: Official population projections of the New York State Department of Commerce were used to estimate total dementia cases calculated at 5% of age 65–84 and 20% of age 85+.)

Alzheimer's disease and other dementias registry

Alzheimer's disease is a neuropsychiatric disorder resulting in progressive and irreversible cognitive impairment due to cellular destruction within the brain. Alzheimer's disease is not considered to be a natural consequence of aging, despite a linear association with age. About 5% of individuals over age 64 are estimated to have dementia of varying severity, with prevalence increasing sharply over age 80. Several studies indicate that approximately 60% of all dementing illness may be diagnosed as Alzheimer's disease.[1] The New York State Department of Health's Alzheimer's Disease and Other Dementias Registry has estimated that by 1995 there will be approximately 163,000 New York State residents over 65 years of age with severe dementia including Alzheimer's disease (Figure 1). These estimates were derived by applying published age specific prevalence rates to population projections.[1,2]

Mortality attributable to dementia in New York State is significant. However, only 445 deaths due to Alzheimer's disease were recorded in 1987.[3] Most deaths among Alzheimer's disease patients are recorded as pneumonia, urinary tract infections and other infectious diseases.[4] Data from the New York Statewide Planning and Cooperative System (SPARCS) uniform hospital discharge data set indicate the disparity between related service utilization and reported cause of death. Over 36,000 hospital discharges were reported in 1986 for 34 reportable dementia International Classification of Diseases, Ninth Revision Clinical Modification (ICD-9-CM) codes.

National estimates of the costs of dementias indicate that $13.26 billion are spent on direct costs and twice that amount is spent on indirect home care.[5] Individual patient care costs were estimated at $17,643 annually by researchers at the Hoover Institution.[6] The annual care cost for New York State is estimated to be $2.17 billion.[7]

The Alzheimer's Disease and Other Dementias Registry was established as a result of legislation which amended the New York State Public Health Law. The amendment was signed into law in July 1986 and requires that physicians and medical care facilities report the diagnosis or confirmation of Alzheimer's disease and other dementias to the Commissioner of Health. Reporting began January 1, 1987 and was defined as information the Commissioner of Health deemed necessary for research purposes on the epidemiology, cause and prognosis of Alzheimer's disease. Additional uses of the registry data will include health care planning, education and improved diagnosis and treatment for patients with Alzheimer's disease or other dementia and their family caregivers.

Confidential case reports (DOH-1988, 2/87) are required for 34 ICD-9-CM codes for dementia or dementia causing diseases. Each of the 34 codes was selected on the basis of progressive irreversible dementia with and without important neurological findings as were diseases which may or may not result in dementia, such as chronic infectious diseases (neurosyphilis, HIV), nutritional disorders (Korsakoff's) and vascular disease (cerebral atherosclerosis). Over 18,000 reports of dementia have been submitted to the registry to date. Analysis of early reports indicates that 59% were for dementias other than Alzheimer's disease.

One important reason for requiring reports based on chronic irreversible dementia is that the division between reversible and irreversible is both clinically and epidemiologically useful in studying the differential diagnosis of dementia. A small percentage of dementias (10–15%) are considered to be arrestable or reversible through management and treatment. The treatable dementias include normal pressure hydrocephalus, brain tumors, hypothyroidism and subdural hematomas.

Since the definitive diagnosis of Alzheimer's disease is only made upon direct examination of brain tissue, usually at autopsy, a necessary part of the registry operations will involve evaluation of the validity of registry reports. There are two methods for evaluating the validity of case reports. The first is through comparison of the clinical work-up for individual patients reported to the registry to a predetermined clinical diagnostic criteria that would serve as a standard. Diagnostic guidelines have been developed by the National Institute of Neurological and Communicative Disorders and Stroke–Alzheimer's Disease and Related Disorders Association (NINCDS-ADRDA) work group,[8] the National Institutes of Health,[9] the National Institute on Aging,[10] the New York Academy of Medicine/New York State Department of Health[11] and by researchers and consortiums of university based Alzheimer's disease registries. The second method of case report validation is by comparison of individual case report diagnosis to an autopsy diagnosis. Both methods require the performance of retrospective analysis of registry case reports.

The New York State Alzheimer's Disease and Other Dementias Registry has been designed to reduce or prevent bias in the data. A necessary part of developing a useful tool for epidemiologic analysis will be the maintenance of high standards of quality control and evaluation of registry data completeness, validity and reliability. As the nation's first state population-based dementia registry, the New York State Alzheimer's Disease and Other Dementias Registry must maintain these standards in order to foster progress in the care and research of Alzheimer's disease.[3,7]

Other Gerontology Program initiatives in Alzheimer's disease include the Alzheimer's Disease Assistance Centers (ADAC) and the Alzheimer Disease Community Services. The ADAC network provides diagnostic, educational and case management services to patients and family caregivers. The Alzheimer's Disease Community Services Program provides funding for local Alzheimer's disease community organizations to provide support, training, respite, information and referral services for Alzheimer's disease patients.

Further information on the registry may be obtained by contacting Todd Gerber, Alzheimer's Disease and Other Dementias Registry, Bureau of Adult and Gerontological Health, Corning Tower, Room 557, Albany, NY 12237-0620, (518) 473-0622.

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Burn injuries and deaths

In New York State, as well as nationwide, nonintentional injuries to children are the leading cause of death and a major cause of disability. In the home, the number of childhood injury deaths from burns for ages 0–14 is more than double the number for other types of injuries. In 1986, the total number of reported burns sustained by 0–14-year-olds in the state was 1,624 (30.9%), with the highest incidence for all age groups occurring in 0–4-year-olds at 1,142 (21.7%).[1]

The highest death rate from fire and smoke was 5.1 per 100,000 for infants followed by 4.6 per 100,000 for adults age 65 and over. For those 65 and over, it was the second leading cause, behind falls, in home injury deaths. A 1985 report on New York State resident death rates by race and age shows that black males and females ages 0–14 and 65 and over are at a higher risk for deaths than white males and females in the same age group.

Burn Injuries. Injury due to burns was analyzed from 1986 hospital discharges from five high-risk cities. The highest rate for burn injuries occurred in children under 15 at 99.6 per 100,000, followed by adults over 64 at 73 per 100,000. This compares to a rate of 56.1 per 100,000 for all ages combined.[2] The leading cause of all burns reported to the State Office of Fire Prevention and Control in 1986 was hot liquid burns in children 0–14 with 51.5% (905) of the 1,752 burns from hot liquids, followed by 50.4% (238) of 472 burns from contact with hot objects, 23.7% (147) of 620 cooking burns and 15.2% (67) of 440 burns from structure fires.[1] In children ages 0–4, the predominant nonfatal burn injury was scalds from hot liquids and beverages or from their containers.

Residential Structure Fires. The New York State Department of State's Office of Fire Prevention and Control reported that in 1985 there were 64,128 building fires in New York State; 39,491 (61.6%) were residential. Nearly 75% of residential fires outside New York City were in one- and two-family dwellings. In New York City, 82% of residential fires occurred in apartment buildings. A total of 455 civilians died in residential structure fires. Of these, children under nine, adults 20–29 and adults over age 70 accounted for the highest percentages. Most residential fires occurred during the late night hours when most people are asleep.[3]

Smoke Detector Use. In 1984, 65% of residential fires (excluding New York City) took place in homes that did not have smoke detectors. Only 21.5% had working smoke detectors, while 11.4% had detectors that failed to operate.[4] A 1984 study analyzed residential fire fatalities and the use of smoke detectors and found that twice as many deaths per 1,000 residential fires occurred in homes where smoke detectors were absent (4.0 per 1,000) as compared to where they were present and operating (1.8 per 1,000). In homes that had smoke detectors that failed to operate, the death rate was found to be 2.8 deaths per 1,000 fires. A possible explanation for a lower rate in homes that had inoperative detectors as compared to homes where they were absent could be due to a heightened awareness of fire and the proper response to fire conditions.[4]

In 1985, 24 civilians died in fires caused by kerosene heaters, compared with eight deaths in 1984 and three deaths in 1983. The death rate in fires caused by kerosene heaters was 39 deaths per 1,000 incidents, compared with a rate of six deaths per 1,000 incidents for structure fires with other causes. Injuries followed a similar pattern with 83 per 1,000 incidents for kerosene heaters versus 23 per 1,000 incidents for other causes.[3]

Prevention. In 1985 in New York State, smoking accidents accounted for 32% of all fatal fires recorded and were the leading cause of fatal incidents. Fire-related injuries associated with certain health risk behaviors suggest the need for passive technology approaches. These approaches include fire safe (self-extinguishing) cigarettes, smoke alarms, automatic sprinkler systems and lowering of hot water temperatures. The passive prevention approach is the most effective deterrent to injury because it provides automatic protection rather than relying on people at risk to voluntarily alter their behavior.

The New York State Health Department's Injury Control Program is funding ten pilot burn injury prevention demonstration projects under a three-year grant from the US Department of Health and Human Services using a combination of passive and active (educational) approaches. These projects will provide for the installation of smoke detectors, lowering of hot water heaters and escape plans from burning structures. It will also include instructions on how to extinguish flames on a person and inspect households for burn hazards. Recipients of these burn prevention activities are rural low-income families with children under age 15, minorities and the handicapped.

Additional information is available from: Burn Injury Prevention Project, Injury Control Program, New York State Department of Health, Corning Tower, Room 621, Albany, NY 12237.

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Infectious arthritides

Joint involvement is frequently a manifestation of infection with a large number of pathogens which can include viruses, bacteria and fungi. The mechanisms for infectious arthritis are not entirely understood and represent a major investigative challenge. It is now clear, however, that there probably are multiple mechanisms that can ultimately induce the articular manifestations of infections.

Mechanisms of Infection

The most obvious mechanism involves direct penetration of the synovial membrane by infectious organisms. Punctures and other types of articular trauma can bring the infection directly into the joint where the organism can replicate. A number of both gram positive and gram negative bacteria cause articular infection in this manner. Sporotrichosis of the joints is often associated with rose thorn or other vegetable matter punctures and can be seen as an occupational mycosis of gardeners.

The second mechanism for articular infection involves the hematogenous spread of the organism. Once in the joint, the organism may be trapped and phagocytic cells and their products can add to the inflammation. Not all infectious organisms cause the same amount of injury. For example, *Staphylococcus aureus* is notable for rapid joint destruction, whereas *Neisseria gonorrhoeae* is not associated with severe and/or irreversible joint destruction.

Viral Infections

Viral disease often leads to joint manifestations. The most ubiquitous examples are the arthralgias resulting from influenza virus infections. Although a prominent clinical feature, the arthralgias of influenza infection are self-limited and generally need no additional intervention. A true, marked inflammation of the joints can occur in the prodromal phase of hepatitis B infection; that is, prior to the onset of clinically apparent liver disease. Approximately 20% of the patients develop articular manifestations that usually disappear with the onset of jaundice. During this phase, the hepatitis B surface antigen (HB_sAg) can be present in the blood and can be demonstrated in synovial tissue by immunofluorescence.

Both vaccine induced and naturally occurring rubella can cause arthritis, involving primarily women and children. In both cases, rubella virus has been isolated from synovial fluid during the acute inflammatory phase. As in other viral diseases, the arthritis is self-limited.

Many other viruses can cause articular disease and in some infections, the predilection for the joints is marked. O'nyong-nyong, an African mosquito-borne arbovirus, literally means "joint breaker," and Ross River virus, another arbovirus, has been implicated as the cause of epidemic polyarthritis in Australia.

Bacterial Infections

Gonococcal arthritis is one of the better known clinical manifestations of infection with *Neisseria gonorrhoeae*. Following disseminated gonococcal infection, approximately 0.5–1% of gonorrhea patients will develop a rather broad syndrome of articular manifestations ranging from migratory polyarthralgias to inflammatory arthritis with purulent joint effusions. *Neisseria gonorrhoeae* is not easily recovered from the synovial fluid or infected tissue with actual isolations occurring less than 50% of the time. Clinically, gonococcal arthritis has some features that set it apart from other bacterial arthritis. There is evidence to suggest that host associated factors such as complement deficiencies play a role in this entity.

Tuberculosis arthritis incidence has decreased in recent decades. When all the extrapulmonary manifestations of tuberculosis are considered, arthritis is a relatively rare event. This arthritis bears many clinical similarities with arthritis caused by fungal infections or by other atypical mycobacteria. Tuberculosis of the spine, also called Pott's disease, attacks the thoracic and lumbar vertebrae often leading to collapse of the affected vertebrae. In other joints, the findings of the synovial fluid are variable, but unlike other bacterial arthroses, synovial fluids and tissues yield the acid-fast mycobacteria with regularity. Granulomatous changes are often seen in the synovium but these can also be caused by other mycobacteria; specific identification is necessary.

Fungal Infections

The frequency of candidal arthritis has increased in recent years reflecting the increased incidence of systemic candidiasis in patients at high risk for opportunistic infections. Premature infants account for the majority of pediatric cases, while in adults, it is seen in intravenous drug users, patients undergoing parenteral therapies and in cancer and AIDS patients. Candidal arthritis occurs mostly in patients with systemic disease and involves the large joints often with adjacent osteomyelitis. Unlike tuberculosis arthritis, Candida organisms are rarely detected in synovial fluid or in synovium.

Reiter's Syndrome

The first case of Reiter's syndrome was described in a patient with a complex syndrome of arthritis and uveitis following a sexually acquired urethritis. Another patient had an illness marked by arthritis, urethritis and conjunctivitis, following an episode of enteritis. These initial cases summarize the most common presentations of Reiter's syndrome. The association of the systemic onset of Reiter's with a preceding sexually acquired urethritis or an illness with enteric bacteria has never been causally proven, but persuasive epidemiologic evidence has just about conclusively allowed for the inclusion of Reiter's syndrome among the arthritides of infectious origin. Urethritis caused by *Chlamydia trachomatis* is the most common precipitating infection in the United States. Occasionally, this agent has been isolated from the conjunctiva, urethra and even synovium and synovial fluid. Antibodies to the chlamydia are present in many patients.

The postdysenteric form of Reiter's syndrome can be precipitated by infection with shigella, *Yersinia enterocolitica*, and to a lesser extent with salmonella. The two precipitating infections (sexually acquired urethritis and enteric disease) cause nearly identical clinical manifestations. Typically, Reiter's is a multisystem disease involving the joints, the skin, the genitourinary tract, the eyes and other organs. Reiter's arthritis is of the reactive type, in which the synovial fluid and tissue are sterile.

Early reports of familial aggregation of Reiter's led to the investigation of a possible genetic basis for the syndrome. The HLA-B27 phenotype is found in approximately 75% of the patients and is present in a similar frequency in sexually acquired urethritis and postdysenteric disease even though neither clinical chlamydial urethritis or gram negative bacterial enteritis alone are associated with the HLA-B27 phenotype. Patients with these precipitating infections appear to be at a greater risk for developing Reiter's syndrome if they express the HLA-B27 phenotype.

Immunologic processes play a role in infectious arthritides, as exemplified by the predisposing influence of the HLA-B27 phenotype but the mechanisms involved require further research.

BEGINNING CHILD PSYCHIATRY

By Paul L. Adams, MD, and Ivan Fras, MD. 620 pp. New York, Brunner/Mazel Publishers, 1988. $50.00 (hardcover)

As the data, accumulated experience, and concepts change in a given field, there is need for a coherent document to reflect these changes. This is especially true of the rapidly developing body of knowledge which constitutes the field of child psychiatry. A textbook is one vehicle in which these changes may be reflected. There are, of course, monographs on specific aspects of a field, and there are compendia of papers, written by many different authors, tied together by the efforts of an editor or, most frequently, editors. Important as such hydra-headed contributions are, it is the textbook with one or two authors which offers a conceptual unity and coherence—a clear reflection of the entire body of the then existing knowledge as seen by the authors.

Leo Kanner's text, first appearing in 1935, reflected the beginnings of the pluralistic, multidisciplinary influences in child psychiatry; Gerald Pierson's *Emotional Disorders of Children* (1949) stressed the psychodynamic viewpoint so prevalent in the 1940s; Harry and Ruth Bakwin reflected the clinical emphasis of the 1950s; and in the Stella Chess and Mehin Hassibi text can be seen the psychopharmacology of the 1970s. Each was the product of its time. Now, for the 1980s, the era stamped by DSM-III and DSM III-R, Paul Adams and Ivan Fras have produced a textbook for this time.

They are well qualified to do so. Paul Adams, now occupying an endowed chair as professor of child psychiatry at the University of Texas Medical Branch at Galveston, has long been concerned with teaching the art and science of child psychiatry. He has documented many of his concerns in what I. Berlin has described as "human documents, moving and poetic." He has authored *Children's Rights* (1941), *Obsessive Child* (1949), *Primer of Psychotherapy* (1982), and *Fatherless Children* (1984). Ivan Fras, now clinical professor of psychiatry at the State University of New York Upstate Medial Center in Syracuse and the clinical campus at Binghamton, has had extensive experience in the teaching and practice of child psychiatry.

The Adams-Fras text, however, does not neglect the past. It respects the origins of child psychiatry in medicine and pediatrics, law, educational theories and practice, movements for social reform, social work and child psychiatry, but it hews closely to the nosology of DSM-III and DSM III-R, the first text in child psychiatry to do so. The major section of the book (its Part II) thus describes three major groups of disorders in accordance with DSM-III: the doing or behavior disorders, the feeling or mood disorders, and the thinking and central processing disorders. For each condition described, the authors have provided a definition and description (usually in accordance with DSM-III), etiology, course and prognosis, treatment, and differential diagnosis. Each entity is illustrated with case vignettes (the significant findings in each carefully italicized), questions for "study and action," and references for further reading. The case illustrations are well chosen, the questions for study are thought provoking, and the problems of definition and nosology are discussed in a reasonable, well-modulated tone, with a clarity and economy of writing, all of which make this a very readable opus.

Drs Adams and Fras, however, have gone beyond DSM III-R. There is a complete section in "Disorders of Childhood" (Section II) on the child as a victim, in which neglect and sexual, physical, and emotional abuse are considered. This section includes a chapter on the role of the child psychiatrist in the courtroom and discusses child custody fights, legal infractions by children, involuntary commitment to mental hospitals, competency determinations, consent and assent to treatment and research, termination of parental rights, personal injury to children, privacy, and confidentiality. The authors speak of these topics with the authority of their own courtroom consultation experiences and the intensity of their own feelings in fighting for the rights of children.

Their compassion and understanding are reflected also in Section III of this text, "The Child as a Family Member." Here the family context of the child is considered more systematically than in the discussion of clinical entities in Section II. The many ways in which families respond to stress and crisis, the supports which help the child and the parental dysfunctions which hurt him, are considered. Separate chapters deal with specific problems; divorce and remarriage, adoption and re-adoption, the meaning of separation and hospitalization, and finally "one outline of what a wholesome family can do for children." It is clear that the nature-nurture dichotomy is not a theme of this book but that the interrelationship between nature and nurture is.

Section IV deals with psychiatric emergencies, attempted suicide, accidental poisoning, violence, bereavement, and trauma. And finally there is a concluding chapter on the field of child psychiatry itself, its place within the context of the neurosciences, medicine, education, and the social sciences. An appendix traces the chronology of child psychiatry from the year 5000 BC, when the earliest cities were built in Mesopotamia, to 1970 when Jean Piaget's book, the *Principles of Genetic Epistemology*, was published. The chronology is wide in scope and concept. All that ever happened on this earth must have had some influence on children!

This book is a reflection of the state of the art and science of child psychiatry of today. It is eclectic in that it deals with many approaches to etiology and treatment of children: behavioral, educational, family psychotherapy, psychoanalytic, and psychopharmacological. It is, however, a reasoned eclecticism, a balancing of what would be of help to a child at this particular time in his life, in the context of the world he lives in. As with most textbooks, however, so much has to be covered that most cannot be considered in depth. Drs Adams and Fras must have recognized this, because they elected to title their book *Beginning Child Psychiatry*. As a result some of us may become impatient with clinical presentations, labeling them as superficial, incomplete, sometimes misleading (as on p 331: "a history of brain damage almost ensures the diagnosis of ADHD in the presence of symptomatology in the hyperactivity-attention deficit-impulsivity clusters)." The discussion of psychopharmacology is also brief and superficial. Yet, this book accomplishes what it sets out to do, "to present an updated view of what can be said about the field of child psychiatry as a growing body of knowledge, skills and attitudes concerning the mental disorders of children."

Typographically this is a beautiful book. The hard cover is a modest gray, with the title emblazoned in gold in a field of dark gray. The paper stock is heavy, the type large and clear. Unfortunately in the copy reviewed, scattered pairs of blank pages appeared in the center of the book, obviously pages which were not printed and which interfered with continuity. It is hoped that this error will be corrected in the subsequent printings, which this book deserves.

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ASSURING QUALITY OUTPATIENT CARE FOR CHILDREN. GUIDELINES AND A MANAGEMENT SYSTEM

By Charles D. Cook, MD, and Jane Heidt. 204 pp, illustrated. New York, Oxford University Press, Inc. 1988. $35.00 (hardcover)

To my knowledge this book represents the first systematic approach to quality health assurance for ambulatory pediatric care and the authors should be commended for this. Dr Cook, the senior author, served for many years as chairman of the Department of Pediatrics at the State University of New York Health Science Center at Brooklyn where he had a large data base of patients, primarily at the affiliated Kings County Hospital. It was there that he developed a system for assuring quality outpatient care for children.

This book is a fine pediatric manual intended more for the managers of pediatric outpatient departments (OPDs) than for pediatricians. Pediatric program directors will also want to study it and insure that their respective OPD managers read it so as to become well versed in its guidelines.

One of the book's problems is that its table of contents does not always follow a logical pattern. The book's mission is set forth in the first three introductory chapters. They emphasize guidelines for the common disease problems seen in the pediatric OPD. In these chapters the authors imply that the term *protocol* is synonymous with *guideline* and use the latter throughout the book. In my view *guideline* implies a less rigid connotation than *protocol.*

Chapter 4 deals with the structural components of the guidelines such as standards of care, encounter forms, and patient education. Chapters 5 through 7, which discuss common disease problems encountered in the pediatric OPD, tend to be redundant at times. The remaining chapters appropriately concentrate on the structures and processes needed to implement the system.

The authors deal with outcome evaluation in chapter 12. They utilize correlational patterns and chi-square analysis. A multivariate analysis could have been used by combining several 2 × 2 contingency tables.

There are a number of specialized health professionals for whom this book will prove extremely valuable. These include quality assurance personnel, hospital clinicians, and those concerned with health care financing. The book will be a valuable asset for all hospital based medical libraries, pediatric OPDs, and neighborhood family health care centers.

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LINEAR GROWTH RETARDATION IN LESS DEVELOPED COUNTRIES

By John C. Waterlow, MD. 313 pp, illustrated. New York, Raven Press, Ltd, 1988. $40.00 (hardcover)

This book provides valuable data on the stature of children in various parts of the developing world. Important information by authoritative investigators emerges on the relationships between stature, nutrition, socioeconomic class and mental development. Specific deficiencies of substances such as the metals zinc and calcium, as well as an excellent review of hormonal factors for growth, are valuable additions to the literature. The question/answer sections are in some ways the most interesting as they indicate the thinking and suggest future projects in this vitally important area of international child health. What is to be done? This should perhaps be the subject of a future conference.

In general, the organization of the book is good, although it would have been helpful to index the countries and ethnic groups. The bibliography is excellent. My only criticism is that the current volume would have been even more valuable had it included growth curves from each underdeveloped country for international use. This book will serve pediatricians, politicians, and child advocates well in changing the status of health in children.

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SOLVING COMMON PEDIATRIC PROBLEMS: AN ALGORITHM APPROACH

By Carmi Z. Margolis, MD. 206 pp, illustrated. Jamaica, NY, Solomon Press, 1988. $29.95 (hardcover)

This book was written for practitioners who care for infants and children, and has as its goal the presentation of an approach to clinical decision making. The format championed is the clinical algorithm. The author makes two contentions: that the clinical algorithm is especially suited for decision making, and that the stepwise nature allows the clinician critically to look at the rationale behind each of the steps. The author does not expect each reader to agree with every algorithm, but rather contends it is the inherent superiority of the algorithmic process that is important.

Dr Margolis is well qualified to write a book on this subject. It is a culmination of more than six years of scholarly work. He acknowledges the assistance of a group of dedicated clinical algorithmicians and many well-known academicians who made substantial contributions to the content.

Dr Margolis begins with the evolution and uses of clinical algorithms. The chapter, like the entire book, is well written and provides the reader with an excellent historical perspective and also a working understanding required for their use. The following chapters discuss the process of developing and applying them.

The body of the text involves the presentation of 28 clinical algorithms that cover most common acute pediatric problems. It might have been helpful to add algorithms on such common problems as exanthems, lumps and bumps, acute abdominal pain, and limp. These omissions among others prevent this book from being used as a general text, but this was not the author's intention. The algorithms are clearly presented and meticulously referenced. The method of presentation makes this text useful for the medical student, resident, experienced clinician, and academic teacher.

Dr Margolis anticipated some opposition and has included a rebuttal in the text. I do agree that the algorithm can facilitate and clarify the clinician's approach to problem solving. I also agree that there is no reason that physicians cannot be taught to think "algorithmically," but it is still my impression that algorithms often result in an oversimplistic approach to clinical situations and that if followed rigidly, without a greater understanding, they could lead one astray.

The algorithm on acute asthma, for example, fails adequately to stratify treatment options for severity of illness. While some patients may be stable enough for nebulized treatment or subcutaneous epinephrine every 15 to 30 minutes, other patients need immediate oxygen therapy and use of multiple agents and modalities over a shorter time period. The fever algorithm addresses infants under age two with temperatures greater than or equal to 40° C. This definition excludes the majority of febrile infants. This algorithm, like others, suffers from a lack of branch points. A child who looks well, but has a temperature of 42.5° C and congenital heart disease should be approached differently than another infant with a temperature of 40.1° C and a completely negative past medical history. The algorithm presented on the injured patient fails to mention the importance of the ABCDE approach (assure airway breathing and circulation, protect the C-spine, completely expose the patient).

In summary, if one does not agree with Dr Margolis' contention that the algorithmic approach is the right one, one would still find this book challenging and thought provoking. If one is enamored of the use of clinical algorithms, then this book should be an essential addition to one's library.

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DIAGNOSIS AND MANAGEMENT OF RENAL DISEASE AND HYPERTENSION

By Anil K. Mandal, MD, and J. Charles Jennette, MD. 540 pp, illustrated. Philadelphia, Pa, Lea & Febiger, 1988. $67.50 (hardcover)

Many years of clinical and research work in nephrology have allowed this reviewer to read and consult a host of textbooks in this field. Writing or editing a book is an arduous and demanding challenge of great responsibility. It is spiritually rewarding, although the possibility of failing to contribute significantly to a discipline and slip into oblivion is, regrettably, more frequent than we would like to acknowledge.

The work by Drs Mandal and Jennette is not just one more book on the subject. This single volume constitutes a compact assembly of vast and comprehensive nephrologic knowledge. It has been put together clearly and concisely; its well organized text is easily readable as the subjects flow logically and smoothly. It is fully illustrated and uses abundant and well designed figures, diagrams, and tables. An ample number of current references provide a strong and up-to-date background to the 19 chapters, of which Drs Jennette and Mandal contributed five and four, respectively. The remaining sections were written by another group of distinguished national (Georgia Medical College, VA Medical Center) and international (Australia, India, Saudi Arabia) experts. The book consists of 540 pages of text which, as described by Dr R. Schrier, who wrote the foreword, emphasizes " . . . clinically useful and applicable information . . . for the practising physician and nephrologist and all those who look after patients with renal diseases." In this reviewer's mind there is no doubt that the book will indeed fulfill this commitment. Drs Mandal and Jennette have combined their expertise in nephropathology and clinical nephrology to deliver a high quality, authoritative, yet eminently practical book at the level of the best presently available contributions in nephrology.

Minor criticism can be made of typos and misspellings that presumably slipped through the final manuscript review. It may be argued that the prominence of hypertension in the book's title constitutes a bit of an inconsistency since only one chapter is devoted to this subject. However, the thorough review and updating of physiology, pathophysiology, semiology, and therapeutics of hypertension, the same approach exhibited in the other chapters, makes this section most comprehensive. Throughout this book, Drs Mandal and Jennette and collaborators transmit didactic knowledge cogently and wisely. This reviewer highly recommends the enjoyment of reading this book through and keeping it handy for consultation.

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MEDICAL TOXICOLOGY. DIAGNOSIS AND TREATMENT OF HUMAN POISONING

By Matthew J. Ellenhorn, MD, and Donald G. Barceloux, MD. 1,512 pp, illustrated. New York, Elsevier Publishing Co, Inc, 1988. $175.00 (hardcover)

This book is a compendium of the literature of poisonings. Its purpose is to "present data necessary to evaluate and treat the clinical effects of toxic substances." Basic information on patient assessment and treatment, pharmacokinetics, drug interactions, and toxic exposures during pregnancy is presented in the initial chapters. The main portion of the book consists of monographs on poisoning produced by drugs, chemical products, and animal and plant toxins.

The monographs follow a general format of introduction, pathophysiology, clinical presentation, laboratory, and treatment, which facilitates finding pertinent information. This format works well when only one entity is described, less well when multiple related entities are included. For example, pathophysiology may contain symptoms not given in clinical presentation, making it necessary to check both sections. The text is interspersed with an impressive collection of tables, illustrations, and graphs, mainly reprinted from other publications.

The quality, accuracy, and clarity of the information varies throughout the text. Extensive quoting from the literature influences the monographs both positively and negatively. When the literature is complete and timely, the resulting monograph is usually good. If the literature is voluminous but outdated, it may be cited at the expense of more timely information with less clinical data. However, adherence to published reports is not always followed. When case reports are rare, symptoms presumed from the pharmacology of the drug may be included (eg, "nausea, vomiting, . . . and abdominal pain resulting from closure of the sphincter of Oddi in patients with pancreatobiliary disorders" are listed as symptoms of pentazocine overdose, yet there were no gastrointestinal symptoms in the two cases reported to date). While citing an article, other references and author's comments are occasionally inserted, resulting in distortions of the information.

In a complicated book of this size, some mistakes are to be expected, but this book has an inordinate amount of errors. Examples: "activated charcoal alone may be the preferred emetic"; "body lice, which burrow into human skin"; "amantadine is a drug with cholinergic properties." Rock cocaine is described as "tiny crystalline pieces of freebase"; however, it is not crystalline and some pieces are quite large. Referring to "loads," the authors state: "this combination street drug contains codeine and glutethimide"; however, a "load" is a variable number of both glutethimide tablets and acetaminophen with codeine, or aspirin with codeine, tablets. Some errors are of omission. There is no mention of hyperinsulinism in response to glucose infusion in chlorpropamide overdose. In treating allergic reactions from snake antivenin, corticosteroids are not mentioned.

Many statements are unclear or strange. Under heroin overdose: "normal appearing blood pressure may mask prior hypertension." For oral hypoglycemic overdose: "glucagon administration must follow the use of glucose." "Insomnia may occur after sudden withdrawal of short-acting benzodiazepines perhaps because of a lag in replacement of endogenous benzodiazepines." "The vast majority of PCP first time users do not like [the effects] and do not seek further encounters" followed by "80% of [PCP] users have pleasurable experiences."

Some treatment recommendations are not standard practice. Examples: immersion in ice for hyperthermia; lavage with patient in left/lateral head down position; IV diazepam as treatment of choice for PCP intoxication; propranolol as antidote for cocaine; chelation therapy for acute arsenic exposure only when the patient is symptomatic.

This book has more information than other similar publications in the field and has better information on the composition of natural toxins, chemical properties of products, and plant intoxication. However, its spotty character with regard to scholarship prevents it from being a definitive primary source of information on current diagnosis and treatment. Nevertheless, there is enough good information in this book to warrant adding it to a library that contains other available toxicology texts.

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COMMON SKIN DISORDERS: A PHYSICIAN'S ILLUSTRATED MANUAL WITH PATIENT INSTRUCTION SHEETS

Third Edition. By Ernst Epstein, MD. 110 pp, illustrated. Oradell, NJ, Medical Economics Books, 1988. $35.95 (paperback)

Dr Epstein states in his preface that *Common Skin Disorders* is directed at physicians who do not specialize in dermatology. The book is divided into three sections. The first and longest is described as a "mini-text" on the most common skin disorders and methods of treatment. Certainly the 40 or so topics in this section would account for 98% of the skin conditions seen by a nondermatologist. Each topic is generally divided into sections on etiology, diagnosis, complications, and treatment.

In many areas this book admirably accomplishes its stated purpose, particularly in the management of common skin conditions not requiring special diagnostic ability or surgical management. For instance, there are excellent chapters on rational use of topical corticosteriods, diagnosing dermatitis, treatment of dermatitis, hand dermatitis, and others. For many skin conditions, diagnosis by a nondermatologist can be difficult. Yet the book places more of an emphasis on treatment rather than diagnosis. For example, in the section on actinic keratoses, the author says that "diagnosis is usually straightforward—the scaly, often red, irregularly surfaced, sharply dermarcated roughenings on the sun-exposed areas of fair skinned persons are distinctive." Following this are three pages on treatment modalities including surgical removal, topical 5 FU, and liquid nitrogen cryotherapy. Even if the diagnosis of actinic keratoses were really "straightforward," by giving detailed instructions Epstein seems to advocate that nondermatologists manage these lesions.

The topic of pigmented nevi is presented in a similar fashion. A detailed description of what should make a physician suspicious of a pigmented lesion might be more worthwhile than the statement, "If you strongly suspect malignancy, full-thickness excision should be performed as initial therapy . . . ."

The second section consists of 16 pages of color photographs. These are also devoted to treatment rather than diagnosis, including many pre- and post-treatment photos.

The third section includes patient instruction sheets meant to be photocopied and handed out to patients. According to the author these sheets should serve five basic purposes: to provide general information before the patient is seen, to inform the patient after the examination; to deal with the "by-the-way" questions; to inform about routine post-op care; and to outline standard but detailed treatments. In general, these sheets serve these purposes well.

I recommend this book to both dermatologists and nondermatologists for the section on patient instruction sheets. I would also recommend this book to nondermatologists for the chapters on management of common skin disorders. However, I would issue the caveat to be aware of the importance of undertaking cases only in those areas in which the physician has been thoroughly trained.

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WHEN LIVING HURTS. DIRECTIVES FOR TREATING DEPRESSION

By Michael D. Yapko, PhD. 250 pp. New York, Brunner/Mazel Publishers, 1988. $25.00 (hardcover)

This book attempts to present various concepts that the author thinks would be of assistance in the treatment of depression. Dr Yapko, who is in private practice in California, has written several books and papers on Ericksonian psychotherapy and on hypnosis. He has also written several papers on depression. In addition to his private practice, he is on the teaching staff of several universities in the San Diego area.

The focus of the book, as stated by Dr Yapko, is on responding to the experience of depression that is already present in the individual. He attempts to deal with this issue by presenting "preventive strategies" for dealing with depression. The book is organized according to a series of "directives" which are grouped according to associated issues. These directives are methods for actively directing the client's experience. Dr Yapko states that he presents the book in this "somewhat unusual way," in order "to illustrate ways to promote experiential learning." The author further states that an even greater goal of this book "is to catalyze the clinician's creativity in generating novel and active ways of facilitating recovery for their depressed clients."

This book is useful in that it presents a different approach to the treatment of depressive illness. As the author states, "many of the strategies in this book are simply vehicles for stimulating clearer thinking and positive action." The author tries to establish various ways to help engage the "client" in a therapeutic milieu. He attempts to use active strategies in order to achieve this goal.

The book is divided into two sections, one on theoretical framework and the other on treatment strategies. Its 12 chapters are put together in an organized fashion. A bibliography and two appendices are included. The author makes an attempt to present his data, sometimes successfully, in a clear and concise manner. Many of the premises presented in the book are not supported by a great deal of research material. The bibliography given is not an extensive one, and the appendix that gives the case narratives only includes four cases.

Dr Yapko tries to present a somewhat different approach to the treatment of depression. Those practitioners who are more interested in a psychotherapeutic or pharmacologic approach may find this book less satisfying. With depressive illness being such a prevalent and difficult to treat illness, it is always helpful to look at a potentially newer treatment approach.

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SOCIAL LEARNING AND SYSTEMS APPROACHES TO MARRIAGE AND THE FAMILY

Edited by Ray DeV. Peters, PhD, and Robert J. McMahon, PhD. 340 pp. New York, Brunner/Mazel Publishers, 1988. $37.50 (hardcover)

Peters and McMahon are two social scientists who were inspired by the Banff Conferences of 1985 concerning marriage and the family to write a book on this subject. They have set out to integrate what has been learned from a family systems approach derived from clinical psychiatry with social learning theories derived from experimental psychology. This book consists of 12 chapters, the first half dealing with psychological processes as they apply to marriage and families and the second half dealing with treatment approaches.

The first half gives much background on topics ranging from parenting problems such as maternal depression to family correlates of spouse abuse, peer relations, and patterns observed in divorce. These chapters are highly readable and well organized. They offer much relevant information on some very complex issues without being facile and without overwhelming the reader. For example, Ross Parke et al, in a chapter entitled "The Role of the Family in the Development of Peer Relations," give fascinating examples of how interactions within the family such as physical play, directiveness, and verbal reasoning may be transmitted and later expressed in peer interactions outside of the family; eg, a physically playful father is seen to produce both sons and daughters with more desirable social attributes.

The second half of the book deals with treatment of such marriage and family problems as male sexual dysfunction, child sexual abuse, marriage conflict, and training programs for families with handicapped children. This half is more uneven than the first half. In an excellent chapter by Pharand et al, entitled "Comprehensive Assessment of Family Functioning," rating scales and checklists, many of which were filled out by family members, were demonstrated as useful tools for identifying areas of conflict, cohesion, and adaptability in families and could also be used to measure changes after psychotherapy. Families were observed to function well no matter what their style provided that individual members were all satisfied with the family style. In this same chapter a plea is eloquently made for therapists and "measurers" to work together to the advantage of all. The complaint is registered that research articles are often written in such a way that they seem to have little clinical relevance and measurement instruments are often not user friendly. It was ironic that this chapter followed immediately after a chapter in which the reader might well have felt inundated by confusing and questionably relevant statistical findings. But that chapter was clearly the exception rather than the rule and the book generally follows in the worthwhile tradition of Dollard and Miller, whose book *Personality and Psychotherapy, an Analysis in Terms of Learning, Thinking and Culture*, published in 1950 and dedicated to "Freud, Pavlov and Their Students," also attempted to integrate these disparate approaches. This need seems certainly as great now as it did then because of the plethora of data being generated by behavior therapy.

Although journal articles proliferate, there is surprisingly little material available in book form that addresses the continuing need to put together what is coming out of laboratories with respect to behavior modification techniques with what is coming out of clinicians' offices regarding marital and family conflicts. This book clearly does not take sides but rather strongly suggests that there is more than enough room for both Freud and Pavlov. It has set out to and succeeded in espousing a "bidirectional" approach and as such merits a place in the library of both therapist and academician dealing with problems relating to marriage and the family.

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HEALTH SERVICES ADMINISTRATION: EDUCATION AND PRACTICE

By Karen M. Lorentzen, RN, EdD, and Linda Roemer, PhD. 118 pp. St Louis, Mo, Warren H. Green, Inc, 1988. $19.50 (softcover)

Considering the current and constant furor and upheavals taking place in the US health care delivery system, this slim, 118-page book presents a rather placid view of the situation. Although the book was published in 1988, none of the 54 items listed in the bibliography is dated later than 1983. Obviously, that gap leaves much of the literature dealing with the impact of the cost containment initiative via the prospective payment system (PPS) and diagnosis related groups (DRGs) out of the discussion.

Since October 1983 when the PPS/DRG reimbursement scheme was initiated, the US health care industry has undergone numerous evolutions directly relating to health services administration. Professional literature has been redundant with articles detailing combinations and permutations, seeking to arrive at the proper organizational structure to survive in the cost containment and competitive health care marketplace. That vibrant tempo of managerial maneuvering is not evident in this book. Heated debates about the "medical-industrial-complex" and its emphasis upon the role of profit-making or nonprofit organizations providing health care services are hardly in evidence. Those aspects certainly do not lead to an accurate portrayal of where we are today in the US health care delivery system.

Starting with a brief history of the development of health care administration, the authors proceed to the two chapters dealing with management. Among the issues and trends noted in chapter 2 are cost containment and access, regulation and competition, and organizational changes. Chapter 3 talks about planning; managing, using analysis, communications, facilitation, and leadership; decision making; and closes with evaluation. While those administrative elements are relevant and appropriate, the missing tempo and atmosphere of the times diminish their importance in discussing today's health care administration.

Most of the book concentrates on the educational process of preparing individuals to become health service administrators. Four out of the seven chapters cover educational programs, field placements, health care administration as a profession, and model syllabi. In addition, professional and health related organizations and related journals are listed in an appendix section.

Since both authors are college level educators, this book does stress the education of administrators rather than the practice of health services administration. In fact, the authors clearly state their intent to do so in the introduction to the book. With that point of view, this book will not be of compelling interest to the majority of practicing physicians.

However, what is more disturbing about this 1988 book is that, essentially, it is "too little and too late." How can we discuss the education of heath care administrators without integrating the movements into alternative care situations? Or the wrenching intrusion of a corporate profit-making culture into the pristine, humane ethics of delivering health care services? As Charles Dickens commented in *A Tale of Two Cities*: "This is the best of times and the worst of times."

High technology medicine in the United States achieves miracles but still leaves people longing for a return to the family physician who was a friend as well as a healer—the physician who cured as well as who cared.

Vital statistics reveal that there is only one communicable disease (influenza/pneumonia) among the top ten killers in America. Are our health care administrators being trained to manage in an environment where individuals suffer from ailments directly linked to life style deficits?

Lastly, and most seriously, this book simply is not up to date. Joint ventures, managed health care plans, holding companies, wellness centers, home health care, organizational restructuring, and the inevitable profit centers approach are only a few of the missing health services administration links. Therefore, this book is not recommended for the practicing physician or for health care administrators. There are suitable alternative publications that do the task more effectively.

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Cigarette smoking and passive smoking linked to cervical cancer

Women who smoke cigarettes and women who are exposed to others' cigarette smoke are at increased risk of developing cervical cancer, according to a study by Slattery et al (*JAMA* 1989; 261:1593–1598). These findings support earlier hypotheses of an association between cigarette smoking and cervical cancer.

Such hypotheses were based on the link between cigarette smoking and squamous cell carcinomas, including those of the lung and larynx, and on the fact that squamous cell carcinoma is a common form of cervical cancer. Although the mechanism by which cigarette smoking affects the development of cervical cancer has not yet been elucidated, studies have demonstrated that substances in cigarette smoke can travel through the blood stream to tissues throughout the body. In fact, such substances, which may constitute the mutagenic and carcinogenic components of tobacco smoke, have been identified in the uterine cervix of cigarette smokers. Furthermore, some of these mutagenic substances exist in greater concentrations in side-stream smoke—the smoke present in areas where other people are smoking—than in mainstream smoke—the smoke inhaled by smokers.

Fifty-six percent of the subjects with cervical cancer were smokers, whereas 75% of the control population had never smoked cigarettes. Among the study population, 87% had carcinoma in situ and 13% had invasive carcinoma. Other characteristics of the study population included lower educational and income levels and greater numbers of sexual partners than control subjects.

The risk of cervical cancer is greatest among women smokers less than 30 years of age and among smokers who have had one or fewer sexual partners. The authors intimate that "this is probably because cigarette smoking will be a greater risk factor in women who do not have other major competing risk factors."

Women exposed to the cigarette smoke of others are also at increased risk of developing cervical cancer. This study demonstrated that "the risk associated with passive smoking . . . is as strong as that observed from personal cigarette smoking." Furthermore, the risk associated with passive smoking was greater in women who did not smoke than among women who smoked. Women exposed to passive smoke in their homes were at greater risk than women exposed to others' smoke outside the home. In addition, personal cigarette smoking in combination with exposure to passive smoke yields an even greater risk for cervical cancer than does each risk factor considered independently.

One strength of this study, as emphasized by the authors, was that subjects' reports of smoking status were verified with tests of serum cotinine levels—a validation procedure not commonly employed in epidemiologic studies. Future studies to identify specific mutagens in the cervical fluid of women exposed to passive smoke would strengthen the association between passive smoking and cervical cancer and would help define the mechanism by which cigarette smoking increases the risk of cervical cancer.

Menopause is associated with an increased risk of atherosclerosis

Menopause has been related to an increased incidence of cardiovascular disease, although some studies have disputed such findings. Witteman et al sought to assess the association between menopause and atherosclerosis by determining the presence of calcified deposits in the abdominal aorta as viewed on lateral radiographs of the lumbar spine (*Br Med J* 1989; 298:642–644).

In this study, menopause constituted the absence of menstruation for at least one year. When linear densities were apparent in an area parallel and anterior to the lumbar spine, calcification was determined to be present.

The onset of menopause, smoking, and serum total cholesterol concentration were significantly associated with increased risk of aortic calcification. "Women with a natural menopause had a 3.4 times greater risk of aortic calcification than premenopausal women . . . , and women who had had a bilateral oophorectomy had a 5.5 times greater risk . . . ." The authors conclude that, "Among women with a natural menopause the risk of aortic calcification adjusted for age and other indicators of cardiovascular risk showed a highly significant trend ($p < 0.001$) with increasing years after the menopause."

The authors caution that although aortic calcification is associated with cardiovascular disease, this study examined calcification in the abdominal aorta, and the results may not extend to the coronary arteries. Bodily changes associated with menopause may affect various arteries differently.

The reduced estrogen levels that result following menopause may directly cause the increased risk of atherosclerosis. Estrogens may have a direct effect on vessel walls. These hypotheses are yet to be proven or disproven. Future studies should also address the effect of postmenopausal estrogen therapy on atherosclerosis.

Illness among commercial air travelers is more common on the ground

In a one-year prospective assessment of the frequency and types of medical emergencies among commercial air travelers, Cummins and Schubach demonstrated that only 25% of the 754 travelers surveyed had a medical problem during flight, while 75% of emergencies occurred when travelers were on the ground in the airport complex (*JAMA* 1989; 261:1295–1299).

The most common medical problems fell into a category labeled "other medical" and were characterized by complaints such as fatigue, sleeplessness, "not feeling right," and presumed alcohol intoxication. During flight, gastrointestinal symptoms predominated, including nausea, vomiting, diarrhea, and abdominal pain. Other common in-flight medical problems involved respiratory com-

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NEWS BRIEFS are compiled and written by Vicki Glaser.

plaints including shortness of breath, hyperventilation, and asthmatic attacks, cardiac disorders that produced symptoms such as chest pain and angina, syncope, seizures, and allergic reactions. Most problems related to diabetes occurred in the airport and were attributable to forgotten or misplaced insulin and hypoglycemic incidents.

In this study, personnel trained as emergency medical technicians effectively treated the majority of travelers who experienced medical emergencies—primarily those emergencies that occurred on the ground. With regard to in-flight emergencies, the authors recommend that flight attendants receive improved training in the handling of medical emergencies, training similar to the basic level acquired by emergency medical technicians.

During an in-flight medical emergency, flight attendants will frequently seek the assistance of any physicians on board. "Of particular importance to the traveling physician is increased knowledge of field assessment of abdominal pain, shortness of breath, chest pain, and syncope and presyncope." As required by the Federal Aviation Administration (FAA), all US commercial aircraft must carry a "doctors only" medical kit, which contains such items as a stethoscope, a sphygmomanometer, three sizes of oropharyngeal airways, sublingual nitroglycerin, and needles and syringes to deliver epinephrine, diphenhydramine, and 50% dextrose. The authors recommend the addition of several medications to this kit: an injectable antiseizure medication, an inhaled bronchodilator, an injectable narcotic antagonist, a parenteral diuretic, and a parenteral antiemetic. The findings of this survey suggest that the "doctors only" medical kit is useful and should continue to be a required component of commercial aircraft.

AIDS educational material may increase rather than reduce fear of AIDS victims

"Workers who received educational brochures or pamphlets about the disease AIDS are more likely to have negative feelings toward AIDS-stricken co-workers than employees who received no information," according to a survey conducted by the Georgia Institute of Technology's Center on Work Performance Problems (Atlanta). This finding sends an important message to the developers of public education campaigns that focus on AIDS: the tone and focus of the message you deliver are crucial. It is not sufficient to merely warn of the danger of AIDS and to focus on the practices that can increase risk of transmitting the disease. The social, emotional, and humanitarian aspects of the AIDS epidemic should be addressed in public education efforts. Only then will such efforts begin to affect people's attitudes towards the disease and its victims, in addition to encouraging the cessation of risky behaviors.

Employees who attended lectures or viewed videotapes on AIDS demonstrated little change in attitude. "However, employees who participated in comprehensive training sessions became significantly less fearful." The director of the survey stressed the need for more comprehensive educational programs focusing on AIDS. "We not only have to be able to deal with what happens when a person with AIDS is in the work force, but also the potential disruption related to the millions of other people who associate with people who have AIDS."

Factors affecting the health care of physicians' children

It would seem obvious that physicians would be virtually guaranteed the highest quality health care for their families. Their knowledge of health matters, advanced education, relationships with colleagues in the medical profession, familiarity with the health care system, and financial status should all serve as advantages in obtaining quality health care. However, the results of a study by Wasserman et al suggest that there are specific differences in the health care received by physicians' children as compared to other children (*Pediatrics* 1989; 83:319–322). The authors found that "although this differential care is in some ways better . . . , in most ways it appears to be worse."

Interviews of 31 pediatricians in a specified geographic region provided the data on physician behavior in the care of physicians' children and other children. The authors detailed six areas in which care of physicians' children differed from the care provided to other children. First, although physician parents tend to have easier access to pediatricians, they were less likely to call for help—perhaps because their increased knowledge of medicine makes physician parents feel that they should be able to care for their children's medical needs. Similarly, physician parents waited an "inappropriately long" time to bring their children to the pediatrician for treatment of an acute illness.

The authors noted differences in communication between pediatricians, their patients, and the parents. Pediatricians were less likely to give detailed instructions to physician parents, and were less likely to mention behavior problems or matters concerning the parent-child relationship. Furthermore, pediatricians recorded less information on the patient's medical, social, and psychologic history in the charts of children with physician parents. The pediatricians' expectations of physician parents' knowledge, the awkwardness associated with assigned roles and dominance, and the embarrassment of discussing personal concerns with a colleague could all contribute to the differences observed in parent-pediatrician communication.

Other findings include the increased likelihood of physician parents to self-refer to a specialist without consulting the pediatrician. Pediatricians were more involved in the day-to-day care of physicians' children in the hospital, visiting these patients more frequently.

The authors propose that, "Simple awareness of the potential for problems can itself help pediatricians and physican parents by alerting them to possible pitfalls." Clarification of roles and emphasis on the need for communication are important facets of any physician-patient relationship.

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Pierre Alexis, MD, New Rochelle. Died July 17, 1988; age 49. Dr Alexis was a 1967 graduate of the Universitaire Katholique de Louvain, Faculte de Medecine, Louvain, Belgium. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Raymond Jerome Boller, MD, New York. Died February 22, 1989; age 73. Dr Boller was a 1942 graduate of the New York University School of Medicine. He was a Diplomate of the American Board of Internal Medicine and a Fellow of the American College of Physicians. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Murray Almon Bratt, MD, Rochester. Died February 2, 1989; age 83. Dr Bratt was a 1932 graduate of the State University of New York at Buffalo School of Medicine. He was a member of the New York Academy of Medicine, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Daniel Gerard Butera, MD, Bayside. Died February 20, 1989; age 80. Dr Butera was a 1933 graduate of the State University of New York Health Science Center at Brooklyn. He was a Diplomate of the American Board of Radiology and a Fellow of the American College of Radiology. His memberships included the Radiological Society of New York State, the Radiological Society of North America, Inc, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Umbert Cimildoro, MD, Oswego. Died January 3, 1989; age 88. Dr Cimildoro was a 1930 graduate of the Facolta di Medicina e Chirurgia dell'Universita di Roma, Rome, Italy. He was a Fellow of the American Academy of Family Physicians. His memberships included the New York Academy of Medicine, the Medical Society of the County of Oswego, and the Medical Society of the State of New York.

Leoni Neumann Claman, MD, New York. Died February 5, 1989; age 88. Dr Claman was a 1924 graduate of Columbia University College of Physicians and Surgeons. Her memberships included the New York Academy of Medicine, the American Academy of Allergy, the American Medical Women's Association, the New York County Medical Society, and the Medical Society of the State of New York.

Anni Gerda Cohn, MD, Poughkeepsie. Died February 15, 1989; age 78. Dr Cohn was a 1938 graduate of the Friedrich-Wilhelms Universitaet Medizinische Fakultaet, Berlin, Germany. Her memberships included the American Psychiatric Association, the American Geriatrics Society, the Dutchess County Medical Society, and the Medical Society of the State of New York.

Joseph Jordan Eller, MD, West Palm Beach, Florida. Died February 13, 1989; age 93. Dr Eller was a 1919 graduate of the University of Rochester School of Medicine-Dentistry. He was a Diplomate of the American Board of Dermatology, Inc. His memberships included the New York Academy of Medicine, the New York State Dermatological Society, the Society for Investigative Dermatology, the Pan American Medical Association, the American Academy of Dermatology, the American Dermatological Association, the American Society of Plastic and Reconstructive Surgery, the New York County Medical Society, and the Medical Society of the State of New York.

Henry Charles Engster, MD, Troy. Died January 29, 1989; age 77. Dr Engster was a 1937 graduate of the Albany Medical College of Union University. He was a Diplomate of the American Board of Urology and a Fellow of the American College of Surgeons. He was a member of the American Urological Association, the Medical Society of the County of Rensselaer, and the Medical Society of the State of New York.

Edwin Wilder Gates, MD, Lewiston. Died January 14, 1989; age 89. Dr Gates was a 1926 graduate of Harvard Medical School, Boston, Massachusetts. He was a Fellow of the American College of Physicians. His memberships included the New York Academy of Medicine, the New York State Society of Internal Medicine, the American Society of Internal Medicine, the Medical Society of the County of Niagara, and the Medical Society of the State of New York.

George Clifford Hamilton, MD, Fort Myers, Florida. Died February 2, 1989; age 88. Dr Hamilton was a 1924 graduate of the Queens University Faculty of Medicine, Kingston, Ontario, Canada. He was a member of the Broome County Medical Society and the Medical Society of the State of New York.

Francis Xavier Ipolyi, MD, Houston, Texas. Died January 30, 1989; age 85. Dr Ipolyi was a 1928 graduate of the Orvosi Fakultas Tudomanyegyetem, Budapest, Hungary. He was a Diplomate of the American Board of Otolaryngology. His memberships included the American Academy of Otolaryngology, the Pan American Medical Association, the Medical Society of the County of Franklin, and the Medical Society of the State of New York.

S. Edward King, MD, New York. Died June 4, 1988; age 88. Dr King was a 1923 graduate of Columbia University College of Physicians and Surgeons. He was a Diplomate of the American Board of Internal Medicine and a Fellow of the American College of Physicians. His memberships included the New York Academy of Medicine, the American Public Health Association, the American Society of Nephrology, the New York County Medical Society, and the Medical Society of the State of New York.

Elmer A. Kleefield, MD, Westport, Connecticut. Died November 6, 1988; age 82. Dr Kleefield was a 1930 graduate of the State University of New York Health Science Center at Brooklyn. He was a Diplomate of the American Board of Internal Medicine and a Fellow of the American College of Physicians. He was a member of the Medical

Society of the County of Queens and the Medical Society of the State of New York.

Harold M. Langs, MD, Island Park. Died January 29, 1989; age 56. Dr Langs was a 1958 graduate of the New York University School of Medicine. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Stanley Mackta, MD, Lido Beach. Died December 9, 1988; age 63. Dr Mackta was a 1958 graduate of the Faculte de Medecine de l'Universite de Lausanne, Lausanne, Switzerland. His memberships included the American Geriatrics Society, the American Academy of Physical Medicine and Rehabilitation, the New York County Medical Society, and the Medical Society of the State of New York.

Norma Lontok Maligaya, MD, Bronx. Died December 30, 1988; age 51. Dr Maligaya was a 1960 graduate of the Faculty of Medicine and Surgery of Santo Tomas, Manila, Philippines. She was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Cecil Mantell, MD, Staten Island. Died February 20, 1989; age 75. Dr Mantell was a 1940 graduate of the McGill University Faculty of Medicine, Montreal, Quebec, Canada. He was a Diplomate of the American Board of Internal Medicine and a Fellow of the American College of Gastroenterology. His memberships included the American Thoracic Society, the American Gastro-enterological Association, the Richmond County Medical Society, and the Medical Society of the State of New York.

Bernard R. Margolius, MD, Catskill. Died January 1, 1989; age 79. Dr Margolius was a 1936 graduate of the State University of New York Health Science Center at Brooklyn. He was a Diplomate of the American Board of Anesthesiology and a Fellow of the American College of Anesthesiologists. His memberships included the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the Medical Society of the County of Greene, and the Medical Society of the State of New York.

Joseph E. Milgram, MD, New York. Died February 19, 1989; age 89. Dr Milgram was a 1924 graduate of Columbia University College of Physicians and Surgeons. He was a Diplomate of the American Board of Orthopedic Surgery, a Fellow of the American College of Surgeons, and a Qualified Fellow of the International College of Surgeons. His memberships included the New York Academy of Medicine, the American Orthopaedic Association, the American Academy of Orthopaedic Surgeons, the Society for Experimental Biology and Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Marino Nataloni, MD, Riverhead. Died February 3, 1989; age 63. Dr Nataloni was a 1957 graduate of the Facolta di Medicina e Chirurgia dell'Universita di Bologna, Bologna, Italy. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

Max Nauman, MD, Forest Hills. Died February 2, 1989; age 96. Dr Nauman was a 1920 graduate of the Medizinische Fakultaet der Julius Maximiliams Universitaet, Wurzburg, Bayern, Germany. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Francis X. Reynolds, MD, Bronxville. Died February 18, 1989; age 67. Dr Reynolds was a 1946 graduate of the New York University School of Medicine. He was a Diplomate of the American Board of Surgery, a Fellow of the American College of Angiology, and a Fellow of the American College of Surgeons. He was a member of the Pan American Medical Association, the Bronx County Medical Society, and the Medical Society of the State of New York.

Harold Carl Rosenthal, MD, Poughkeepsie. Died January 26, 1989; age 86. Dr Rosenthal was a 1929 graduate of the Cornell University Medical College. He was a member of the Dutchess County Medical Society and the Medical Society of the State of New York.

Walter Schaefer, MD, Corning. Died January 23, 1989; age 91. Dr Schaefer was a 1923 graduate of the Schlesische-Friedrich-Wilhelms Universitaet Medizinische Fakultaet, Breslau, Germany. He was a member of the Steuben County Medical Society and the Medical Society of the State of New York.

Samuel Jay Stabins, MD, Rochester. Died January 27, 1989; age 88. Dr Stabins was a 1925 graduate of the Emory University School of Medicine, Atlanta, Georgia. He was a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons. His memberships included the New York Academy of Medicine, the American Surgical Association, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

David Steinberg, MD, Coconut Creek, Florida. Died January 29, 1989; age 85. Dr Steinberg was a 1931 graduate of the New York Medical College. He was a member of the New York Academy of Medicine, the Bronx County Medical Society, and the Medical Society of the State of New York.

David Joshua Surrey, MD, Smithtown. Died February 5, 1989; age 82. Dr Surrey was a 1935 graduate of the Medizinische Fakultaet der Universitaet Bern, Switzerland. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

James J. Toomey, MD, Millbrook. Died February 6, 1989; age 94. Dr Toomey was a 1921 graduate of Fordham University School of Medicine. He was a Diplomate of the American Board of Urology, a fellow of the American College of Surgeons, and a Qualified Fellow of the International College of Surgeons. His memberships included the New York Academy of Medicine, the American Urological Association, the Dutchess County Medical Society, and the Medical Society of the State of New York.

Russell H. Wixson, MD, Clearwater, Florida. Died January 21, 1989; age 79. Dr Wixson was a 1934 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a Fellow of the American Academy of Family Physicians. His memberships included the New York Academy of Medicine, the American Occupational Medical Association, the American Geriatrics Society, the Medical Society of the County of Niagara, and the Medical Society of the State of New York.

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1988; 88:456-462). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith[2] reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al.* Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83:925-927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics*, ed 12. Philadelphia, WB Saunders Co, 1983, pp 337-338.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

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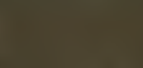

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Warnings: Abnormal elevation of serum potassium levels (greater than or equal to 5.5 mEq/liter) can occur with all potassium-conserving diuretic combinations, including 'Dyazide'. Hyperkalemia is more likely to occur in patients with renal impairment, diabetes (even without evidence of renal impairment), elderly or severely ill patients. Since uncorrected hyperkalemia may be fatal, serum potassium levels must be monitored at frequent intervals especially in patients first receiving 'Dyazide', when dosages are changed or with any illness that may influence renal function.

If hyperkalemia is suspected, obtain an ECG and monitor serum potassium. If hyperkalemia develops, discontinue 'Dyazide' and initiate thiazide therapy if needed. Persistent hyperkalemia may require dialysis. Monitor serum electrolytes frequently in patients with mild renal dysfunction and in diabetic patients. In patients who may develop respiratory or metabolic acidosis, monitor serum electrolytes and acid/base balance frequently.

Precautions: The bioavailability of the hydrochlorothiazide and triamterene components of 'Dyazide' is about 50% of the maximum obtainable with oral therapy. Theoretically, a patient transferred from therapy with hydrochlorothiazide with or without triamterene might show an increase in blood pressure, fluid retention, or change in serum potassium. Extensive clinical experience with 'Dyazide', however, suggests that these conditions have not been commonly observed in clinical practice. (See CLINICAL PHARMACOLOGY.) Use thiazides cautiously in patients with impaired hepatic function. They can precipitate coma in patients with severe liver disease; potassium depletion induced by the thiazide may be important in this connection; administer 'Dyazide' cautiously and be alert for such early signs of impending coma as confusion, drowsiness and tremor; if mental confusion increases, discontinue 'Dyazide' for a few days; attention must be given to other factors that may precipitate hepatic coma, such as blood in the gastrointestinal tract or preexisting potassium depletion. If patients develop hypokalemia, which is uncommon with 'Dyazide', increase potassium intake (i.e., with supplements or potassium-rich foods). If repeat determinations show serum potassium concentrations below 3.0 mEq/L, discontinue 'Dyazide' and initiate potassium chloride supplementation. Institute corrective measures cautiously and monitor serum potassium concentrations frequently, especially in patients receiving digitalis or those with a history of cardiac arrhythmias. Diuretics may aggravate existing electrolyte imbalances, especially at high dosages or in patients on salt-restricted diets. Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Chloride replacement may be required in the treatment of metabolic acidosis. If dilutional hyponatremia develops, restrict water intake. In actual salt depletion, initiate sodium chloride replacement. Use 'Dyazide' cautiously in patients with a history of renal stone formation.

If hyperkalemia develops when treating for hypokalemia, take corrective measures. Also discontinue 'Dyazide' and, if appropriate, substitute a thiazide diuretic until potassium levels return to normal. Do periodic BUN and serum creatinine determinations, especially in the elderly and in patients with suspected or confirmed renal insufficiency. Serum PBI levels may decrease without signs of thyroid disturbance. Discontinue thiazides before conducting parathyroid function tests.

Angiotensin-converting enzyme (ACE) inhibitors can elevate serum potassium; use with caution with 'Dyazide'. Concurrent use with chlorpropamide may increase the risk of severe hyponatremia. A few occurrences of acute renal failure have been reported in patients on 'Dyazide' when treated with indomethacin. Therefore, caution is advised in administering nonsteroidal anti-inflammatory agents with 'Dyazide'. Diuretics reduce renal clearance of lithium and increase the risk of lithium toxicity. Clinically insignificant reductions in arterial responsiveness to norepinephrine have been reported. Thiazides have also been shown to increase the paralyzing effect of nondepolarizing muscle relaxants such as tubocurarine; therefore use cautiously in patients undergoing surgery. Monitor electrolytes in patients taking amphotericin B, corticosteroids or corticotropin concomitantly. Thiazides may potentiate the action of other antihypertensive drugs. The effects of oral anticoagulants may be decreased when used concurrently with hydrochlorothiazide; dosage adjustments may be needed. 'Dyazide' may raise the level of blood uric acid; dosage adjustments of antigout medication may be needed to control hyperuricemia and gout. The following agents given with triamterene may promote serum potassium accumulation and possibly result in hyperkalemia, especially in patients with renal insufficiency: blood from blood bank (may contain up to 30 mEq of potassium per liter of plasma or up to 65 mEq of potassium per liter of whole blood when stored for more than 10 days); low-salt milk (may contain up to 60 mEq of potassium per liter); potassium-containing medications (such as parenteral penicillin G potassium), and salt substitutes (most contain substantial amounts of potassium). Exchange resins, such as sodium polystyrene sulfonate, whether administered orally or rectally, reduce serum potassium concentrations by sodium replacement of the potassium; fluid retention may occur in some patients because of the increased sodium intake. Chronic or overuse of laxatives may reduce serum potassium concentrations by promoting excessive potassium loss from the intestinal tract; laxatives may interfere with the potassium-retaining effects of triamterene. The effectiveness of methenamine may be decreased when used concurrently with hydrochlorothiazide because of alkalinization of the urine. 'Dyazide' will interfere with the fluorescent measurement of quinidine.

There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy only if clearly needed. Thiazides and triamterene cross the placental barrier and appear in cord blood. The use of thiazides in pregnancy requires weighing the anticipated benefit against possible hazards, including fetal or neonatal jaundice, pancreatitis, thrombocytopenia, and possibly other adverse reactions which have occurred in the adult. Thiazides appear, and triamterene may appear, in breast milk. If use of the drug is essential, the patient should stop nursing. Safety and effectiveness in children have not been established.

Adverse Reactions: The serious adverse effects associated with 'Dyazide' have commonly occurred in less than 0.1% of patients treated with this product. Anaphylaxis, rash, urticaria, photosensitivity, cardiac arrhythmias, postural hypotension, diabetes mellitus, hyperkalemia, hyperglycemia, glycosuria, hyperuricemia, hypokalemia, hyponatremia, acidosis, hypochloremia, jaundice and/or liver enzyme abnormalities, pancreatitis, nausea and vomiting, diarrhea, constipation, abdominal pain, acute renal failure, interstitial nephritis, renal stones composed primarily of triamterene, elevated BUN and serum creatinine, abnormal urinary sediment, leukopenia, thrombocytopenia and purpura, megaloblastic anemia, muscle cramps, weakness, fatigue, dizziness, headache, dry mouth, impotence, sialadenitis. Thiazides alone have been shown to cause the following additional adverse reactions: paresthesias, vertigo, xanthopsia, transient blurred vision, allergic pneumonitis, pulmonary edema, respiratory distress, necrotizing vasculitis, exacerbation of lupus, aplastic anemia, agranulocytosis, hemolytic anemia. In neonates and infants: thrombocytopenia and pancreatitis—rarely, in newborns whose mothers have received thiazides during pregnancy.

Supplied: Capsules containing 25 mg. hydrochlorothiazide and 50 mg. triamterene, in bottles of 1000 capsules; in Single Unit Packages (unit-dose) of 100 (intended for institutional use only); in Patient-Pak™ unit-of-use bottles of 100.

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** Ad Hoc Committee on Cardiovascular Pulmonary Disease Risk Factors in Minority Populations. 1986 Forum on Cardiovascular Disease Risk Factors in Minority Populations Bethesda, Maryland U.S Department of Health and Human Services, 1986*

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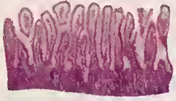
Aspirin and other nonsteroidal anti-inflammatory drugs weaken mucosal defenses, which may lead NSAID users to become prone to duodenal ulcers.[1] For those NSAID users who do develop duodenal ulcers, CARAFATE® (sucralfate/Marion) is ideal first-line therapy. Carafate rebuilds mucosal  defenses through a unique, nonsystemic mode of action. Carafate enhances the body's natural healing ability while it protects damaged mucosa from further injury. So the next time you see an arthritis patient with a duodenal ulcer, prescribe nonsystemic Carafate: therapy for the ulcer-prone patient.

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BRIEF SUMMARY

CONTRAINDICATIONS
There are no known contraindications to the use of sucralfate

PRECAUTIONS
Duodenal ulcer is a chronic, recurrent disease. While short-term treatment with sucralfate can result in complete healing of the ulcer, a successful course of treatment with sucralfate should not be expected to alter the post-healing frequency or severity of duodenal ulceration

Drug Interactions: Animal studies have shown that simultaneous administration of CARAFATE (sucralfate) with tetracycline, phenytoin, digoxin, or cimetidine will result in a statistically significant reduction in the bioavailability of these agents The bioavailability of these agents may be restored simply by separating the administration of these agents from that of CARAFATE by two hours This interaction appears to be nonsystemic in origin, presumably resulting from these agents being bound by CARAFATE in the gastrointestinal tract. The clinical significance of these animal studies is yet to be defined. However, because of the potential of CARAFATE to alter the absorption of some drugs from the gastrointestinal tract, the separate administration of CARAFATE from that of other agents should be considered when alterations in bioavailability are felt to be critical for concomitantly administered drugs

Carcinogenesis, Mutagenesis, Impairment of Fertility: Chronic oral toxicity studies of 24 months' duration were conducted in mice and rats at doses up to 1 gm/kg (12 times the human dose). There was no evidence of drug-related tumorigenicity A reproduction study in rats at doses up to 38 times the human dose did not reveal any indication of fertility impairment Mutagenicity studies were not conducted

Pregnancy: Teratogenic effects Pregnancy Category B Teratogenicity studies have been performed in mice, rats, and rabbits at doses up to 50 times the human dose and have revealed no evidence of harm to the fetus due to sucralfate There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when sucralfate is administered to a nursing woman

Pediatric Use: Safety and effectiveness in children have not been established

ADVERSE REACTIONS
Adverse reactions to sucralfate in clinical trials were minor and only rarely led to discontinuation of the drug In studies involving over 2,500 patients treated with sucralfate, adverse effects were reported in 121 (4 7%).

Constipation was the most frequent complaint (2 2%) Other adverse effects, reported in no more than one of every 350 patients, were diarrhea, nausea, gastric discomfort, indigestion, dry mouth, rash, pruritus, back pain, dizziness, sleepiness, and vertigo

OVERDOSAGE
There is no experience in humans with overdosage. Acute oral toxicity studies in animals, however, using doses up to 12 gm/kg body weight, could not find a lethal dose Risks associated with overdosage should, therefore, be minimal

DOSAGE AND ADMINISTRATION
The recommended adult oral dosage for duodenal ulcer is 1 gm four times a day on an empty stomach

Antacids may be prescribed as needed for relief of pain but should not be taken within one-half hour before or after sucralfate

While healing with sucralfate may occur during the first week or two, treatment should be continued for 4 to 8 weeks unless healing has been demonstrated by x-ray or endoscopic examination.

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Reference:
1 Eliakim R, Ophir M, Rachmilewitz D: *J Clin Gastroenterol* 1987, 9(4):395-399

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June 30–July 1. **Euthanasia: An Old/New Dilemma for Physicians.** San Francisco. Contact: Extended Programs in Medical Education, University of California, Room U-569, San Francisco, CA 94143-0742. Tel: (415) 476-4251.

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June 6. **Sudden Infant Death Syndrome (S.I.D.S.)—To Monitor or Not.** Marriott Hotel, Omaha. Contact: Cindy S. Hanssen, University of Nebraska Medical Center, Center for Continuing Education, 42nd & Dewey Ave, Omaha, NE 68105-1065. Tel: (800) 228-9630.

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June 17–July 1. **Radiology in Scandinavia and the Soviet Union.** Copenhagen, Stockholm, and Leningrad. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077-1925. Tel: (404) 641-9773.

ENGLAND

June 24–30. **Challenge 89—Decisions in Imaging.** London. Contact: University Imaging Associates, LAC/USC Medical Center, Box 66, 1200 North State St, Los Angeles, CA 90033. Tel: (213) 226-7245.

FRANCE

June 2–9. **"Paris in Spring" Meeting for Anesthesiologists.** Paris. Contact: George Silvay, MD, PhD, Department of Anesthesiology, Box 1010, Mt Sinai School of Medicine, 1 Gustave L. Levy Pl, New York, NY 10029-6574. Tel: (212) 241-7630.

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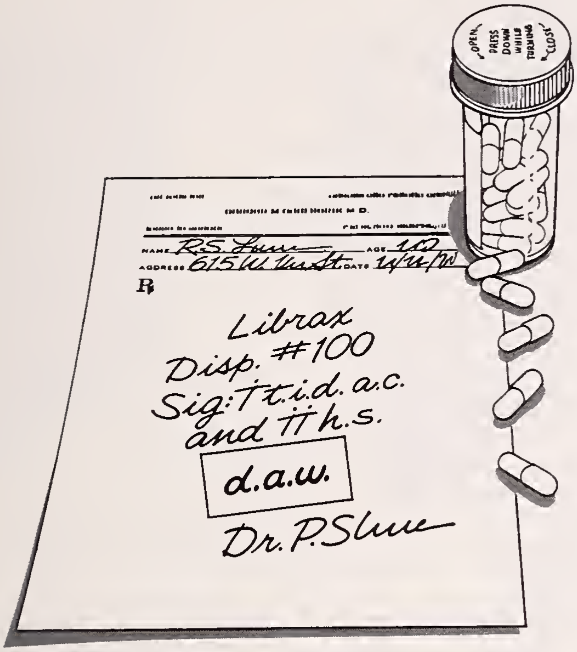

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THE PEACEMAKER.

Antianxiety
Antisecretory
Antispasmodic

Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium bromide.

Please consult complete prescribing information, a summary of which follows:

* **Indications:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows: "Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.
Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

> ***Usage in Pregnancy:*** **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur.

Withdrawal symptoms of the barbiturate type have occurred after discontinuation of benzodiazepines (see Drug Abuse and Dependence).

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established. Inform patients to consult physician before increasing dose or abruptly discontinuing this drug.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

Drug Abuse and Dependence: Withdrawal symptoms similar to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of chlordiazepoxide; more severe seen after excessive doses over extended periods; milder after taking continuously at therapeutic levels for several months. After extended therapy, avoid abrupt discontinuation and taper dosage. Carefully supervise addiction-prone individuals because of predisposition to habituation and dependence.

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*Librax has been evaluated as possibly effective as adjunctive therapy in the treatment of peptic ulcer and IBS.

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Please see summary of prescribing information on adjacent page.

NEW YORK STATE JOURNAL OF MEDICINE

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Contents

COMMENTARIES

Ticks, Lyme disease, and spotted fever 313
PASCAL JAMES IMPERATO, MD

Acute renal failure 314
RODRIGO E. URIZAR, MD; JILL A. LARGENT, MD; RICARDO MUNOZ, MD

RESEARCH PAPER

Pitfalls in Tay-Sachs carrier detection: Physician referral patterns and patient ignorance 317
DEBORAH A. SHAPIRO, BA; LAWRENCE R. SHAPIRO, MD

REVIEW ARTICLE

Selected tickborne infections: A review of Lyme disease, Rocky Mountain spotted fever, and babesiosis 320
MAHFOUZ H. ZAKI, MD, DrPH

ESSAYS IN PEDIATRICS

Acute renal failure in children: Etiology, diagnosis, and management 336
MELANIE SWEET, MD; KANWAL K. KHER, MD; SUDESH P. MAKKER, MD

HISTORY

The history of Kashin-Beck disease 343
LEON SOKOLOFF, MD

COMMITTEE REPORT

Prevention of perinatal transmission of hepatitis B virus: Prenatal screening of all pregnant women for hepatitis B surface antigen 352

CASE REPORTS

Cholestatic hepatitis after therapy with amoxicillin/clavulanate potassium 355
JOEL E. SCHNEIDER, MD; MARTIN S. KLEINMAN, MD; JAMES W. KUPIEC, MD

Use of gallium citrate Ga 67 scintigraphy in recurrent Merkel cell tumor 356
ORLANDO L. MANFREDI, MD; KENNETH ROMANO, MD; PAMELA JACKSON, BS

Perforation of a gastrojejunal ulcer into the pericardium 358
NURHAN YAZICIOGLU, MD; MEHMET C. OZ, MD; CEMI DEMIROGLU, MD

Postoperative fistula following insertion of a penile prosthesis in a diabetic patient 359
MITCHELL H. BAMBERGER, MD; ALEXANDER SOTIROPOULOS, MD

LETTERS TO THE EDITOR

Buttocks blow barotrauma and pneumomediastinum 361
FRANK M. TAMARIN, MD; BERNARD GITLER, MD; JOSE PEREZ, MD; ROBERT D. BRANDSTETTER, MD

Needle localization of occult breast cancer and subsequent management 361
HENRY SCHNEIDERMAN, MD; JONATHAN R. SPORN, MD; PETER DECKERS, MD

Universal precautions 362
PAMELA VAN WAGENEN

Penile prosthesis as an adjunct to intermittent self-catheterization in patients with neuropathic bladders 363
JERRY J. WEINBERG, MD, GOPAL H. BADLANI, MD

ESSAY REVIEW

Family portraits from a family practice 364
PASCAL JAMES IMPERATO, MD

LEADS FROM EPIDEMIOLOGY NOTES 366
BOOK REVIEWS 369
BOOKS RECEIVED 373
NEWS BRIEFS 374
MEDICAL SOCIETY NEWS 376
OBITUARIES 378
MEDICAL MEETINGS AND LECTURES 6A

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Indications and Usage: Axid is indicated for up to eight weeks for the treatment of active duodenal ulcer. In most patients, the ulcer will heal within four weeks.

Axid is indicated for maintenance therapy for duodenal ulcer patients at a reduced dosage of 150 mg h.s. after healing of an active duodenal ulcer. The consequences of continuous therapy with Axid for longer than one year are not known.

Contraindication: Axid is contraindicated in patients with known hypersensitivity to the drug and should be used with caution in patients with hypersensitivity to other H_2-receptor antagonists.

Precautions: *General* – 1. Symptomatic response to nizatidine therapy does not preclude the presence of gastric malignancy.

2. Because nizatidine is excreted primarily by the kidney, dosage should be reduced in patients with moderate to severe renal insufficiency.

3. Pharmacokinetic studies in patients with hepatorenal syndrome have not been done. Part of the dose of nizatidine is metabolized in the liver. In patients with normal renal function and uncomplicated hepatic dysfunction, the disposition of nizatidine is similar to that in normal subjects.

Laboratory Tests – False-positive tests for urobilinogen with Multistix® may occur during therapy with nizatidine.

Drug Interactions – No interactions have been observed between Axid and theophylline, chlordiazepoxide, lorazepam, lidocaine, phenytoin, and warfarin. Axid does not inhibit the cytochrome P-450-linked drug-metabolizing enzyme system; therefore, drug interactions mediated by inhibition of hepatic metabolism are not expected to occur. In patients given very high doses (3,900 mg) of aspirin daily, increases in serum salicylate levels were seen when *nizatidine*, 150 mg b.i.d., was administered concurrently.

Carcinogenesis, Mutagenesis, Impairment of Fertility – A two-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a two-year study in mice, there was no evidence of a carcinogenic effect in male mice; although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups. The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid.

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, mouse lymphoma assay, chromosome aberration tests, and a micronucleus test.

In a two-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy – Teratogenic Effects – Pregnancy Category C – Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in one fetus and at 50 mg/kg it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in one fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers – Studies conducted in lactating women have shown that <0.1% of the administered oral dose of nizatidine is secreted in human milk in proportion to plasma concentrations. Caution should be exercised when administering nizatidine to a nursing mother.

Pediatric Use – Safety and effectiveness in children have not been established.

Use in Elderly Patients – Ulcer healing rates in elderly patients are similar to those in younger age groups. The incidence rates of adverse events and laboratory test abnormalities are also similar to those seen in other age groups. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Clinical trials of nizatidine included almost 5,000 patients given nizatidine in studies of varying durations. Domestic placebo-controlled trials included over 1,900 patients given nizatidine and over 1,300 given placebo. Among reported adverse events in the domestic placebo-controlled trials, sweating (1% vs 0.2%), urticaria (0.5% vs < 0.01%), and somnolence (2.4% vs 1.3%) were significantly more common in the nizatidine group. A variety of less common events was also reported; it was not possible to determine whether these were caused by nizatidine.

Hepatic – Hepatocellular injury, evidenced by elevated liver enzyme tests (SGOT [AST], SGPT [ALT], or alkaline phosphatase), occurred in some patients and was possibly or probably related to nizatidine. In some cases, there was marked elevation of SGOT, SGPT enzymes (greater than 500 IU/L) and, in a single instance, SGPT was greater than 2,000 IU/L. The overall rate of occurrences of elevated liver enzymes and elevations to three times the upper limit of normal, however, did not significantly differ from the rate of liver enzyme abnormalities in placebo-treated patients. All abnormalities were reversible after discontinuation of Axid.

Cardiovascular – In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in two individuals administered Axid and in three untreated subjects.

CNS – Rare cases of reversible mental confusion have been reported.

Endocrine – Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to Axid. Impotence and decreased libido were reported with equal frequency by patients who received Axid and by those given placebo. Rare reports of gynecomastia occurred.

Hematologic – Fatal thrombocytopenia was reported in a patient who was treated with Axid and another H_2-receptor antagonist. On previous occasions, this patient had experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported.

Integumental – Sweating and urticaria were reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported.

Hypersensitivity – As with other H_2-receptor antagonists, rare cases of anaphylaxis following administration of nizatidine have been reported. Because cross-sensitivity in this class of compounds has been observed, H_2-receptor antagonists should not be administered to individuals with a history of previous hypersensitivity to these agents. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

Other – Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine administration have been reported.

Overdosage: Overdoses of Axid have been reported rarely. The following is provided to serve as a guide should such an overdose be encountered.

Signs and Symptoms – There is little clinical experience with overdosage of Axid in humans. Test animals that received large doses of nizatidine have exhibited cholinergic-type effects, including lacrimation, salivation, emesis, miosis, and diarrhea. Single oral doses of 800 mg/kg in dogs and of 1,200 mg/kg in monkeys were not lethal. Intravenous median lethal doses in the rat and mouse were 301 mg/kg and 232 mg/kg respectively.

Treatment – To obtain up-to-date information about the treatment of overdose, a good resource is your certified regional Poison Control Center. Telephone numbers of certified poison control centers are listed in the *Physicians' Desk Reference (PDR)*. In managing overdosage, consider the possibility of multiple drug overdoses, interaction among drugs, and unusual drug kinetics in your patient.

If overdosage occurs, use of activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. Renal dialysis for four to six hours increased plasma clearance.

PV 2096 AMP [013089]

Additional information available to the profession on request.

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[1] Data on file, Lilly Research Laboratories.

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VASOTEC®
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Contraindications: VASOTEC® (Enalapril Maleate, MSD) is contraindicated in patients who are hypersensitive to this product and in patients with a history of angioedema related to previous treatment with an ACE inhibitor.

Warnings: *Angioedema* Angioedema of the face, extremities, lips, tongue, glottis, and/or larynx has been reported in patients treated with ACE inhibitors, including VASOTEC. In such cases, VASOTEC should be promptly discontinued and the patient carefully observed until the swelling disappears. In instances where swelling has been confined to the face and lips, the condition has generally resolved without treatment, although antihistamines have been useful in relieving symptoms. Angioedema associated with laryngeal edema may be fatal. **Where there is involvement of the tongue, glottis, or larynx likely to cause airway obstruction, appropriate therapy, e.g., subcutaneous epinephrine solution 1:1000 (0.3 mL to 0.5 mL), should be promptly administered.** (See ADVERSE REACTIONS.)

Hypotension: Excessive hypotension is rare in uncomplicated hypertensive patients treated with VASOTEC alone. Heart failure patients given VASOTEC commonly have some reduction in blood pressure, especially with the first dose, but discontinuation of therapy for continuing symptomatic hypotension usually is not necessary when dosing instructions are followed; caution should be observed when initiating therapy. (See DOSAGE AND ADMINISTRATION.) Patients at risk for excessive hypotension, sometimes associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death, include those with the following conditions or characteristics: heart failure, hyponatremia, high-dose diuretic therapy, recent intensive diuresis or increase in diuretic dose, renal dialysis, or severe volume and/or salt depletion of any etiology. It may be advisable to eliminate the diuretic (except in heart failure patients), reduce the diuretic dose, or increase salt intake cautiously before initiating therapy with VASOTEC in patients at risk for excessive hypotension who are able to tolerate such adjustments. (See PRECAUTIONS, *Drug Interactions* and ADVERSE REACTIONS.) In patients at risk for excessive hypotension, therapy should be started under very close medical supervision and such patients should be followed closely for the first two weeks of treatment and whenever the dose of enalapril and/or diuretic is increased. Similar considerations may apply to patients with ischemic heart disease or cardiovascular disease in whom an excessive fall in blood pressure could result in a myocardial infarction or cerebrovascular accident.

If excessive hypotension occurs, the patient should be placed in supine position and, if necessary, receive an intravenous infusion of normal saline. A transient hypotensive response is not a contraindication to further doses of VASOTEC, which usually can be given without difficulty once the blood pressure has stabilized. If symptomatic hypotension develops, a dose reduction or discontinuation of VASOTEC or concomitant diuretic may be necessary.

Neutropenia/Agranulocytosis: Another ACE inhibitor, captopril, has been shown to cause agranulocytosis and bone marrow depression, rarely in uncomplicated patients but more frequently in patients with renal impairment, especially if they also have a collagen vascular disease. Available data from clinical trials of enalapril are insufficient to show that enalapril does not cause agranulocytosis at similar rates. Foreign marketing experience has revealed several cases of neutropenia or agranulocytosis in which a causal relationship to enalapril cannot be excluded. Periodic monitoring of white blood cell counts in patients with collagen vascular disease and renal disease should be considered.

Precautions: *General: Impaired Renal Function:* As a consequence of inhibiting the renin-angiotensin-aldosterone system, changes in renal function may be anticipated in susceptible individuals. In patients with severe heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with ACE inhibitors, including VASOTEC, may be associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death.

In clinical studies in hypertensive patients with unilateral or bilateral renal artery stenosis, increases in blood urea nitrogen and serum creatinine were observed in 20% of patients. These increases were almost always reversible upon discontinuation of enalapril and/or diuretic therapy. In such patients, renal function should be monitored during the first few weeks of therapy.

Some patients with hypertension or heart failure with no apparent preexisting renal vascular disease have developed increases in blood urea and serum creatinine, usually minor and transient, especially when VASOTEC has been given concomitantly with a diuretic. This is more likely to occur in patients with preexisting renal impairment. Dosage reduction and/or discontinuation of the diuretic and/or VASOTEC may be required.

Evaluation of patients with hypertension or heart failure should always include assessment of renal function. (See DOSAGE AND ADMINISTRATION.)

Hyperkalemia Elevated serum potassium ($>$ 5.7 mEq/L) was observed in approximately 1% of hypertensive patients in clinical trials. In most cases these were isolated values which resolved despite continued therapy. Hyperkalemia was a cause of discontinuation of therapy in 0.28% of hypertensive patients. In clinical trials in heart failure, hyperkalemia was observed in 3.8% of patients, but was not a cause for discontinuation.

Risk factors for the development of hyperkalemia include renal insufficiency, diabetes mellitus, and the concomitant use of potassium-sparing diuretics, potassium supplements, and/or potassium-containing salt substitutes, which should be used cautiously, if at all, with VASOTEC. (See *Drug Interactions.*)

Surgery/Anesthesia: In patients undergoing major surgery or during anesthesia with agents that produce hypotension, enalapril may block angiotensin II formation secondary to compensatory renin release. If hypotension occurs and is considered to be due to this mechanism, it can be corrected by volume expansion.

Information for Patients:

Angioedema Angioedema, including laryngeal edema, may occur especially following the first dose of enalapril. Patients should be so advised and told to report immediately any signs or symptoms suggesting angioedema (swelling of face, extremities, eyes, lips, tongue, difficulty in swallowing or breathing) and to take no more drug until they have consulted with the prescribing physician.

Hypotension: Patients should be cautioned to report lightheadedness especially during the first few days of therapy. If actual syncope occurs, the patients should be told to discontinue the drug until they have consulted with the prescribing physician.

All patients should be cautioned that excessive perspiration and dehydration may lead to an excessive fall in blood pressure because of reduction in fluid volume. Other causes of volume depletion such as vomiting or diarrhea may also lead to a fall in blood pressure; patients should be advised to consult with the physician.

Hyperkalemia Patients should be told not to use salt substitutes containing potassium without consulting their physician.

Neutropenia Patients should be told to report promptly any indication of infection (e.g., sore throat, fever) which may be a sign of neutropenia.

NOTE: As with many other drugs, certain advice to patients being treated with enalapril is warranted. This information is intended to aid in the safe and effective use of this medication. It is not a disclosure of all possible adverse or intended effects.

Drug Interactions:

Hypotension. Patients on Diuretic Therapy Patients on diuretics and especially those in whom diuretic therapy was recently instituted may occasionally experience an excessive reduction of blood pressure after initiation of therapy with enalapril. The possibility of hypotensive effects with enalapril can be minimized by either discontinuing the diuretic or increasing the salt intake prior to initiation of treatment with enalapril. If it is necessary to continue the diuretic, provide close medical supervision after the initial dose for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and DOSAGE AND ADMINISTRATION.)

Agents Causing Renin Release: The antihypertensive effect of VASOTEC is augmented by antihypertensive agents that cause renin release (e.g., diuretics).

Other Cardiovascular Agents: VASOTEC has been used concomitantly with beta-adrenergic-blocking agents, methyldopa, nitrates, calcium-blocking agents, hydralazine, prazosin, and digoxin without evidence of clinically significant adverse interactions.

Agents Increasing Serum Potassium. VASOTEC attenuates potassium loss caused by thiazide-type diuretics. Potassium-sparing diuretics (e.g., spironolactone, triamterene, or amiloride), potassium supplements, or potassium-containing salt substitutes may lead to significant increases in serum potassium. Therefore, if concomitant use of these agents is indicated because of demonstrated hypokalemia, they should be used with caution and with frequent monitoring of serum potassium. Potassium-sparing agents should generally not be used in patients with heart failure receiving VASOTEC.

Lithium. A few cases of lithium toxicity have been reported in patients receiving concomitant VASOTEC and lithium and were reversible upon discontinuation of both drugs. Although a causal relationship has not been established, it is recommended that caution be exercised when lithium is used concomitantly with VASOTEC and serum lithium levels should be monitored frequently.

Pregnancy—Category C. There was no fetotoxicity or teratogenicity in rats treated with up to 200 mg/kg/day of enalapril (333 times the maximum human dose). Fetotoxicity, expressed as a decrease in average fetal weight, occurred in rats given 1200 mg/kg/day of enalapril but did not occur when these animals were supplemented with saline. Enalapril was not teratogenic in rabbits. However, maternal and fetal toxicity occurred in some rabbits at doses of 1 mg/kg/day or more. Saline supplementation prevented the maternal and fetal toxicity seen at doses of 3 and 10 mg/kg/day, but not at 30 mg/kg/day (50 times the maximum human dose).

Radioactivity was found to cross the placenta following administration of labeled enalapril to pregnant hamsters.

There are no adequate and well-controlled studies in pregnant women. VASOTEC® (Enalapril Maleate, MSD) should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers Milk in lactating rats contains radioactivity following administration of ^{14}C enalapril maleate. It is not known whether this drug is secreted in human milk. Because many drugs are secreted in human milk, caution should be exercised when VASOTEC is given to a nursing mother.

Pediatric Use: Safety and effectiveness in children have not been established.

Adverse Reactions: VASOTEC has been evaluated for safety in more than 10,000 patients, including over 1000 patients treated for one year or more. VASOTEC has been found to be generally well tolerated in controlled clinical trials involving 2987 patients.

Hypertension. The most frequent clinical adverse experiences in controlled trials were: headache (5.2%), dizziness (4.3%), and fatigue (3%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in controlled clinical trials were: diarrhea (1.4%), nausea (1.4%), rash (1.4%), cough (1.3%), orthostatic effects (1.2%), and asthenia (1.1%).

Heart Failure: The most frequent clinical adverse experiences in both controlled and uncontrolled trials were: dizziness (7.9%), hypotension (6.7%), orthostatic effects (2.2%), syncope (2.2%), cough (2.2%), chest pain (2.1%), and diarrhea (2.1%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in both controlled and uncontrolled clinical trials were: fatigue (1.8%), headache (1.8%), abdominal pain (1.6%), asthenia (1.6%), orthostatic hypotension (1.6%), vertigo (1.6%), angina pectoris (1.5%), nausea (1.3%), vomiting (1.3%), bronchitis (1.3%), dyspnea (1.3%), urinary tract infection (1.3%), rash (1.3%), and myocardial infarction (1.2%).

Other serious clinical adverse experiences occurring since the drug was marketed or adverse experiences occurring in 0.5% to 1% of patients with hypertension or heart failure in clinical trials in order of decreasing severity within each category:

Cardiovascular: Myocardial infarction or cerebrovascular accident, possibly secondary to excessive hypotension in high-risk patients (see WARNINGS, *Hypotension*); cardiac arrest, pulmonary embolism and infarction, rhythm disturbances; atrial fibrillation, palpitation.

Digestive. Ileus, pancreatitis, hepatitis or cholestatic jaundice, melena, anorexia, dyspepsia, constipation, glossitis.

Nervous/Psychiatric. Depression, confusion, ataxia, somnolence, insomnia, nervousness, paresthesia.

Urogenital: Renal failure, oliguria, renal dysfunction (see PRECAUTIONS and DOSAGE AND ADMINISTRATION), prostate hypertrophy.

Respiratory: Bronchospasm, rhinorrhea, asthma, upper respiratory infection.

Skin: Herpes zoster, pruritus, alopecia, flushing, photosensitivity.

Other Muscle cramps, hyperhidrosis, impotence, blurred vision, taste alteration, tinnitus.

A symptom complex has been reported which may include fever, myalgia, and arthralgia; an elevated erythrocyte sedimentation rate may be present. Rash or other dermatologic manifestations may occur. These symptoms have disappeared after discontinuation of therapy.

Angioedema: Angioedema has been reported in patients receiving VASOTEC (0.2%). Angioedema associated with laryngeal edema may be fatal. If angioedema of the face, extremities, lips, tongue, glottis, and/or larynx occurs, treatment with VASOTEC should be discontinued and appropriate therapy instituted immediately. (See WARNINGS.)

Hypotension: In the hypertensive patients, hypotension occurred in 0.9% and syncope occurred in 0.5% of patients following the initial dose or during extended therapy. Hypotension or syncope was a cause for discontinuation of therapy in 0.1% of hypertensive patients. In heart failure patients, hypotension occurred in 6.7% and syncope occurred in 2.2% of patients. Hypotension or syncope was a cause for discontinuation of therapy in 1.9% of patients with heart failure. (See WARNINGS.)

Clinical Laboratory Test Findings:

Serum Electrolytes. Hyperkalemia (see PRECAUTIONS), hyponatremia.

Creatinine, Blood Urea Nitrogen: In controlled clinical trials, minor increases in blood urea nitrogen and serum creatinine, reversible upon discontinuation of therapy, were observed in about 0.2% of patients with essential hypertension treated with VASOTEC alone. Increases are more likely to occur in patients receiving concomitant diuretics or in patients with renal artery stenosis. (See PRECAUTIONS.) In patients with heart failure who were also receiving diuretics with or without digitalis, increases in blood urea nitrogen or serum creatinine, usually reversible upon discontinuation of VASOTEC and/or other concomitant diuretic therapy, were observed in about 11% of patients. Increases in blood urea nitrogen or creatinine were a cause for discontinuation in 1.2% of patients.

Hemoglobin and Hematocrit: Small decreases in hemoglobin and hematocrit (mean decreases of approximately 0.3 g % and 1.0 vol %, respectively) occur frequently in either hypertension or heart failure patients treated with VASOTEC but are rarely of clinical importance unless another cause of anemia coexists. In clinical trials, less than 0.1% of patients discontinued therapy due to anemia.

Other (Causal Relationship Unknown): In marketing experience, rare cases of neutropenia, thrombocytopenia, and bone marrow depression have been reported.

Liver Function Tests: Elevations of liver enzymes and/or serum bilirubin have occurred.

Dosage and Administration: *Hypertension:* In patients who are currently being treated with a diuretic, symptomatic hypotension occasionally may occur following the initial dose of VASOTEC. The diuretic should, if possible, be discontinued for two to three days before beginning therapy with VASOTEC to reduce the likelihood of hypotension. (See WARNINGS.) If the patient's blood pressure is not controlled with VASOTEC alone, diuretic therapy may be resumed.

If the diuretic cannot be discontinued, an initial dose of 2.5 mg should be used under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, *Drug Interactions.*)

The recommended initial dose in patients not on diuretics is 5 mg once a day. Dosage should be adjusted according to blood pressure response. The usual dosage range is 10 to 40 mg per day administered in a single dose or in two divided doses. In some patients treated once daily, the antihypertensive effect may diminish toward the end of the dosing interval. In such patients, an increase in dosage or twice-daily administration should be considered. If blood pressure is not controlled with VASOTEC alone, a diuretic may be added.

Concomitant administration of VASOTEC with potassium supplements, potassium salt substitutes, or potassium-sparing diuretics may lead to increases of serum potassium (see PRECAUTIONS).

Dosage Adjustment in Hypertensive Patients with Renal Impairment: The usual dose of enalapril is recommended for patients with a creatinine clearance $>$30 mL/min (serum creatinine of up to approximately 3 mg/dL). For patients with creatinine clearance $\leq$30 mL/min (serum creatinine $\geq$3 mg/dL), the first dose is 2.5 mg once daily. The dosage may be titrated upward until blood pressure is controlled or to a maximum of 40 mg daily.

Heart Failure: VASOTEC is indicated as adjunctive therapy with diuretics and digitalis. The recommended starting dose is 2.5 mg once or twice daily. After the initial dose of VASOTEC, the patient should be observed under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, *Drug Interactions.*) If possible, the dose of the diuretic should be reduced, which may diminish the likelihood of hypotension. The appearance of hypotension after the initial dose of VASOTEC does not preclude subsequent careful dose titration with the drug, following effective management of the hypotension. The usual therapeutic dosing range for the treatment of heart failure is 5 to 20 mg daily given in two divided doses. The maximum daily dose is 40 mg. Once-daily dosing has been effective in a controlled study, but nearly all patients in this study were given 40 mg, the maximum recommended daily dose, and there has been much more experience with twice-daily dosing. In addition, in a placebo-controlled study which demonstrated reduced mortality in patients with severe heart failure (NYHA Class IV), patients were treated with 2.5 to 40 mg per day of VASOTEC, almost always administered in two divided doses. (See CLINICAL PHARMACOLOGY, *Pharmacodynamics and Clinical Effects.*) Dosage may be adjusted depending upon clinical or hemodynamic response. (See WARNINGS.)

Dosage Adjustment in Heart Failure Patients with Renal Impairment or Hyponatremia In heart failure patients with hyponatremia (serum sodium $<$130 mEq/L) or with serum creatinine $>$1.6 mg/dL, therapy should be initiated at 2.5 mg daily under close medical supervision. (See DOSAGE AND ADMINISTRATION, *Heart Failure*, WARNINGS, and PRECAUTIONS, *Drug Interactions.*) The dose may be increased to 2.5 mg b.i.d., then 5 mg b.i.d. and higher as needed, usually at intervals of four days or more, if at the time of dosage adjustment there is not excessive hypotension or significant deterioration of renal function. The maximum daily dose is 40 mg.

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Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

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Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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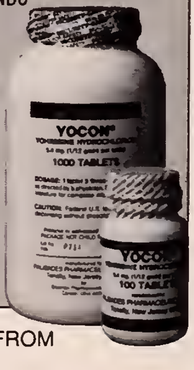

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices. Information must be submitted at least three months prior to the event.*

JULY 1989

AROUND THE STATE

MANHATTAN

July 10–13. **Neuropharmacology of Serotonin.** Vista International Hotel. Contact: Conference Director, The New York Academy of Sciences, 2 E 63rd St, New York, NY 10021. Tel: (212) 838-0230.

SARATOGA SPRINGS

July 6–7. **Regional Conference on Chronic Pain and Its Management.** Ramada Renaissance. Contact: Barbara G. Post, Program Coordinator, Sunnyview Hospital and Rehabilitation Center, 1270 Belmont Ave, Schenectady, NY 12308. Tel: (518) 382-4500.

AROUND THE NATION

CALIFORNIA

July 21–22. **American In-Vitro Allergy/Immunology Society Annual Program.** 12 Cat I Credits. San Diego Marriott Hotel and Marina. Contact: Betty Kahler, Administrator, American In-Vitro Allergy/Immunology Society, PO Box 459, Lake Jackson, TX 77566. Tel: (409) 297-5636.

July 17–30. **Third Annual Symposium on Magnetic Resonance Imaging.** 23 Cat I Credits. Ritz-Carleton Resort Hotel, Laguna Niguel. *July 31–Aug 4.* **Modern Radiology Practice.** 24 Cat I Credits. Four Seasons Biltmore Hotel, Santa Barbara. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 3077-1925. Tel: (404) 641-9773.

MASSACHUSETTS

July 13–15. **Advances in Cardiology.** 15 Cat I Credits. Country Inn and Conference Center at Jiminy Peak, Hancock. *July 20–22.* **Special Challenges in General Medicine.** 15 Cat I Credits. Country Inn and Conference Center at Jiminy Peak, Hancock. Contact: Berkshire Area Health Education Center,

(*continued on p 18A*)

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Carcinogenesis, Mutagenesis, Impairment of Fertility: Chronic oral toxicity studies of 24 months' duration were conducted in mice and rats at doses up to 1 gm/kg (12 times the human dose) There was no evidence of drug-related tumorigenicity A reproduction study in rats at doses up to 38 times the human dose did not reveal any indication of fertility impairment Mutagenicity studies were not conducted

Pregnancy: Teratogenic effects Pregnancy Category B Teratogenicity studies have been performed in mice, rats, and rabbits at doses up to 50 times the human dose and have revealed no evidence of harm to the fetus due to sucralfate There are, however, no adequate and well-controlled studies in pregnant women Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed

Nursing Mothers: It is not known whether this drug is excreted in human milk Because many drugs are excreted in human milk, caution should be exercised when sucralfate is administered to a nursing woman

Pediatric Use: Safety and effectiveness in children have not been established

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Don't miss this opportunity to gain Category 1 credits over a weekend when you are free from office hours. Bring your family to New York City to enjoy the many sights.

All physicians, members of the allied professions, and their guests are invited to attend the Medical Society of the State of New York's Annual CME Assembly to be held September 15–16, 1989, at the New York Hilton. The two days of meetings will provide the individual physician with a possible 14 credits out of a total of 68 Category 1, CME credit hours being offered. The Medical Society of the State of New York is accredited by the Accreditation Council for continuing Medical Education to sponsor CME for physicians.

The theme of the CME sessions will be "Case Management and Clinical Care Dilemmas." The chosen subjects are varied and topical, e.g., the battered woman syndrome (Psychiatry)—child abuse (Pediatrics/Forensic Medicine)—cocaine (Substance Abuse)—osteoporosis (American Medical Women's Assoc./Orthopaedic Surgery)—malpractice (Mock Trial, Medical Liability Mutual Insurance Co.) are but a few. . . . Our programs are designed to be interesting and informative.

HOTEL THEATRE DESK

Located in the main lobby of the New York Hilton to accommodate those persons wishing to purchase tickets to any of the various New York theatres.

TECHNICAL EXHIBITS

Technical exhibits include pharmaceutical products, medical and surgical suppliers, health and pension plans, office management systems and more. Representatives from several companies will be in the Rhinelander Gallery Exhibit Hall on Friday and Saturday to introduce you to their products and services.

HOTEL RESERVATIONS

Physicians must make their own reservations with the New York Hilton, no later than August 31, 1989. After that date, rooms are on an availability basis only. If making reservations by telephone, please mention that you are attending the Medical Society Convention for billing at the special rate.

AWARDS TO BE GIVEN AT THE CME ASSEMBLY

Suiter Lectureship presented during the Internal Medicine Program on Saturday, 9/16. Suiter Lectureship is given for a non-surgical subject. The recipient of this award will be announced.

Albert F. R. Andresen Memorial Lecture presented during the Gastroenterology/Colon and Rectal Surgery Program on Friday, 9/15. The Albert F. R. Andresen Memorial Lecture is for a lecture on Gastroenterology. Adrian J. Greenstein, M.D., Professor of Surgery, Mount Sinai School of Medicine, NYC will receive this award.

Lucien Howe Prize presented during the Neurology/Ophthalmology Program on Saturday, 9/16. Lucien Howe Prize is given for the best original contribution to our knowledge of some branch of surgery, preferably ophthalmology. Myles Behrens, M.D. of Columbia University, NYC will be the recipient of this award.

1989 ANNUAL CME ASSEMBLY

Medical Society of the State of New York
The New York Hilton, New York City
September 15–16, 1989

"CASE MANAGEMENT AND CLINICAL CARE DILEMMAS"

FRIDAY, SEPTEMBER 15

8:30 a.m.–9:30 a.m.	Medical Technology, *Nassau A*
9:30 a.m.–12:30 p.m.	Gastroenterology/Colon & Rectal Surgery, *Sutton Center*
	Computers in Medicine, *Nassau A*
	Occupational & Environmental Health/Family Practice, *Murray Hill B*
	Physical Medicine & Rehabilitation, *Murray Hill A*
	Medical Liability Mutual Insurance Company, *Sutton South*
2:00 p.m.–5:00 p.m.	Chest Diseases/Radiology, *Sutton Center*
	Cardiovascular Diseases, *Sutton South*
	American Medical Women's Association/Orthopaedic Surgery Society, *Murray Hill A*
	Neurology, *Nassau A*
	Neurosurgery, *Murray Hill B*
	Emergency Medicine/Critical Care, *Nassau B*

SATURDAY, SEPTEMBER 16

8:30 a.m.–9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m.–12:30 p.m.	Substance Abuse, *Sutton North*
	Internal Medicine (All Day), *Sutton Center*
	Neurology/Ophthalmology (All Day), *Beekman Parlor*
	Psychiatry (All Day), *Nassau B*
	Obstetrics & Gynecology/Urology, *Murray Hill A*
	General Surgery, *Sutton South*
	Dermatology/Plastic Reconstructive Surgery (All Day), *Nassau A*
	Allergy & Immunology, *Sutton North*
2:00 p.m.–5:00 p.m.	Internal Medicine (All Day), *Sutton Center*
	Neurology/Ophthalmology (All Day), *Beekman Parlor*
	Psychiatry (All Day), *Nassau B*
	Pediatrics/Forensic Medicine, *Sutton North*
	Otolaryngology/Neurosurgery/Plastic Reconstructive Surgery, *Sutton South*
	Plastic Surgery, *Regent Parlor*
	Dermatology/Plastic Reconstructive Surgery (All Day), *Nassau A*

CME CREDITS: The Medical Society of the State of New York (MSSNY) is accredited by the Accreditation Council for Continuing Medical Education to sponsor CME for physicians. MSSNY designates all CME activities in this CME Assembly as meeting the criteria for Category 1 of the AMA, PRA. The credits are enumerated for each session. Those programs approved for credit by the American Academy of Family Physicians (AAFP) are so designated.

◆

Technical Exhibits

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Advance Registration Form

1989 Annual CME Assembly, September 15–16
The New York Hilton, New York City

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COMMENTARIES

Ticks, Lyme disease, and spotted fever

Ticks have long been regarded by many nonphysicians primarily as harmless ecto-parasites rather than as vectors of serious disease. This long held view emerged from the empirical observation that tick bite exposure rarely resulted in such serious diseases as Rocky Mountain spotted fever, tick paralysis, babesiosis, and an assortment of other bacterial and viral diseases. With the appearance of Lyme disease, that perception is changing.

The intensification and frequency of contact between man and ticks have increased as highways and suburbs have brought people closer to open and wild areas where these arthropods and their usual hosts live. In the cities nature's reclaiming of abandoned areas and the establishment of pathogens among ticks in city parks and open spaces can carry tickborne infections over the urban frontier.[1,2] For many years, Rocky Mountain spotted fever in New York City was only observed among those who acquired it elsewhere, frequently on Long Island.[3] That has now changed, at least in one small area of the Bronx.[1,2] It is too early to tell if the cluster of cases acquired in the Bronx in 1987 and reported by Salgo et al[1] in 1988 are predictive of future, wider trends or merely an isolated occurrence in a discrete subenvironment. This occurrence, however, tells us that urban boundaries may not provide protection from the pathogens which ticks can transmit.

The principal vector of Rocky Mountain spotted fever, *Dermacentor variabilis*, the dog tick, never became a refugee from progressive urbanization. Rather it retreated to viable corners of the city where it has continued to thrive. During most of the 1970s, regular annual analyses of captured dog ticks in New York City revealed them to be free of *Rickettsia rickettsii*, the causative agent of Rocky Mountain spotted fever (personal observations, New York City Department of Health, 1972–1978). As documented in the late 1980s this may no longer be the case, at least in the Soundview Park section of the Bronx.[1]

Although cases of Rocky Mountain spotted fever and babesiosis regularly occur every year in the eastern United States, their total numbers and incidence are not great enough to make them prime suspects for many physicians in diagnosing febrile illnesses. Many physicians have never encountered a case of either Rocky Mountain spotted fever or babesiosis. For them these diseases have remained rarities, whose modes of transmission and clinical features have been all but forgotten. Lyme disease has now begun to alert physicians, not only to it, but also to other tickborne infections.

Unlike the large dog tick which transmits Rocky Mountain spotted fever, the deer tick vector of Lyme disease in the eastern United States (*Ixodes dammini*) is extremely small, no larger than a grain of pepper when not engorged with blood. This tick also transmits babesiosis. Lyme disease is now the most prevalent arthropod transmitted infection in the United States. It is also a disease which is second only to acquired immunodeficiency syndrome (AIDS) in the level of press and media coverage that it has received. At present the disease is confined in New York State to six counties and within those counties to specific "hot spots" where the vector, causative spirochete (*Borrelia burgdorferi*), animal reservoirs, and humans come together. The clinical diagnosis, while clear-cut in some instances, is more elusive in others. Serologic confirmation of both early and late Lyme disease is hampered by the less than ideal levels of specificity and sensitivity of currently available tests.

In this issue of the *Journal*, Zaki[4] presents a comprehensive overview of Lyme disease, babesiosis, and Rocky Mountain spotted fever. These are the three tickborne diseases of greatest concern in this part of the country. From this review, it is obvious that our knowledge of Lyme disease, its management, and its prevention are still evolving. Yet another message is also clear. Physicians must think of tickborne infections whenever they are confronted with patients with febrile illnesses. The fact that both tick vectors are geographically widespread coupled with the presence of wild hosts means that the introduction of the causative agents and subsequent disease can occur. Thus the appearance of "hot spots" of Lyme disease[5] or of Rocky Mountain spotted fever in areas previously free of these diseases should come as no surprise. This means that neither disease can be ruled out on the basis of a negative travel history to known endemic areas.

The epidemiology of Lyme disease is complex, involving an arthropod vector with a developmental life cycle dependent on two different animal hosts, the white-footed mouse and the white-tailed deer. Control of the disease is equally complex and facile solutions such as destroying deer populations are of questionable value. There are a number of currently available pesticides that are highly effective against *Ixodes dammini* ticks. Their widespread use in the prevention of tickborne diseases is impeded by a number of factors including expense, toxicity, and lack of residual killing capacity a few days after spraying. Sevin, whose active ingredient is carbaryl, is highly effective against adult ticks. Although it is not toxic to humans when used as directed by manufacturers, it is extremely toxic to bees. It is presently being evaluated under field conditions on Long Island. Diazinon, which was found to be highly effective in field tests conducted in New Jersey, is lethal to adult ticks. However, it is also very toxic to birds. Dursban is toxic to adult ticks and is presently undergoing field tests in Connecticut.[6]

The most promising of the pesticides is Damminix, whose active ingredient is permethrin. This product appears to have little toxicity for man and other mammals but is very toxic to tick larvae and nymphs. Field tests with it have focused on using saturated cotton balls which are in turn inserted into biodegradable cardboard tubes. The tubes are placed in locations frequented by mice which take the cotton back to their nests. There the product kills the tick nymphs and larvae without harming the mice. Unfortunately, Damminix tubes must be distributed every ten feet or so for them to have an appreciable impact on tick populations. Because Damminix is an expensive product, it is unlikely that it will be widely used for tick control in the immediate future. A significant decline in cost would place it in a front line position for tick control because of its efficacy and low toxicity. As Zaki[4] suggests, much research needs to be done before we can elaborate successful and acceptable strategies for broad environment-based control.

Meanwhile, there is much that individuals can do to minimize their exposure to ticks as outlined by Zaki.[4] The correct removal of already attached ticks, their preservation, and their analysis for pathogens are important steps in determining risk exposure and necessary medical interventions. Even in the absence of a history of tick exposure, physicians must be alert to the possibility of a tickborne infection in anyone presenting with an obscure febrile illness. The diagnosis of these diseases is frequently rendered difficult by the absence of pathognomonic signs and symptoms. In such instances a correct diagnosis may not be made unless the treating physician thinks of it as a possibility. In assessing a patient's exposure history, one must remember that even a stroll in the grass or puttering in the garden may represent risk.

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Acute renal failure

Sudden impairment of the kidney's function, or acute renal failure (ARF), is a highly complex, multifactorial condition of partially unraveled pathophysiology.[1-3] ARF lasts from a few hours to weeks,[1,4-6] resulting in high morbidity and mortality, particularly in the neonate and in the elderly.[3,7-9] While current patient prognosis and kidney survival do not necessarily tally, mortality remains considerably higher in complicated cases (53%) than in those without complications (7.4%).[10] The array of symptoms and signs associated with ARF stems collectively from hypoperfusion or prerenal factors (acute dehydration and shock), from toxic and inflammatory renal parenchymal (glomerulo-tubulo-interstitial-vascular) diseases, and from obstruction of urine flow, or post-renal phenomena. Remarkably, prolonged untreated hypovolemia remains the most frequent cause of parenchymal renal damage (vasomotor nephropathy) in childhood and in old age.

In this issue of the *Journal*,[11] Drs Sweet, Kher, and Makker present a cautious, well organized, and practical diagnostic and therapeutic approach to treating children with ARF. Reviews discussing new developments in the field of ARF are published frequently[1-10,12-14]; they provide useful knowledge, particularly to non-nephrologists, to whom our comments are directed. What reports such as this[11] do not emphasize enough, however, is that primary life support and kidney saving therapy must be initiated as soon as possible by the attending pediatrician. Moreover, further management of potential or actual renal failure varies widely, depending on the etiology. Patients with ARF are often in a precarious condition, requiring expert round-the-clock handling of their medical needs by a multidisciplinary team. In patients experiencing multiple trauma or undergoing major surgical intervention, basic "golden hour" principles facilitate prompt and accurate therapeutic decisions. Evaluation by the nephrologist must be accomplished as soon as, or even before, the diagnosis of ARF is demonstrated. Timely patient transfer to appropriate facilities for ARF management is decisive. A telephone consultation with the nephrologist is most useful to obtain time-saving clinical support before transport and to minimize complications. The more severe the damage sustained by the patient, the more likely the need for an intensive care unit setting and for invasive monitoring procedures. Provided they are not contraindicated, definitive diagnostic and therapeutic interventions should proceed smoothly and rapidly before severe azotemia sets in. Additionally, during management, the alert physician should anticipate complications, avoiding therapeutic zealotry responsible for serious iatrogenic deficits and overcorrections.

Thus, what most patients need is reasonable stabilization without total normalization of weight, acid base equilibrium, and other parameters. Clinical findings are indeed more important than laboratory results. The management of a young child with ARF is simpler and safer if, for example, body weight is maintained below ideal, allowing daily losses of up to about 100 g, a situation experienced by most patients due to obligatory fluid deficits and transient starvation. This situation can be improved by early dialysis or continuous arteriovenous hemofiltration (CAVH).[15] Stable or progressive weight increments may, surreptitiously, expand the circulating volume and precipitate hypertension and cardiovascular decompensation. Maximized caloric intake with appropriate fluid balance should be provided to keep up with the hypercatabolism characteristic of ARF. Although CAVH may present some technical problems in young children, the procedure is practical and life saving in patients with fluid overload. Both dialysis and CAVH allow significant nutritional improvement in severe ARF.

Diminished urinary output (oliguria) and minimal or zero excretion of urine (anuria), once considered absolute diagnostic signs of ARF, are absent in the 30% to 40% of patients who excrete normal or large urine volumes.[12] Other traditional clinical and laboratory findings of ARF depend on the severity and duration of the insult, and may be shown by patients with oliguric vasomotor nephropathy. The only reliable response to a selectively reduced glomerular filtration rate (GFR) is the elevation of serum creatinine, which may be difficult to detect with current techniques to measure this waste product. Concurrent with hypercreatinemia, hyperkalemia, acidosis, and azotemia develop. These abnormalities, significantly influenced by extrarenal GFR-unrelated factors and controllable by the physician, may occur while the serum creatinine level remains normal. Conversely, spuriously elevated serum creatinine due to interference by noncreatinine chromogens, ie, bilirubin, creates factitiously depressed GFR. Biochemical alterations of the urine and urinary sediment pathology bear closer relationship with the damaged nephronal segment. Thus, the diagnosis of vasomotor nephropathy can be clinched by simple determination of the "urinary indices"[13,14] and by microscopic examination of the urine. From this point on, in the absence of significant parenchymal renal disease, definitive corrective measures can be clearly identified and applied.

It is in patients with parenchymal renal disease that the use of kidney biopsy enjoys general acceptance.[16] ARF due to intrinsic, nonstreptococcal, renal disease usually requires histologic confirmation of localization, nature, and degree of lesions. In addition, kidney biopsy allows early action, before lesions become irreversible, and prevents complications and the application of dangerous or useless therapies. In the absence of contraindications, biopsy is performed in most cases of ARF due to parenchymal causes, particularly those with progressive course and/or persistent oligoanuria. In vasomotor and obstructive nephropathy, kidney biopsy lacks specificity and serves no therapeutic purpose. Meaningful prognostic information may be obtained in some cases by a late biopsy.

In summary, most patients with initial hypovolemia or with mild parenchymal inflammatory diseases require only general supportive treatment. Simpler and highly successful therapy can be administered in most individuals without intrinsic renal problems when hypovolemia and obstruction are approached early and aggressively. Low dose dopamine administered soon after onset of ARF may preserve renal blood flow. In seriously ill young children with intrinsic renal disease, serum creatinine and urea of less than 8 mg/dL and 200 mg/dL, respectively, are considered sufficient to institute dialysis; waiting to reach those numbers may even create too great a delay for meaningful therapeutic accomplishments, because other metabolic abnormalities are allowed to occur.

We believe these general "rules" afford the patient more appropriate, timelier, and safer therapy; they should be enforced over and above the minutia of rigid dosages and blood levels to start therapy. In the very young and the old with multiorgan failure (major trauma or surgery-related conditions, in sepsis, consumption coagulopathy, and other medical catastrophes), a much lower intervention threshold is required to treat the disorder efficiently. Early dialysis, the most valuable therapeutic asset for these patients, may be transformed into a serious liability when used late. Similarly, dopamine given initially in ARF, particularly beyond the newborn period, has proven beneficial.[17] Peritoneal dialysis, the preferred technique in the young patient, should be used liberally, since it provides comprehensive, highly effective, and time tested therapy. More specific remedies, however, will undoubtedly be developed in the near future as the understanding of renal failure progresses.

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RESEARCH PAPER

Pitfalls in Tay-Sachs carrier detection: Physician referral patterns and patient ignorance

DEBORAH A. SHAPIRO, BA,* LAWRENCE R. SHAPIRO, MD

ABSTRACT. Tay-Sachs carrier detection testing prior to pregnancy is more desirable than intra-pregnancy testing because it is technically easier, less expensive, and associated with less anxiety and less urgency. However, more than 80% of individuals referred for testing were pregnant women and/or their partners. The literature of the last several years has been silent on these and related issues. A questionnaire designed to determine the factors responsible for these circumstances was sent to 404 physicians (231 obstetrician/gynecologists and 173 internists and family physicians). Of the delivered questionnaires, 49.5% were completed and returned. The majority of obstetrician/gynecologists responding refer patients for Tay-Sachs carrier detection. Internists and family physicians do not, and their responses indicate a lack of pertinent knowledge. In addition, despite physician referral, patients delayed testing until pregnancy. While educational efforts have been targeted to obstetrician/gynecologists, expanded educational activities for internists, family physicians, and patients are required.

(NY State J Med 1989; 89:317–319)

Tay-Sachs disease is a progressive disorder of the central nervous system which is invariably fatal in early childhood, and which occurs mainly in individuals of Eastern European Jewish ancestry. It is inherited in an autosomal recessive fashion, and heterozygotes can be detected on the basis of heat inactivation of hexosaminidase A in serum, tears, leukocytes, and platelets. Tay-Sachs disease can be diagnosed prenatally by means of mid-trimester amniocentesis and first trimester chorionic villus sampling. Because the mode of inheritance and target population are known, and because carriers can be detected and prenatal diagnosis accomplished, carrier detection for Tay-Sachs disease became a model for genetic screening as early as 1972.[1]

New York Medical College/Westchester County Medical Center provides comprehensive medical genetic services to the seven-county region of the lower Hudson Valley (New York State Health Services Area (HSA) No. 6), which comprises approximately two million people and has 25,000 births per year. The area is characterized by urban, suburban, and relatively rural areas. The Tay-Sachs carrier detection program has been in place since 1977. Up to the summer of 1986, when this study was undertaken, 2,424 patients had been screened. In addition to early mass screening activities associated with community educational efforts, the National Tay-Sachs and Allied Diseases Association has sent several mailings to physicians in the region with information pertaining to guidelines for screening and the target population. In addition, several mailings were specifically directed to the obstetricians and gynecologists in the region.

Despite these educational activities, a review of genetics laboratory records from 1981 through the commencement of this study in the summer of 1986 indicated that more than 80% of the individuals tested at Westchester County Medical Center were pregnant women and/or their partners. Nearly all referrals originated from obstetrician/gynecologists when a patient of Jewish ancestry became pregnant; very few referrals were received from internists or family physicians. A review of the literature indicates that physician knowledge and/or patient response to Tay-Sachs carrier detection has not been investigated since 1980.[2-4] In addition, referral patterns in this geographic area, characterized by an affluent and identifiable high risk population, had not been investigated.

With some exceptions,[5] it is the general consensus that testing prior to pregnancy is more desirable because of the time limitations of pregnancy, which result in a sense of urgency and anxiety. Moreover, while testing prior to pregnancy can be accomplished by means of a serum assay in both males and females, the technically more difficult and expensive leukocyte, tear, or platelet assays must be used in pregnant females, because the serum assay gives rise to false positive results during pregnancy.[6]

This study was undertaken to determine the factors responsible for the undesirable delay in testing until pregnancy, and to formulate recommendations to correct this problem.

PATIENTS AND METHODS

A two-page questionnaire was prepared consisting of nine objective questions. The questions were designed to determine whether or not a physician referred patients for Tay-Sachs carri-

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* Class of 1989 New York Medical College.

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TABLE I. Physician Responses

Referral of Patients (181 Responses)	Obstetrician/Gynecologist* (N = 89)	Specialty Internist/Family Physician* (N = 27)	Not Identified (N = 65)
Refer	77.5% (69)	11.1% (3)	60.0% (39)
Do not refer	18% (16)	88.9% (24)	40.0% (26)
Incomplete response	4.5% (4)	0	0

Frequency of Referral (103 Responses)	Total	Obstetrician/Gynecologist	Specialty Internist/Family Physician	Not Identified
Seldom	27% (30)	13	2	15
Occasionally	42.3% (45)	34	1	10
Frequently	26.1% (28)	22	1	5

Reasons for Not Referring (66 Responses)	Total	Obstetrician/Gynecologist	Specialty Internist/Family Physician	Not Identified
Unaware of availability	21.2% (14)	6	5	3
Not part of routine	24.2% (16)	3	5	8
Do not deal with target population	60.6% (40)	8	12	20
Cannot identify target population	59.1% (39)	10	14	15
Disagree with testing	0	0	0	0
Other	7.6% (5)	0	4	1

* *P* <0.001 by chi-square test indicating significant difference in referral pattern.

er testing, to determine the reasons why a physician tended not to refer patients, to assess the frequency and object of each physician's referrals, to explore the reasons why referral was delayed until pregnancy, and to determine if a physician had a method for identifying patients who should be screened. Two questions evaluated the effect of prior mailings to physicians concerning Tay-Sachs carrier detection and the knowledge derived therefrom. Two other questions requested permission for a brief telephone and/or personal interview, as well as identifying information if such permission were granted.

A total of 404 questionnaires were mailed to 231 obstetrician/gynecologists and 173 internists and family practitioners in the seven-county lower Hudson Valley region (HSA No. 6). An established computerized mailing list of all of the obstetrician/gynecologists in the region was used and a list of internists and family physicians was compiled from the listings in the telephone directory (Yellow Pages) of Dutchess, Orange, Putnam, Rockland, Sullivan, Ulster, and Westchester counties. A second mailing was sent four weeks after the first to all physicians except those who voluntarily identified themselves on the returned questionnaires (physicians were instructed to disregard the second questionnaire if they had already completed and returned one).

The results were tabulated by determining the percentage of physicians who selected a particular answer. Attempts were made to contact by telephone those physicians who agreed to follow-up interviews (approximately 60 phone calls were made), but in each case an answering service or office employee indicated that the physician was not available.

RESULTS

Of the 404 questionnaires mailed, 38 were not deliverable or were otherwise invalid. Of the 366 questionnaires that were delivered, 49.5% (181) were completed and returned after two mailings. Forty-nine percent of responses were from obstetrician/gynecologists, 15% from internists/family physicians, and 36% from physicians whose specialty was not indicated on the questionnaire.

Of the total responses, 61.3% of physicians (111) indicated that they did refer patients for Tay-Sachs carrier detection and 36.5% (66) indicated that they did not. The results of physician responses regarding referral of patients are presented in Table I. As shown in the table, the percentage of obstetrician/gynecologists who referred is significantly greater than the percentage of internists/family physicians who referred ($p < 0.001$ by chi-square test). Most physicians indicated they made "occasional" referrals, with an approximately equal portion divided between "frequent" and "seldom." The reasons for not referring are also presented in Table I. Of physicians who did not refer, none disagreed with the value of Tay-Sachs carrier detection. However, the majority indicated that they did not deal with the target population, and 59.1% could not identify the target population.

Table II presents the characteristics of the patients referred for testing, the majority of whom were pregnant. Table III presents the reasons for predominance of pregnancy-related referrals, indicating that most physicians did not defer referrals until pregnancy but that patients delayed testing until pregnant. Table IV presents physician referral guidelines and shows that more physicians use their own guidelines.

DISCUSSION

We conclude that the majority of obstetrician/gynecologists (77.5%) refer patients for Tay-Sachs carrier detection, while the great majority of internists/family physicians (88.9%) do not. Of those physicians whose specialty could not be identified, 60.0% did refer patients. It is possible that this group comprises mainly obstetrician/gynecologists, in view of the fact that 49.2% of those physicians who refer identified themselves as obstetrician/gynecologists. The frequency of referral is difficult to interpret since the physician who refers frequently may have a larger proportion of the target population in his or her practice

TABLE II. Characteristics of Patients Referred for Testing (N = 111)

Pregnancy Related	70.3% (78)
Couples	49.5% (55)
Pregnant woman	39.6% (44)
Father of fetus	49.5% (55)
Relatives of couple	2.7% (3)
Unrelated to Pregnancy	26.1% (29)
Couples	41.4% (46)
Single females	29.7% (33)
Single males	5.4% (6)

TABLE III. Reasons for Pregnancy-Related Referral (N = 111)

Reason	Percentage of Responses Received (N)
Patients delay until pregnant	66.6 (74)
Pregnancy is first contact with physician	12.6 (14)
Physician does not consider screening until pregnancy	8.0 (9)
Physician considers pregnancy best time for testing	2.7 (3)
Belief that pregnancy does not affect testing	0 0

* Not all physicians responded to every question.

as compared to the physician who seldom refers. When one reviews the reasons for not referring for Tay-Sachs carrier detection (Table I), 60.6% of the physicians reported that they do not deal with the target population; however, more than half indicated that they could not identify the target population. This suggests that those physicians who believe that they do not deal with the target population may not recognize members of this group in their practices. In addition, 21.2% of the physicians who did not refer were unaware of the availability of testing. The inability to identify the target population, combined with a lack of awareness of the availability of testing, indicates a lack of knowledge on the part of non-referring physicians. Since 88.9% of non-referring physicians are internists/family physicians, it is apparent that educational efforts need to be directed to this group.

TABLE IV. Physician Referral Guidelines (N = 111)

	Yes	No
Physicians aware of referral guidelines	49.5% (55)	42.3% (47)
Physicians have own referral guidelines	62.2% (69)	34.2% (38)
For patients of Jewish ancestry	36.9% (41)	—
For patients with family history of Tay-Sachs disease	15.3% (17)	—

In 66.6% of the responses, the physician indicated that either the patient was noncompliant and delayed testing until pregnant, or the physician was first consulted in connection with the patient's pregnancy (12.6%). In addition, the fact that only 5.4% of physicians referred unmarried males for testing (Table II) indicates that internists/family physicians do not refer for Tay-Sachs testing. The lack of referral for Tay-Sachs carrier detection prior to marriage and/or pregnancy by internists and family physicians, combined with patient ignorance, result in testing being done during pregnancy in more than 80% of the cases.

Recommendations for physician education with regard to Tay-Sachs disease were made in 1974,[2] 1978,[3] and 1980.[4] While educational efforts for obstetrician/gynecologists have apparently been successful, these educational efforts now must be directed to internists and family physicians, so that Tay-Sachs carrier detection can have a broader base and be accomplished prior to pregnancy. In addition, continued and expanded educational efforts for the patient and community would probably result in more patients being tested prior to pregnancy.

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Selected tickborne infections

A review of Lyme disease, Rocky Mountain spotted fever, and babesiosis

MAHFOUZ H. ZAKI, MD, DRPH

Ticks can be distinguished from insects by three main features. First, the head, thorax, and abdomen are all fused together; second, the nymph and adult stages have four pairs of legs; and third, they have no antennae. There are two basic types of ticks, the *Ixodidae*, or hard ticks, and the *Argasidae*, or soft ticks.

Ticks are among the most efficient vectors of disease in man and animals. They are persistent blood suckers, attach firmly to the skin, feed slowly, and may go unnoticed for a long period. Their wide host range includes domestic and wild animals, which ensures ample opportunities to acquire and transmit infection. They have few natural enemies, and they possess a dorsal shield or scutum which makes them resistant to environmental stress. They have a high reproductive ability, with the females of certain species capable of laying thousands of eggs. Some species transmit the pathogens through the eggs (transovarial transmission) or from larvae to nymphs or nymphs to adults (transstadial transmission). Furthermore, ticks can live for two or three years, which increases the chances of acquiring and transmitting an infection.[1]

Human infections transmitted by ticks include viral, bacterial, and protozoan pathogens. Viral infections include Colorado tick fever, Powassan encephalitis, Russian spring-summer encephalitis, louping ill, and others caused by several classified and unclassified viruses.

Bacterial infections include Rocky Mountain spotted fever, Lyme disease, Q fever, relapsing fever, and tularemia. A few protozoan infections common to animals, such as babesiosis, are accidentally transmitted by ticks to humans.

This report is a review of selected human tickborne infections, their clinical features, modes of transmission, diagnostic methods, treatment regimens, and methods of prevention and control.

LYME DISEASE

Lyme disease, the most prevalent arthropod-borne infection in the United States, is a multisystemic inflammatory disease that is caused by the spirochete *Borrelia burgdorferi* and is usually transmitted by the bite of an infected deer tick, *Ixodes dammini* (Fig 1). On the West Coast of the United States, the black legged tick *Ixodes pacificus* has been incriminated as the vector of transmission.[2]

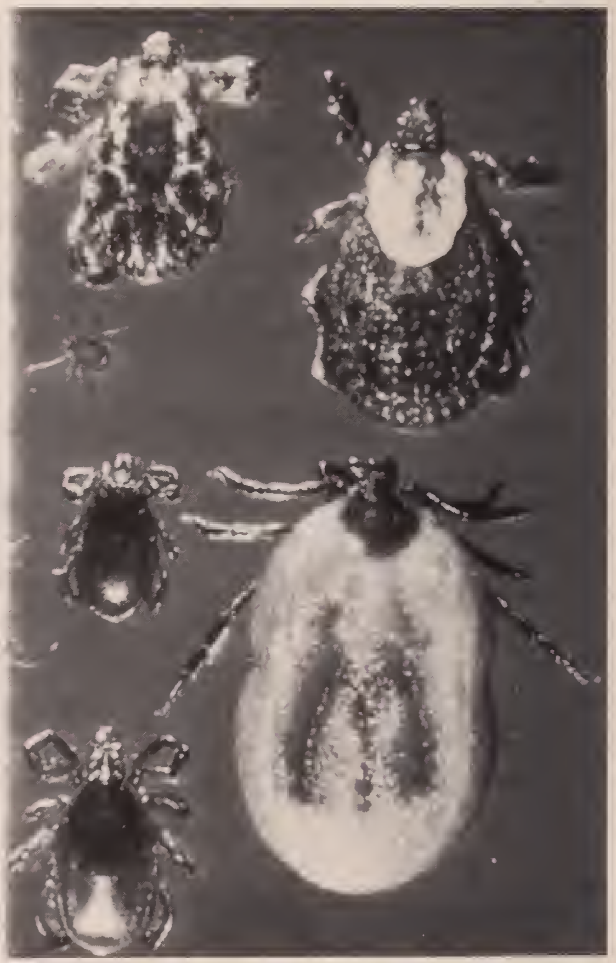

FIGURE 1. *Ixodes dammini* and *Dermacentor variabilis*. Left column (from top to bottom): male *D variabilis;* larva *I dammini;* male *I dammini;* female *I dammini*. Right column (from top to bottom): female *D variabilis;* female *D variabilis* (engorged) (courtesy of Dr Edward M. Bosler).

The pathognomonic lesion of Lyme disease is a skin lesion called erythema chronicum migrans (ECM). This characteristic lesion was described by Afzelius[3] in Sweden in 1909. In 1955, Binder et al[4] demonstrated that erythema chronicum migrans was infectious in nature.

In 1970, Scrimenti[5] reported the first case of erythema

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FIGURE 2. Erythema migrans in Lyme disease (courtesy of Dr Bernard W. Berger).

migrans in Wisconsin. In 1977, Steere et al[6] reported an epidemic of oligoarticular arthritis in children and adults in three Connecticut communities and suggested transmission by an arthropod vector.

In 1982, Burgdorfer et al[7] detected a treponema-like spirochete in an adult *Ixodes dammini*. Samples of serum from patients with Lyme disease were shown to contain antibodies to this spirochete. The authors suggested that the newly discovered spirochete might be "involved in the etiology of Lyme disease."

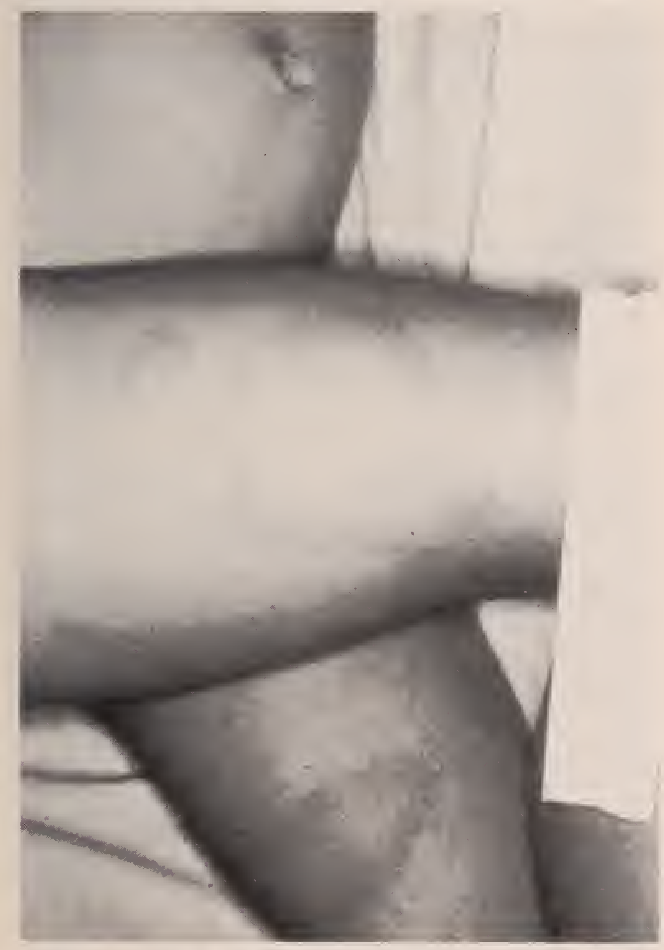

FIGURE 3. Erythema migrans in Lyme disease.

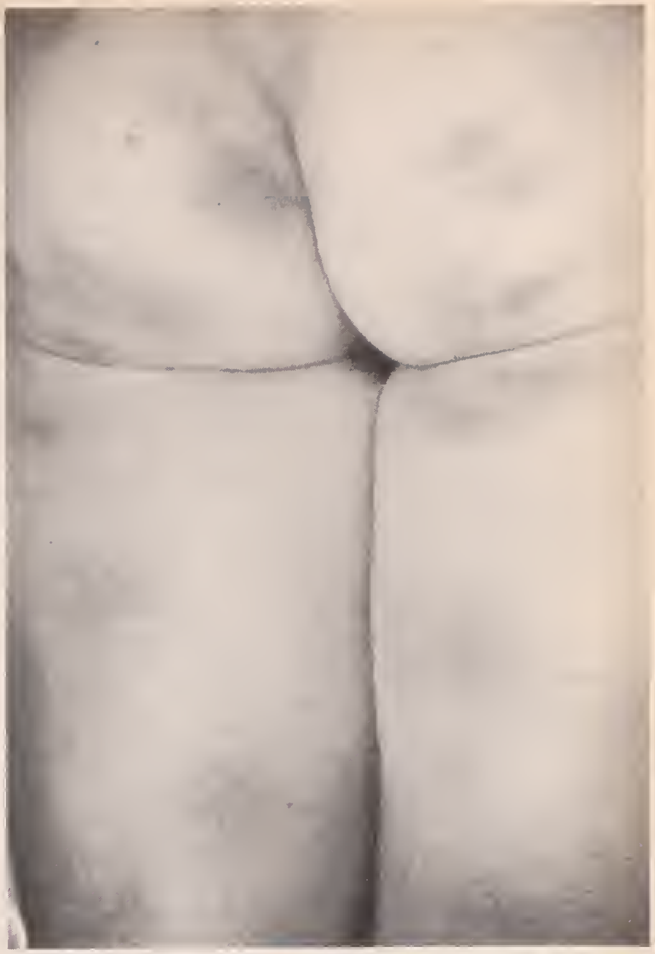

FIGURE 4. Erythema migrans in Lyme disease (courtesy of Dr Bernard W. Berger).

In 1983, Steere et al[8] recovered spirochetes from the blood, skin lesions, and cerebrospinal fluid of three of 56 patients with Lyme disease and from the gut of 21 of 110 nymphal or adult *Ixodes dammini*. These isolates and the original one isolated by Burgdorfer had the same morphologic and immunologic features.

During the same year, Benach et al[9] isolated spirochetes from the blood of two of 36 patients who had signs suggestive of Lyme disease. The authors alluded to the likelihood that the isolated spirochete is the etiologic agent for one or more of the clinical manifestations of Lyme disease.

The erythema chronicum migrans, recently called erythema migrans (EM), appears a few days to a few weeks after a tick bite, initially as a red macule or papule which expands to form a large ring several inches in diameter (Fig 2). The ring may be uniformly red or may show central clearing. In some instances it may take on the appearance of a bull's eye. Skin lesions may be multiple, with secondary ones appearing a few days after the initial one (Figs 3, 4). In a group of 24 patients reported by Steere et al,[10] the skin lesions lasted about three weeks. Infection with *Borrelia burgdorferi* does not always result in the appearance of the skin eruption. About 10% to 30% of patients do not have the initial pathognomonic rash.

During the early stages, a few patients may be asymptomatic, while others may experience malaise, fever, fatigue, headache, nausea, vomiting, backache, sore throat, and lymphadenopathy.

If not treated properly at an early stage, patients with Lyme disease may develop serious neurologic, cardiac, or arthritic complications.

Neurologic complications may occur four to six weeks after infection in 15–25% of patients and include aseptic meningitis, encephalitis, cranial nerve palsies, particularly Bell's palsy, and peripheral neuropathy. Of a group of 36 patients with late Lyme disease studied by Halperin et al,[11] 13, or 36%, had neurophysical evidence of peripheral neuropathy.

Cardiac manifestations, which occur in less than 7% of cases, include myocardial conduction abnormalities (atrioventricular node and bundle branch) and, on rare occasions, myocarditis.

As the disease progresses, joint complications occur in about 50% of patients. In the first cluster of 51 patients with arthritis investigated by Steere et al[6] in Connecticut, 69% were monoarticular, 29% oligoarticular, and 2% polyarticular. The large joints, especially the knee joints, are the ones most often affected (84%). Other joints that may be involved are the ankle, wrist, and temporomandibular. Joint manifestations are usually unilateral or asymmetrical and often move from one joint to the other. The duration of the first attack varies from one week to six months. A certain percentage of patients continue to complain of arthralgia several months after the initial attack.

Lyme Disease and Pregnancy Outcomes. Transplacental transmission of *Borrelia burgdorferi* has been documented in a woman in whom Lyme disease developed during the first trimester of pregnancy. The infant died of congenital heart disease during the first week of life.[12] In 1985, the Centers for Disease Control (CDC) reported that of 19 women who were suffering from Lyme disease during pregnancy, five had adverse fetal outcomes. Since the adverse outcomes differed in nature, no conclusion could be drawn about a cause and effect relationship.[13]

Williams and coworkers[14] compared sera of 281 infants born in endemic areas with those of 181 infants born in control, nonendemic areas. Twenty-six or 10.2% of serum samples from the endemic areas had detectable antibodies to *Borrelia burgdorferi* as compared with four or 2.4% in control areas. Testing was performed by the immunofluorescent antibody (IFA) and the enzyme-linked immunosorbent assay (ELISA) tests. Six newborns had significant antibody levels (IFA 1 : 64 or greater or ELISA 0.20 or greater). Of these, five newborns were from endemic areas. Of the 282 newborns from endemic areas, 25 (8.9%) had codifiable malformations, as compared with 20 (11.0%) of the 181 newborns from the control areas. The authors concluded that no association between the presence of antibodies to *Borrelia burgdorferi* and the occurrence of congenital anomalies could be demonstrated. Babies with detectable antibodies tended to be of a slightly lower birth weight, small for gestational age, and had some degree of neonatal jaundice. However, the observed differences were insignificant.[14]

The lack of a strong association between Lyme disease during pregnancy and fetal anomalies does not mean that no precautionary measures should be taken. As a matter of fact, pregnant women who reside in endemic areas should take all necessary measures to avoid exposure to ticks. In the event of unavoidable exposure, it is important that pregnant women know how to recognize the early signs and symptoms of Lyme disease in order to get proper and prompt medical care.

Long term, well-controlled studies are urgently needed to determine the risk to the fetus if the pregnant woman acquires the *Borrelia* infection during pregnancy.

Lyme Disease and Blood Transfusion. It is a well-known fact that *Borrelia burgdorferi* are difficult to culture from blood, which strongly suggests that the organism does not have a prolonged blood stage. This minimizes the risk of transmission by transfusion. There have been no published reports of Lyme disease being acquired by transfusion of blood or blood products. On a worldwide basis, thousands of cases of Lyme disease have been reported in the last five years, and not one case has been documented to have been contracted through blood transfusions. It is thus thought that this mode of transmission does not play a significant role in Lyme disease. Current blood banking procedures designed to exclude obviously sick patients from donating blood are sufficient to prevent the unlikely event of transfusion acquired Lyme disease.

Diagnosis. Lyme disease should be considered in the differential diagnosis in a patient from an endemic area, or a patient who had been to an endemic area and who presents with a skin lesion simulating erythema chronicum migrans with or without a history of a tick bite. It should be realized that more than one half of the patients with confirmed Lyme disease give no history of a tick bite. Later in the disease process, organ involvement should help the physician in suspecting *Borrelia* infection.

A definitive diagnosis of Lyme disease can be made by the isolation and culture of *Borrelia burgdorferi* from blood or biological specimens. The process of isolating the spirochete, however, is difficult and the yield is very low. Reliance has to be placed on the clinical manifestations of the disease and the immunogenic response to infection.

As in many infectious diseases, specific IgM antibodies appear early in the disease process, followed by specific IgG and IgA antibodies during the second or third month of infection, which may remain for years. Serologic testing in Lyme disease is handicapped for several reasons. Many individuals harbor spirochetes as part of their normal flora and can give positive results against some common antigens. In addition, the early administration of antibiotics often delays or aborts the development of recognizable antibodies for several months.

The serologic tests commonly used include the IFA and the ELISA. In a study by Russell et al,[15] the sensitivity and specificity of the IFA and ELISA tests were determined in sera from patients with Lyme disease, patients with other treponemal infections, and healthy individuals. Sera from patients with syphilis, yaws, and pinta reacted with the *Borrelia burgdorferi* antigens. When the patients with treponemal infections were excluded, both tests were highly specific—97% for IFA and 100% for ELISA. In the authors' experience, both tests were highly sensitive and specific in complicated Lyme disease but were insensitive in the erythema migrans stage.

In the experience of Craft et al,[16] the ELISA test was more sensitive and specific than the IFA test. In a study by

Berardi et al,[17] the antibody capture immunoassay was capable of detecting elevated specific IgM in 67% of patients with erythema migrans and in 93% of patients during convalescence. In a study by Volkman,[18] using highly sensitive Western blots, more than 50% of a control population with no history of tick bites or ECM and with a negative specific ELISA had antibodies against a flagellar antigen (41KD).

Grodzicki and Steere[19] compared the indirect ELISA with immunoblotting and, in their experience, immunoblotting was superior to the indirect ELISA for diagnosing early Lyme disease.

Coleman and Benach[20] used flagellin-enriched fraction as well as eluted polypeptides as antigen in the ELISA test and, in their experience, this antigen was superior to the standard ELISA antigen.

Hyde and coworkers[21] found that at least three of the major antigenic components of the spirochete can be detected in the urine of mice and humans using monoclonal antibodies. This technique, however, has to be evaluated in controlled trials before it can be accepted as a reliable diagnostic tool.

The aforementioned studies demonstrate the futility of depending solely on the serologic tests for diagnosis in early Lyme disease. The physician has to rely on the history of exposure in an endemic area, the presence of skin lesions, and the clinical manifestations, and, if possible, must complement these findings with appropriate serologic studies. It should be emphasized that some patients who have neurologic manifestations but do not demonstrate high antibody titers in their sera often have diagnostic antibody levels in their cerebrospinal fluid. Although attempts are currently being made to improve the sensitivity and specificity of the serologic tests, physicians must keep in mind the limitations of the existing tests.

One of the problems often encountered in the diagnosis of late Lyme borreliosis occurs as a result of early administration of ineffective antibiotic therapy resulting in seronegativity. In some of those patients, the presence of a high level of spontaneous suppressor cell activity was found to be an effective tool in diagnosis.[22] These tests, however, are not done routinely as part of the internal medicine practice but are rather of research interest at present.

Treatment. Almost four decades ago, penicillin was used for the treatment of erythema chronicum migrans and acrodermatitis chronica atrophicans in Sweden.[23] Since the recognition of Lyme disease as a medical entity, several antibiotics have been used for treatment with varying degrees of success. Unfortunately, there is no consensus at present on the optimum antibiotic for early or late Lyme disease or on the duration of therapy.

Evaluation of therapeutic trials in Lyme borreliosis has faced several obstacles. These include the difficulty encountered in diagnosing early cases in the absence of ECM or a history of a tick bite, the lack of definitive criteria of cure, and the difficulty in determining whether the persistent sequelae following treatment represent treatment failures or are a result of the host response to infection.

With the identification and isolation of *Borrelia burgdorferi*, in vitro sensitivity studies were performed by several investigators. However, because of the lack of uniformity in the methodology used and the difficulty in isolating the spirochetes from biologic specimens, reliable comparisons between the various antibiotics were not possible.

Early Lyme disease. In the late 1970s and early 1980s, Steere and coworkers[24] demonstrated that penicillin and tetracycline were more effective in the treatment of erythema chronicum migrans than erythromycin. Ironically, in vitro studies showed that *Borrelia burgdorferi* was very sensitive to erythromycin. In the authors' experience, 250 mg orally four times a day for ten days decreased the resolution time of ECM and reduced the incidence of later complications. Of the 40 patients treated with penicillin, complications developed in three, as compared with four of 29 treated with erythromycin and none of 28 treated with tetracycline. Late complications included recurrent attacks of arthritis, meningoencephalitis, and myocarditis. A few patients had to be retreated with the same regimens.

Among the antibiotics that have been successfully used in early Lyme disease are amoxicillin, 500 mg–1 g orally three times a day for 21 days with or without probenecid, 500 mg with each dose of amoxicillin. Amoxicillin is preferred to ampicillin because it is more completely absorbed than ampicillin, thus producing higher serum levels for an equivalent dose and allowing a three times/day rather than four times/day administration. Doxycycline in a 100 mg dose orally twice a day has also been used in early Lyme disease. It is often preferred to tetracycline because it is administered twice daily instead of four times a day, with limited gastrointestinal discomfort.

Late Lyme disease. In a controlled therapeutic trial by Steere and coworkers,[25] benzathine penicillin was administered to 20 patients with established Lyme arthritis in a dose of 2.4 million U intramuscularly weekly for three weeks. Another group of 20 patients was given saline and used as controls. Seven of the 20 treated patients had complete resolution of the arthritis (35%) and remained well for a follow-up period of 33 months. None of the control group showed any improvement. In another group of 20 patients, 20 million U of intravenous penicillin G was administered daily for ten days. Of the 20 patients, 11 had complete resolution (55%). This study showed that high doses of penicillin do not necessarily result in a better response.

One of the antibiotics found to be effective in late Lyme disease is the aminothiazolyl cephalosporin, ceftriaxone. In a controlled therapeutic trial by Dattwyler and coworkers,[26] 23 patients with ECM and evidence of involvement of two or more systems (central or peripheral nervous system, cardiovascular, or musculoskeletal systems) were divided at random into two groups. One group of 13 patients received ceftriaxone, 2 g intravenously every 12 hours for 14 days, and the other group of ten patients was given penicillin G, 4 million U every four hours intravenously for ten days. In the authors' experience, ceftriaxone was significantly more effective than penicillin in the treatment of late Lyme disease. The authors, however, found that in those patients whose arthritis did not respond to ceftriaxone, a three-month therapy with amoxicillin and probenecid was helpful. These preliminary findings have to be tested in well-controlled therapeutic trials with long follow-up periods.

During pregnancy. Phenoxymethyl penicillin (penicillin V), 250 to 500 mg orally four times/day, is considered by some investigators as the drug of choice for pregnant and lactating women and for children with deciduous dentition.[27]

For adults allergic to tetracycline or penicillin, erythromycin, 250 to 500 mg orally four times/day for ten to 21 days, although less effective, is sometimes used.[27] For children allergic to penicillin, erythromycin, 30 mg/kg per day orally in divided doses for ten to 21 days, can be used.[27]

A few patients may not respond to the first treatment regimen and may have to be retreated. Berger[28] evaluated the efficacy of antibiotic treatment of 161 patients with erythema chronicum migrans. The antibiotics used were tetracycline or penicillin alone or in combination with probenecid. Two of 80 patients with a minor illness and 17 of 81 patients with more serious disease required retreatment.

It may be helpful in the selection of the most appropriate treatment regimen to realize that *Borrelia burgdorferi* is similar in many respects to *Treponema pallidum*, which requires a high concentration of the antibiotic over a long period to achieve bactericidal effect.[29] Physicians treating patients with Lyme disease should keep this in mind even in the early stages because of the possibility of early dissemination. In these situations, physicians should select the antibiotic that is sustained at a high concentration in both body fluids and tissues over a long period.

Treatment regimens have been arbitrarily administered over periods ranging from seven to 30 days. Long term, controlled therapeutic trials are urgently needed to determine the optimum duration of therapy in the various stages of the disease process.

Epidemiology. The reporting of the characteristic skin lesion, erythema chronicum migrans, in a patient in Wisconsin in 1970[5] and the outbreak of arthritis among those exposed to ticks in 1975 stimulated research to determine the etiology and clinical features of the new syndrome.[6] Research in this country led to the identification of a spirochete in a tick suspected of transmission in 1982, and the spirochetal etiology was proposed.[7] A year later, *Borrelia burgdorferi* were isolated from blood and other biological specimens.[8,9]

On a worldwide basis, the disease has been reported from many European areas, including Scandinavia, England, France, Italy, Central Europe, Spain, Portugal, and the Balkans. The disease has also been reported from the European part of the Soviet Union, from North Africa, and Iran.[30]

In an attempt to estimate the prevalence of the disease in this country and for surveillance purposes, the Centers for Disease Control (CDC) established diagnostic criteria in 1982. These relied mainly on the presence of erythema chronicum migrans. With the development of specific tests for *Borrelia burgdorferi* in 1984, the criteria incorporated, in addition, the results of laboratory testing. Cases were also classified according to whether the patient acquired the infection in an endemic area or in a nonendemic area as follows:[31]

Case Definition for Lyme Disease
I. Exposure in an endemic area.
  A. Erythema chronicum migrans (ECM) with exposure in endemic area 30 days before onset of ECM.
  B. In the absence of ECM, involvement of at least one organ system (cardiac, neurologic, and arthritic) and a positive laboratory test for *Borrelia burgdorferi.*
II. No exposure in an endemic area.
  A. ECM with involvement of at least two organ systems.
  B. ECM and a positive laboratory test.

An area is designated as endemic if *Borrelia burgdorferi* are present and can be transmitted by an appropriate vector, which is usually *Ixodes dammini.* This designation also helps to explore the role played by other vectors in transmission.

More than 7,000 cases were reported to the CDC by the end of 1987 (personal communication, CDC, 1989). The number of states reporting Lyme disease in this country increased from 11 in 1980 to 43 in 1989. Most of the reported cases, however, were acquired in seven states: New York, New Jersey, Connecticut, Massachusetts, Rhode Island, Wisconsin, and Minnesota.[31] Endemic areas will most probably expand due to the introduction of the *Ixodes* ticks in non-infested areas, infection of the ticks in previously non-infected areas, and better recognition of the disease.

The number of cases reported to CDC has shown a marked rise over the past few years. Whether this increase in reporting is totally a result of a true increase in incidence or is due in part to the augmented awareness of the disease is conjectural at present. These surveillance statistics do not reflect the actual incidence of the disease for several reasons. Variations in the case definition, the reporting mechanism, the scrutiny in diagnostic verification, media coverage, and professional interest in the disease, are all factors that affect the incidence data. This does not mean that the CDC data serve no useful purpose. As a matter of fact, they are excellent indicators for the demonstration of trends and the geographic extension of the disease and for the delineation of the role of other arthropods in transmission.

In New York State, the number of cases doubled in successive years between 1981 and 1984 (Fig 5). Laboratory testing during that period was performed only by the New York State Department of Health Unit at Stony Brook. Due to the increased demand for this laboratory service, the State Health Department relinquished this responsibility in 1985 to local laboratories, which resulted in sporadic and fragmentary reporting in the following two years.[32] This explains the decline in the number of reported cases between 1984 and 1986 (Fig 5). On January 1, 1986, Lyme disease reporting became mandatory in New York State. The following definitions have been adopted by the State Health Department:

Case definitions:
I. A confirmed case is defined as one of the following:

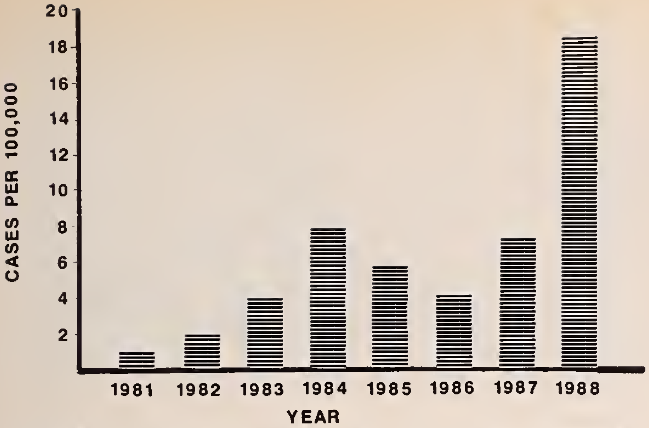

FIGURE 5. Lyme disease case rate per 100,000, New York State excluding New York City, 1981–1988 (adapted from reference no. 32).

A. Culture positive or tissue identification of *Borrelia burgdorferi*.
B. A four-fold titer rise to Lyme-specific antibodies in acute and convalescent sera drawn at least two weeks apart.
C. A person with a history of exposure in an endemic county and
  1. Erythema migrans (EM), or
  2. Clinically compatible late manifestations and positive serology.

II. A presumptive case is defined as:
  A. A person with no history of exposure in an endemic county and
    1. EM, or
    2. Clinically compatible late manifestations and positive serology.
  (Note: On determination that a county is endemic, all presumptive cases with exposure in that county in the previous 12 months will be reassigned to confirmed cases.)

III. An unconfirmed case is defined as a reported case that does not meet the above criteria.
IV. Pending: investigation not complete.

Since reporting became mandatory there has been a sharp increase in the number of reported cases from endemic areas, and new cases have been reported from nonendemic areas.

At present, Lyme disease is endemic in six counties in New York State: Suffolk, Westchester, Nassau, Putnam, Dutchess, and Ulster (Fig 6). Most of the cases, however, occur in Suffolk and Westchester counties. There is no doubt that the media coverage that surrounded Lyme disease during the past two years is responsible to some extent for the increase in reporting. At the same time, one has to assume that many cases are not reported because of the difficulty in obtaining a positive serologic finding in the early stages, the vague symptoms in some patients and the lack of skin eruptions, inadequate reporting by physicians, and the reluctance of many physicians to fill out the lengthy and rather complicated form required by the State Health Department.

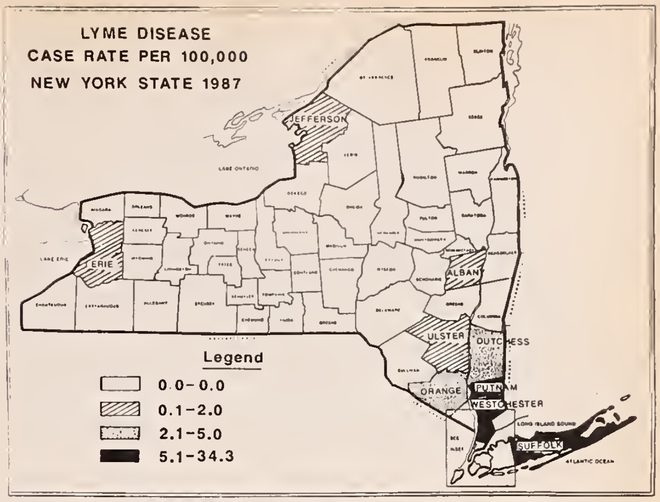

FIGURE 6. Lyme disease case rate per 100,000, New York State, 1987 (adapted from reference no. 33).

In order to improve the reporting of Lyme disease both quantitatively and qualitatively, the Suffolk County Department of Health Services assigned a nurse-epidemiologist to visit the physicians who treat patients with Lyme disease to abstract their records and report those patients who meet the criteria of a definitive case.

Within each endemic county, the disease is usually concentrated in certain foci where the agent, the vector, the animal hosts, and humans interact. In Suffolk County the incidence is highest in the east end of the county, especially on the south shore (Fig 7). Incidence rates vary from a low of 14 per 100,000 in the Town of Babylon to a high of 2,130 per 100,000 in Shelter Island (Fig 8).

The extremely high incidence rates in the towns of Shelter Island, East Hampton, and Southampton can be attributed to an abundance of animal hosts, high infestation rates, high infection rates, and excessive chances of human exposure. The number of Lyme disease cases rose sharply between 1986, the first year of mandated reporting, and 1988, partly because of the implementation of an active case finding program.

Age and sex. The disease affects all age groups in Suffolk County, especially persons below 50 years of age (Fig 9). This age distribution may be slightly different from the early experience in Westchester County, where most of those affected were of a younger age group. The disease was equally distributed between males and females during 1986 and 1987 (Fig 9). During 1988 there was a slight preponderance of females, with a male/female ra-

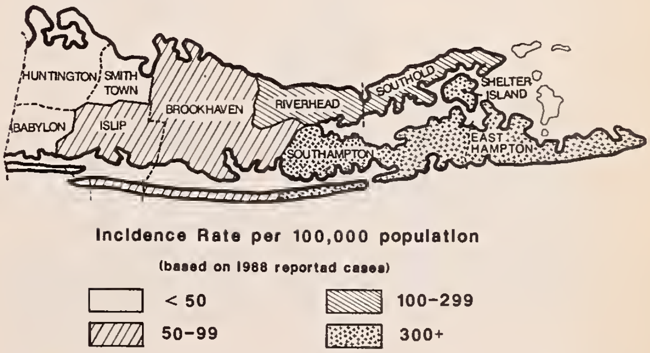

FIGURE 7. The incidence of Lyme disease in Suffolk County.

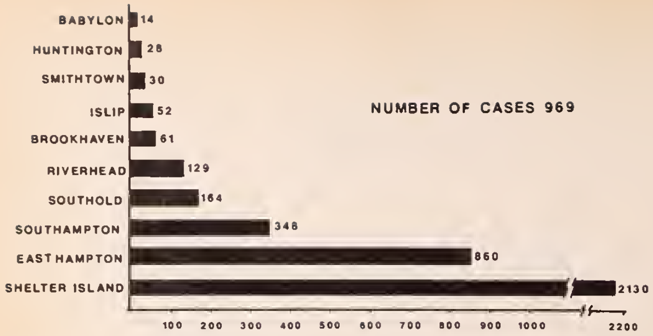

FIGURE 8. Lyme disease incidence rates by township, Suffolk County, 1988.

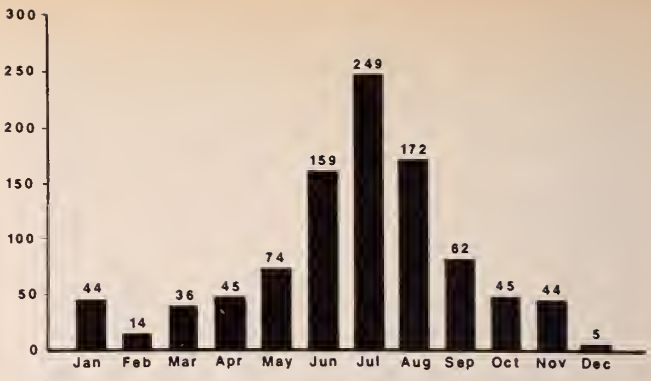

FIGURE 10. Lyme disease cases by date of onset, Suffolk County, 1988.

tio of 0.9. Other endemic areas have reported higher male/female ratios; 1.9 in New Jersey[33] and 1.2 in Westchester County.[34]

Seasonal distribution. Most of the cases (76%) occur between the months of May and September, a period of activity of the vector transmitter, outdoor living, and human exposure to the vector (Fig 10).

Prevalence of Borrelia burgdorferi *infection.* Field studies have demonstrated high infection rates of the *Ixodes dammini* in many endemic areas. Between 10% and 35% of the ticks examined from Connecticut were infected,[35] and more than 50% of those from Shelter Island, New York.[36]

Bosler et al[37] reported on a few studies that show high infection rates in canines and horses. Between 34% and 42% of asymptomatic dogs had antibodies against *Borrelia burgdorferi*. In another study, 24% of horses from a New England endemic area had evidence of infection.[38]

Seroprevalence surveys indicate a relatively high infection rate in humans in endemic areas. In a study by Graham et al[39] of 2,044 Suffolk County employees who might have been exposed to ticks in their routine daily activities, 143 or 7.0% had positive titers by the IFA and ELISA tests. In another survey by Hanrahan et al[40] on Fire Island, New York, 6.8% had evidence of infection.

Benach and coworkers[41] examined 41 serum samples from patients with confirmed babesiosis for antibodies to *Borrelia burgdorferi*. Twenty-three (56%) had titers of IgG antibodies to the spirochete ≥1:128 and 14 (34%) sera had IgM antibodies ≥1:128. The authors also examined 13 pairs of sera from patients with Lyme disease from areas endemic for both diseases and found that six patients had antibodies to *Babesia microti*. The authors did not attribute their findings to antigenic cross reactions but raised the possibility of simultaneous exposure.[41]

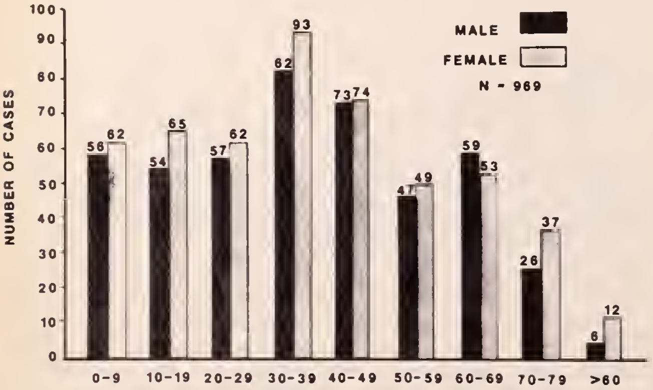

FIGURE 9. Lyme disease cases by age and sex, Suffolk County, 1988.

Lastavica and coworkers[42] recently described a focal epidemic of Lyme disease which spread from a nature preserve to an adjacent community in coastal Massachusetts, whereby 35% of the 190 residents became infected with *Borrelia burgdorferi* between 1980 and 1987.

It thus seems that in heavily endemic areas, high infection rates can be expected in the tick population, domestic animals, and humans, and that infection can spread to neighboring communities.

ROCKY MOUNTAIN SPOTTED FEVER

This disease, which is caused by *Rickettsia rickettsii*, is transmitted by the bite of an infected tick. It usually takes a few hours of attachment of the tick before the rickettsiae become reactivated to infect humans.

In the eastern and southern United States, the common vector is the American dog tick, *Dermacentor variabilis*. In the southwestern states, the lone star tick, *Amblyomma americanum*, is the vector responsible for transmission.

Following an incubation period of three to 14 days, the patient experiences a sudden onset of fever, malaise, headache, chills, and conjunctival injection. Many patients, however, may have atypical symptoms such as nausea, vomiting, abdominal pain, diarrhea, joint pains, or stiff neck. Two to three days later, a maculopapular rash appears first on the wrists and ankles and extends to cover the extremities and the rest of the body (Fig 11). One characteristic feature of the rash is that it often covers the palms and soles. In severe cases the rash may become petechial, confluent, or largely hemorrhagic (Figs 12, 13). In some patients, the rash may not progress beyond the maculopapular stage and in others, it may not appear until late in the disease process.

Rocky Mountain spotted fever (RMSF) may present in an unusual fulminant viscerotrophic variety with delayed or absent cutaneous manifestations. Davis and Bradford[43] reported two cases of RMSF, one in an eight-year-old girl and one in an 18-year-old man, whose main complaints were fever and abdominal pain, especially in the right lower quadrant simulating appendicitis. The girl had, in addition, a petechial rash on her lower arms and lower legs. An appendectomy was performed on both patients.[43]

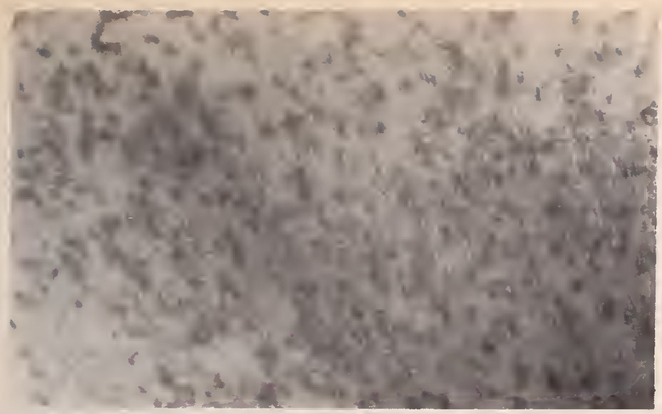

FIGURE 11. Rash of Rocky Mountain spotted fever (from the Centers for Disease Control).

Complications of the disease include pneumonitis, myocarditis, renal failure, gangrene of digits and scrotum, and encephalitis. Thrombocytopenia and disseminated intravascular coagulation are often noted in severe cases.

The case fatality rate among untreated patients is about 15% to 20%. The overall mortality rate in the United States is about 3%.[44] Early recognition and appropriate treatment significantly reduces the case fatality rate especially among children and young adults.

Diagnosis. The early diagnosis of Rocky Mountain spotted fever is of considerable importance, since the disease is amenable to antibiotic therapy. Unfortunately, the salient features in the first few days may simulate other bacterial or viral infections, especially if the patient gives no history of a tick bite. Even in the presence of the rash, the disease can be mistaken for other exanthems such as meningococcemia, atypical measles, rubella, and murine typhus. A definitive diagnosis can only be achieved through laboratory testing.

The nonspecific Weil-Felix test (agglutination of bacillus Proteus OX-19 and OX-2 by the serum of Rocky Mountain spotted fever patients) has been used for the past 50 years in the diagnosis of many rickettsial infections. Agglutinins appear on about the fourth day of sickness and increase to a maximum by the beginning of the third week. Physicians should be careful in interpreting the results of the Weil-Felix reaction, since the test can be positive in many non-rickettsial infections such as urinary tract infections caused by *Proteus*, leptospirosis, brucellosis, borrelia infections, typhoid fever, serious liver disease, and occasionally in pregnancy. A single Weil-Felix agglutination titer of at least ≥1:160, preferably ≥1:320, or a fourfold rise in paired sera are considered indicative of infection.

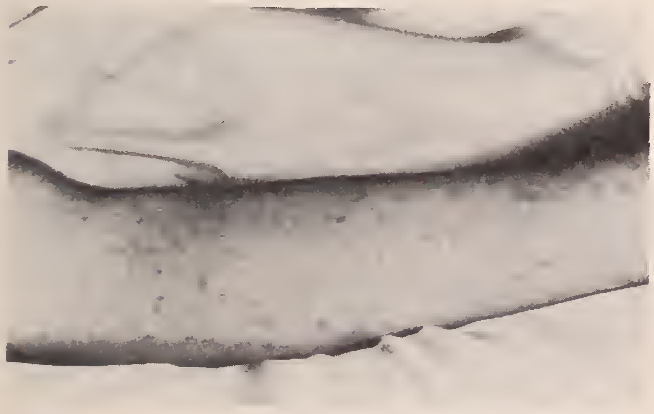

FIGURE 12. Rash of Rocky Mountain spotted fever (from the Centers for Disease Control).

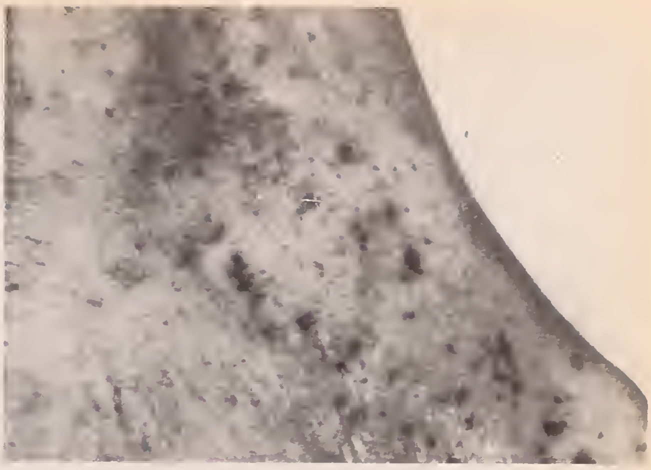

FIGURE 13. Rash of Rocky Mountain spotted fever (from the Centers for Disease Control).

During the early 1940s, the more specific complement fixation test (CF) was developed and used by a few laboratories. A single CF titer of ≥1:16 is considered a positive reaction. The complement fixation test, however, lacks sensitivity, especially if antibiotic therapy was initiated early in the disease process, and is no longer recommended.[45,46]

The last decade witnessed the introduction of several sensitive and highly specific serologic tests. These include the indirect fluorescent antibody (IFA), latex agglutination (LA), indirect hemagglutination (IHA), and microagglutination (MA) tests. A single titer of ≥1:64 for IFA or of ≥1:128 for the LA, IHA, or MA are often considered indicative of infection.[45-47]

In 1981, the Centers for Disease Control established the following criteria for a laboratory-confirmed case of Rocky Mountain spotted fever: "a fourfold increase in antibody titer between acute and convalescent serum specimens by CF, IFA, IHA, LA, or MA; a single convalescent titer ≥1:16 (CF) or ≥1:64 (IFA) in a clinically compatible case; isolation of spotted fever group rickettsiae; or fluorescent antibody (FA) staining of biopsy or autopsy specimens."[45]

In a study by Kaplan and Schoenberger[45] of 1,774 laboratory-confirmed cases reported to CDC between 1981 and 1984, the authors found that the indirect fluorescent antibody and indirect hemagglutination tests were more sensitive than the Weil-Felix, complement fixation, or latex agglutination tests.

Clements and coworkers[48] found that a microtiter enzyme linked immunosorbent assay (ELISA) test using density gradient-purified *Rickettsia rickettsii* was more sensitive than the IFA test in detecting antibodies in single convalescent phase serum. The test was also found most appropriate for detecting IgG antibodies in seroepidemiology and serodiagnosis since it is capable of detecting the IgG levels in a single dilution of serum up to one year after infection.[48]

Because of the vague symptoms in the prodromal period, the disease may simulate upper respiratory and other nonspecific infections. With the appearance of the rash, additional conditions should be considered in the differential diagnosis, namely, atypical measles, meningococcemia, enteroviral infections, typhus, and typhoid fevers. A patient presenting with fever, a maculopapular rash, a history of tick bite, and a high titer in the Weil-Felix test is often diagnosed, and justifiably so, as having Rocky Mountain spotted fever. A few cases investigated by the Suffolk County Department of Health Services with similar histories and clinical and laboratory findings, however, were finally confirmed by serologic testing with specific antigens as measles.[49] In some of these cases of atypical measles, titers as high as 1:640 for Proteus OX-19 were encountered. A similar experience has been reported by Neiburg et al,[50] who demonstrated rising measles antibody titers in six out of 46 paired sera from patients suspected of having Rocky Mountain spotted fever.

On the other hand, an atypical rash, lack or questionable history of a tick bite, and low titers in the Weil-Felix reaction should not exclude a diagnosis of Rocky Mountain spotted fever. One should keep in mind that the prescription of antibiotics during the early stages of a febrile condition often aborts the full development of the rash and delays the emergence of agglutinins. A presumptive diagnosis of Rocky Mountain spotted fever, based on the clinical impression with or without a history of contact with ticks, should justify the administration of an effective antibiotic such as tetracycline. To wait for confirmatory tests and withhold antibiotic therapy is not only medically imprudent but can also be life threatening.

There are no special diagnostic laboratory findings in RMSF. In some severe cases, an elevated creatinine phosphokinase, azotemia, jaundice, hyperbilirubinuria, and abnormal liver function tests have been noted.

Treatment. Patients suspected of having Rocky Mountain spotted fever should be treated immediately on the basis of history and clinical findings.

The two drugs that have been found effective against *Rickettsia rickettsii* are chloramphenicol and tetracycline. Chloramphenicol is administered orally in a dose of 50 mg/kg/day for adults and 50–100 mg/kg/day for children in four divided doses. It is the drug of choice especially for cases in which meningococcemia could not be ruled out, in severe cases, and in pregnant women.[51] Because of the drug's hematopoietic toxicity, the usual precautionary measures regarding chloramphenicol administration should be taken. These include platelet count, hematocrit, and white blood cell count.

Tetracycline hydrochloride is administered orally in a dose of 25–40 mg/kg/day for adults and children to a maximum of 2.0 g/day in four divided doses between meals.[51] In order to avoid dental discoloration of the permanent teeth of children under age eight, the dose should not exceed 25 mg/kg/day. Patients should avoid milk and antacids during tetracycline administration. Doxycycline, 50–100 mg orally or intravenously twice/day, is better absorbed than tetracycline and is recommended especially in the presence of azotemia.

Supportive care is as important as antibiotic therapy. Attention should be given to correcting dehydration, electrolyte imbalance, oliguria, inadequate intravascular fluids, etc. Antibiotic therapy can be terminated two to four days after the patient's temperature returns to normal. The duration of therapy is usually seven to ten days.

The following case illustrates the importance of initiating antibiotic therapy in suspected cases of Rocky Mountain spotted fever prior to laboratory confirmation. A middle-aged man who resided in New York City was bitten by a tick while working in the garden of his summer home in Suffolk County. On the third day, he became ill with fever (103°F), malaise, myalgia, chills, retro-orbital pain, and mild diarrhea. On the fifth day, he visited a physician who prescribed procaine penicillin G. Since the patient's condition did not improve, he revisited the physician on the seventh day. The physician noticed a maculopapular rash on the trunk and recommended hospitalization. The patient was admitted the same evening to a New York City hospital with a temperature of 104°F. Rocky Mountain spotted fever was one of the diseases considered in the differential diagnosis. Blood samples were taken for culture and antibody screening.

On the 11th day, a petechial rash appeared on his palate and extremities. The patient was started on steroids, ampicillin, and doxycycline. The fever persisted, the patient became stuporous, had focal and generalized seizures, and became comatose, with fixed, dilated pupils. Doxycycline was discontinued, and the patient was put on chloramphenicol. By the 15th day, the maculopapular rash spread over the chest, extremities, and scrotum. The patient died 18 days after the tick bite. Whether this patient's death could have been averted had appropriate antibiotic therapy been administered early in the illness is purely conjectural at present.

Epidemiology. During the last two decades, between 500 and 1,200 cases of Rocky Mountain spotted fever were reported annually to the Centers for Disease Control.[52–54] The two most endemic regions in this country are the South Atlantic and the West South Central regions (Fig 14). The highest incidence rates have been reported from Oklahoma, North Carolina, South Carolina, Virginia, Georgia, and Montana. Figure 15 shows the trend in this country from 1955 to 1987. The upward trend during the early 1970s might have been stimulated by the introduction of the CDC surveillance program. There has been a gradual decline in the number of reported cases from the

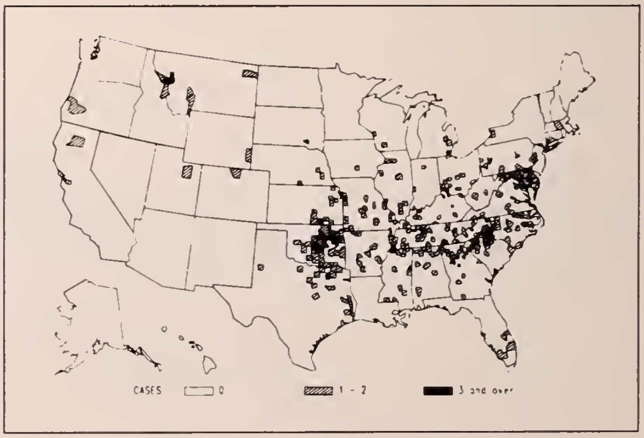

FIGURE 14. Typhus fever, tickborne (Rocky Mountain spotted fever)—reported cases by county, United States, 1987 (from the Centers for Disease Control[52]).

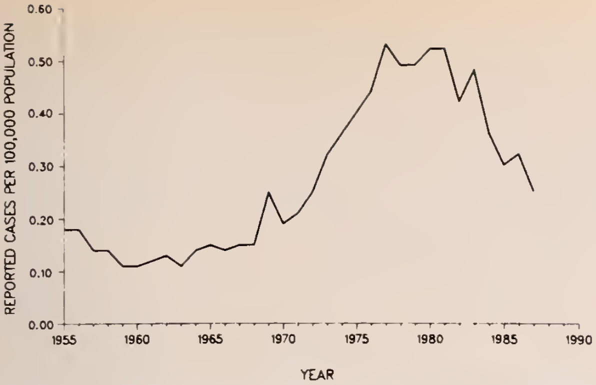

FIGURE 15. Typhus fever, tickborne (Rocky Mountain spotted fever)—by year, United States, 1955–1987 (from the Centers for Disease Control[52]).

1,192 cases in 1981 to 604 in 1987.[52–54] No apparent reasons can account for this decline.

In New York State, the number of cases reported annually between 1977 and 1988 varied from ten to 42 cases (Table I). Most of the New York State cases, with a few exceptions, originated from Suffolk County until 1986.

The incidence of the disease declined dramatically in Suffolk County during the past few years, with three cases reported annually during 1987 and 1988. This decline in incidence cannot be attributed to any extraordinary control measures implemented by the county during the past few years.

In the spring of 1987, four persons who had not been out of New York City during the three weeks preceding their sickness came down with RMSF.[55] Three of these patients were residents of the Soundview area of the Bronx. Of 66 specimens of the American dog tick (*Dermacentor variabilis*) collected from a nearby local park, five, or 8%, were positive for *Rickettsia rickettsii*. Five additional cases occurred among city residents during the year (Fig 16).

The expansion of Rocky Mountain spotted fever from the endemic foci to urban areas emphasizes the importance of considering RMSF in the differential diagnosis of a patient with suspected febrile illness in the absence of a history of travel to endemic areas.

Age and sex. The disease occurs more frequently among the younger age groups in the eastern United States. This is due to the fact that the main vector of transmission in the eastern region is the American dog tick *Dermacentor variabilis.* Although an outdoor tick, it is often carried by pets, which have close contact with children. The few cases reported from the western states occur mostly among adults, since the main vector of transmission is the wood tick, *Dermacentor andersoni.* On a nationwide basis, more than 40% of all cases occur among persons under 20 years of age, with males comprising more than 60% of cases. The case fatality rate is higher for those 40 years of age or older: 5.8% as compared with 1.9% for younger patients.[44]

TABLE I. Incidence of Tickborne Infections in New York State, 1977–1988

Year	Rocky Mountain Spotted Fever	Babesiosis	Lyme Disease*
1977	42	1	—
1978	33	8	33
1979	35	8	—
1980	14	12	11
1981	18	1	106
1982	18	12	207
1983	10	15	411
1984	13	14	826
1985	17	19	600
1986	26	18	443
1987	15	7	877
1988	10	8	1,963
Totals	251	123	5,477

* Reportable as of 1986.

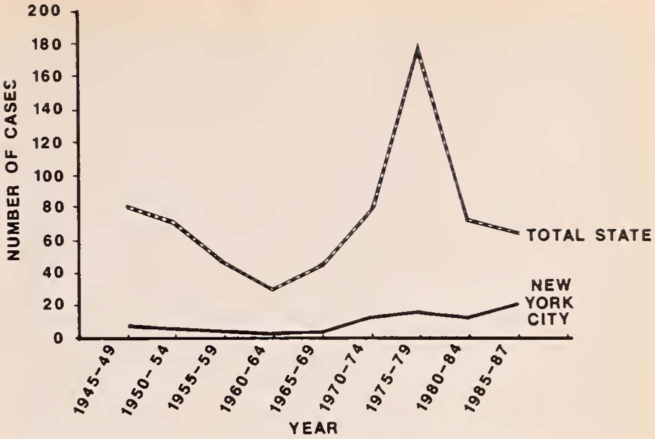

FIGURE 16. Rocky Mountain spotted fever reported cases, New York State and New York City, 1945–1987 (adapted from reference no. 33).

Seasonal distribution. Most cases of RMSF occur between the months of April and October.

BABESIOSIS

Babesiosis is a tickborne, malaria-like illness caused by a protozoan of the genus *Babesia.* Several species of this organism can infect many domestic and wild animals, especially cattle, sheep, goats, equines, hogs, dogs, and cats, leading to a disease called piroplasmosis. On rare occasions, man can get infected accidentally.

Most of the human infections in this country are caused by *Babesia microti*, which are transmitted to man by the bite of an infected deer tick, *Ixodes dammini.*[56]

Although the American dog tick, *Dermacentor variabilis*, is prevalent in many areas known to be endemic for babesiosis, it does not play a role in the transmission of human babesiosis. Some *Dermacentor* species have been known to transmit equine babesiosis.[57]

The reservoirs of infection are believed to be rodents for human infections with *B microti*, cattle for bovine infections with *B bovis*, and dogs for canine infection with *B canis.*

The babesia infect the erythrocytes, leading to hemolysis. In contrast with the malaria parasite, *Babesia microti* does not produce any pigments within the erythrocytes. Infection with *Babesia microti* may lead to many outcomes ranging from the asymptomatic infection to severe, even fatal disease, with fever, chills, and hemolytic anemia. Asymptomatic infections occur frequently in young adults and represent a special risk through blood donations. Smith et al[58] reported on a transfusion-acquired *Babesia* infection in a 72-year-old man who received frequent red blood cell transfusions. One of the identified blood donors had a positive *Babesia* titer of 1:512.

The first identified European patients and a few of the American ones were asplenic, and their cases were rather severe, often fatal. It was thus assumed that the absence of the spleen was responsible for the serious sequelae. Later, it was realized that an intact spleen was not always associated with mild infections. Many of the patients who had severe or fatal infections with extensive parasitemia and hemolysis had intact spleens. High parasitemia has been observed, on a few occasions, in splenectomized patients who were also on steroid therapy. Whether the spleen itself or the condition that resulted in splenectomy was responsible for the serious sequelae is not known at present.

Following an incubation period of one week to several months after a tick bite, the patient experiences malaise, headache, fatigue, fever, sweats, chills, myalgia, and, in some patients, nausea and vomiting. The fever is irregular and does not follow the classical periodicity characteristic of malaria.

Physical examination often reveals splenomegaly and hepatomegaly. Hemolytic anemia and hemoglobinuria are common findings. Hemolytic changes include reticulocytosis, reduced serum haptoglobin levels, and moderate thrombocytopenia. Parasitemia can be observed in 1% to 10% of the erythrocytes in the peripheral blood. Extensive parasitemia has been noticed, on occasion, in about 80% of peripheral erythrocytes. Other common findings are mild elevations of serum bilirubin, SGOT, and alkaline phosphatase.

Diagnosis. Babesiosis should be considered in the differential diagnosis of a febrile patient from an endemic area—especially during the tick season—who presents with a malaria-like illness. Definitive diagnosis can be established by finding the characteristic intraerythrocytic forms (rings, tetrads, pyriforms) on thin or thick Giemsa-stained blood smears. Ring forms of *Babesia* can be distinguished from those of *Plasmodium falciparum* by the absence of pigment. Another feature that differentiates *Babesia* from *Plasmodia* is the absence of the schizonts and gametocytes often observed in *Plasmodia* infected smears.

Confirmation of diagnosis can be made by either the intraperitoneal inoculation of hamsters with the patient's blood and the demonstration of parasitemia in blood smears or by serologic tests. Antibodies to *Babesia microti* can be demonstrated in the patient's serum within the first few weeks of illness by the IFA test. Titers may rise to 1:1024 and then gradually fall during the following months.[59]

Treatment. A few drugs have been tried for the treatment of patients with babesiosis, but none so far is considered the drug of choice. Antimalarial drugs such as chloroquine, sulfadiazine, and pyrimethamine cause slight symptomatic improvement but are unsuccessful in reducing the parasitemia.

Pentamidine, an antitrypanosomal drug, has been found to be effective in high doses, which are relatively toxic. In a therapeutic trial involving three non-splenectomized patients, Francioli et al[60] administered pentamidine isethionate in a single intramuscular dose of 4 mg/kg daily for 14 days. Parasitemia disappeared after the fifth day. Five weeks later, babesia organisms could still be recovered by animal inoculation. In another patient, the drug had to be suspended after seven days because of increased creatinine concentration.[61] Among the adverse effects accompanying pentamidine therapy are pain at the injection sites and the formation of sterile abscesses. The authors concluded that pentamidine controlled the clinical manifestations, reduced the parasitemia, but did not eradicate the organism.[60]

Diminazene aceturate, another anti-trypanosomal drug, was effective in one patient. The patient, however, developed Guillain-Barré syndrome during convalescence. Although no cause and effect relationship was established, the incident discouraged further trials with the drug.[61]

Chemotherapy with a combination of clindamycin and quinine has been tried in a few patients with varying degrees of success. The following cases illustrate the problems in treating patients with *Babesia* infection.

A premature female infant was born of a mother who was suffering from fever before parturition. The infant had respiratory distress and necrotizing enterocolitis. She received 11 blood transfusions from ten separate donors. During the eighth week of life, her blood smear showed an 8% parasitemia with intraerythrocytic ring forms suggestive of *Plasmodium falciparum*. The infant was treated unsuccessfully with chloroquine. Because of the persistent parasitemia, quinine, 25 mg/kg body weight orally, and clindamycin, 20 mg/kg body weight intramuscularly, were administered for three days. The parasitemia drastically declined. When the therapy was discontinued, the parasitemia recurred, and therapy had to be resumed. After seven days of therapy, blood smears did not show any babesia.[62]

In another trial reported by Dammin et al,[63] effective therapy was achieved in two patients aged 60 and 73 years following the administration of quinine (650 mg orally every six to eight hours) and clindamycin (300 mg or 750 mg intravenously every six hours) for 10–11 days. Smith et al,[58] on the other hand, were unable to treat a 72-year-old man who had transfusion-acquired babesiosis with combined quinine and clindamycin therapy.

In a splenectomized woman with a 15% parasitemia, Cahill et al[64] found that red cell exchange permanently reduced the parasitemia to less than 1%. The patient had previously been treated unsuccessfully with chloroquine and pentamidine isethionate.

In another trial, Jacoby et al[65] found that whole blood exchange was life saving in severe babesia infections. In the authors' opinion, whole exchange has the advantage of providing free haptoglobin, which is present in fresh frozen plasma, decreases intracellular and extracellular parasitemia, and also removes "the serum factors that have been implicated in the pathogenesis of renal complications of babesiosis."[65]

Epidemiology. The first case of human babesiosis was reported from Yugoslavia in a farmer who was exposed to tick-infested cattle in 1956. The farmer had undergone a splenectomy 11 years earlier.[66]

The second human babesiosis case involved a 46-year-old man from California who, in 1966 (two years after splenectomy), was hospitalized with chills and fever. He was diagnosed as having malaria and was treated for 15 weeks with chloroquine. Later, tetrad intraerythrocytic organisms were noted in his blood. Serologic tests were negative for *Plasmodia* but were positive for *Babesia* antibodies, the species of which was believed to be of equine origin.[67] The third case occurred in Ireland and the fourth in Yugoslavia. Between 1969 and 1975, seven additional cases were reported from Nantucket Island in Massachusetts. Additional cases were reported from Cape Cod, Martha's Vineyard, Massachusetts, and Long Island, New York.

The first case of human babesiosis in New York State occurred in Shelter Island, Suffolk County, in 1975.[68] The investigation that resulted in the identification of this case involves clinical and epidemiologic features that may be of interest to the practicing physician and is, therefore, reported here.

On July 18, 1975, a 59-year-old man visited his family physician complaining of what appeared to be an insect bite above the left ankle. The bite site was red, swollen, and painful. The patient's temperature was 100.4°F. Warm wet dressing was prescribed in addition to 500 mg ampicillin twice daily. Six days later the patient revisited his physician complaining of generalized aches and pains. Physical examination did not reveal any abnormal findings, but the bite site had improved. Ampicillin therapy was continued. Two days later, he complained of low back pain and frequency and urgency of micturition. Sulfisoxazole was prescribed. Nine days after his initial visit to the physician, the patient experienced shaking chills and left flank pain. In the absence of his physician, he was seen by another physician who recommended hospitalization.

On admission to the hospital, the patient gave a history of fever, myalgia, arthralgia, headache, nausea, anorexia, and diarrhea. Physical examination revealed abnormal tenderness, distended bowel, and an enlarged liver. The spleen was normal. The admission note included a statement to "rule out hepatitis and rickettsial disease." He was started on tetracycline for possible Rocky Mountain spotted fever, but this was discontinued the next day when blood smears showed "ringed trophozoites of malaria—possibly *Plasmodium vivax*." He was placed on chloroquine and primaquine during his first four days of hospitalization. His temperature ranged between 100°F and 103°F, became normal on the fifth hospital day, and remained so until discharge. During his hospital stay of 16 days, there was a drop in his hemoglobin from 11.8 g to 9.3 g. Other positive laboratory results included a mildly elevated SGOT and bilirubin. Serologic test for hepatitis was negative; so were the urine and blood cultures and stool cultures for ova and parasites.

Recognizing the fact that *Plasmodium vivax* infections do not usually relapse after five years, an attempt was made to identify the source of infection. An epidemiologic investigation was conducted which entailed the interview of 13 families and the relatives of the patient, a family of five Vietnamese refugees, and one World War II veteran. The latter had a history of malaria in 1947, was treated, and did not suffer a relapse. Of the 400 mosquitoes collected around the patient's home, only one was of the *Anopheline* species. No other significant findings were identified.

Because of the inconclusive evidence thus far obtained, subsequent blood films were taken for examination. These revealed trophozoites resembling those of *Plasmodium malariae*, a species known to produce relapse several years after the initial infection.

The patient was born in Italy and served in Albania and Yugoslavia during World War II. He came to the United States in 1949 and had not been in an endemic malarial area in 25 years. In 1955–1956 he travelled to Florida, California, and Canada. Since then he had only travelled locally and rarely left Shelter Island during the previous year. He had no history of malaria or of a suspicious illness. However, in view of the patient's negative travel history and denial of blood transfusion or the use of shared syringes, the diagnosis of malaria was reevaluated.

Subsequent blood films revealed a persistent parasitemia and numerous organisms similar to those observed in the original films, in addition to some new intracellular organisms conforming morphologically with *Babesia microti*. This diagnosis was later confirmed by the Centers for Disease Control on morphologic and serologic grounds. The patient's condition gradually improved and he did not visit his physician for almost two years. On his last visit, he had symptoms suggestive of Lyme disease. He refused to undergo further testing and eventually moved to Florida (E. Grunwaldt, personal communication, 1989).

In the United States, most of the babesiosis cases occur in a few endemic areas within the states of Massachusetts and New York. The endemic areas include Nantucket Island, Martha's Vineyard, and Cape Cod in Massachusetts, and Shelter Island, Fire Island, and the eastern towns of Long Island in New York.

Age and sex. Between 1977 and 1988, 123 babesiosis cases were reported to the New York State Department of Health. Of these, 104 occurred in Suffolk County. Of the 104 cases, 90, or 86%, involved persons 40 years of age or older, and 63 patients, or 61%, were males.

Prevalence of infection. Paired sera from 102 persons on Shelter Island were tested in June and October 1978 by the indirect fluorescent antibody test. The point prevalence was 4.4% in June and 6.9% in October. None of the 300 serum samples obtained from the New York City metropolitan area were positive for *B microti* antibodies.[69]

Ruebush and coworkers[70] tested serum samples from 577 patients undergoing routine diagnostic studies in Nantucket Hospital and from 133 residents and visitors of the hospital. Eleven of the 577, or 1.9%, and ten of the 133, or 7.5%, had antibody titers of ≥1:64 to *B microti* by the IFA test.

CONTROL OF TICKBORNE INFECTIONS

The control of any arthropod-borne infection, tickborne diseases included, can only be formulated after a thorough understanding of the life cycle of the arthropod, available reservoirs, modes of transmission of the pathogen, diagnostic tools, and prophylactic measures.

The deer tick (*Ixodes dammini*) responsible for the transmission of Lyme disease and babesiosis has a life cycle of about two years (Fig 17). The female tick deposits her eggs outdoors in the spring. A few weeks later, they hatch into free-living larvae. The larvae feed once over a

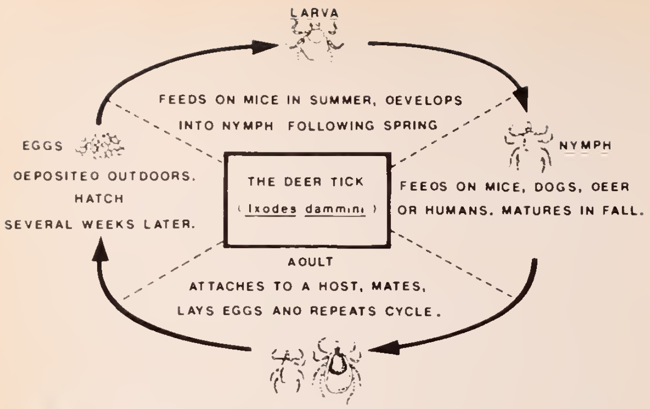
LARVA
FEEDS ON MICE IN SUMMER, DEVELOPS INTO NYMPH FOLLOWING SPRING
EGGS
DEPOSITED OUTDOORS. HATCH SEVERAL WEEKS LATER.
THE DEER TICK (Ixodes dammini)
NYMPH
FEEDS ON MICE, DOGS, DEER OR HUMANS. MATURES IN FALL.
ADULT
ATTACHES TO A HOST, MATES, LAYS EGGS AND REPEATS CYCLE.

FIGURE 17. The life cycle of the deer tick.

few days during the summer, usually on the blood of small rodents such as mice. As the cold weather approaches, they enter a resting stage. The following spring, the immature six-legged larvae develop into larger eight-legged nymphs. During the summer, the nymphs attach themselves and feed on the blood of several animal hosts. The white-footed mouse, *Peromyscus leucopus*, is one of the most frequently infested hosts. Other animals such as dogs, deer, horses, raccoons, and opossums act as occasional hosts.

In the fall, the nymphs mature into adult ticks. They can be found in tall grass and brush a few feet above the ground, where they can easily attach themselves to larger animals such as the white-tailed deer, where the ticks mate. Other hosts include dogs, horses, and humans. The males die soon after mating, while the females continue to feed for egg development. Only the female ticks survive the winter. They then deposit their eggs outdoors in the spring and die. The eggs hatch in the spring and the cycle is repeated.

The American dog tick (*Dermacentor variabilis*) has a much shorter life cycle (Fig 18).

Control of arthropod-borne infections can be achieved through one or more of the following:

- Destruction of the vector
- Use of repellents
- Reduction in the density of animal hosts
- Habitat modification
- Public education of those living in endemic areas, and
- Professional education.

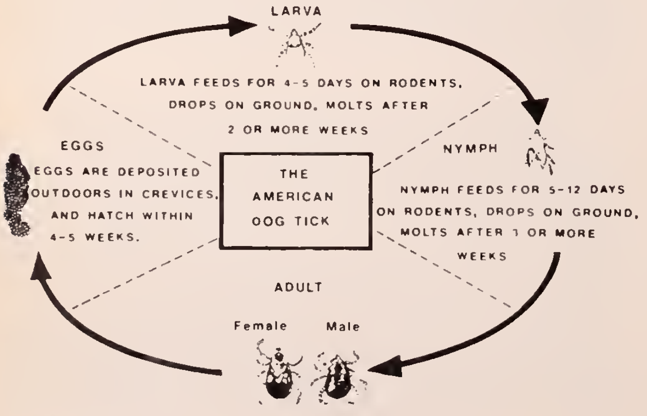
LARVA
LARVA FEEDS FOR 4-5 DAYS ON RODENTS, DROPS ON GROUND, MOLTS AFTER 2 OR MORE WEEKS
EGGS
EGGS ARE DEPOSITED OUTDOORS IN CREVICES, AND HATCH WITHIN 4-5 WEEKS.
THE AMERICAN DOG TICK
NYMPH
NYMPH FEEDS FOR 5-12 DAYS ON RODENTS, DROPS ON GROUND, MOLTS AFTER 1 OR MORE WEEKS
ADULT
Female
Male

FIGURE 18. The life cycle of the American dog tick.

Destruction of the Vector. In many arthropod-borne infections this can be accomplished by reducing the chances of breeding of the arthropod. If this is not totally successful, attempts are made to kill the larval or immature forms, and if these fail, destruction of the adults has to be undertaken.

In the case of mosquitoes, eggs are usually deposited in stagnant water. After hatching, the larvae and pupae live in water bodies until maturation. Control of mosquito-transmitted diseases relies therefore on the cleaning of ditches and removal of debris, thus reducing the chances of egg deposition. This is complemented by larval control through biologic and chemical insecticides, and if these cannot achieve effective control, adult control can be accomplished through the use of chemical insecticides.

The situation in tickborne diseases is, unfortunately, quite different for two reasons. First, the eggs of the deer tick or the American dog tick are laid on the ground, and second, the immature forms live on mice and several domestic and wild animals.

A few acaricides have been used in the past for the control of *Dermacentor variabilis*, *Dermacentor andersoni*, and *Amblyomma americanum*, which are responsible mainly for the transmission of Rocky Mountain spotted fever. The adult forms of these ticks are often found on shrubs, high grass, in trails, paths, and campsites, and they often infest dogs and cats. Acaricides such as chloropyrifos (Dursban), diazinon, carbaryl, dioxathion (Deltic), baygon, and permethrin have been used with limited success for area control. Pet collars and shampoos have also been utilized to reduce tick infestation in dogs and cats.

Destruction of the adult *Ixodes dammini* has been tried by using two acaricides, carbaryl and diazinon, which were sprayed by a high pressure hydraulic sprayer in a few wooded areas during the fall.[71] A marked reduction in the *Ixodes dammini* populations was observed following application. The next fall, the *Ixodes* populations were similar in the treated and untreated areas.

The total reliance of *Ixodes dammini* on domestic and wild animals, especially deer, mice, and other rodents, for survival makes area control through the spraying of acaricides rather unproductive.

Recently, a method was introduced to reduce the larval and nymphal stages of *Ixodes dammini*.[72] This method involves the placement of cardboard tubes containing permethrin-treated cotton balls on the ground about ten yards apart. Applications are to be repeated twice a year during the tick season. The white-footed mice are expected to use the cotton balls as nesting material and, in the process, the larvae and nymphs of *Ixodes dammini* are killed. Initial field studies showed that 72% of the mice in treated areas were free of ticks, as compared with 1% in the untreated areas. According to the manufacturer, the use of the permethrin treated cotton can drastically reduce the risk of transmission of Lyme disease.

A few issues have to be addressed prior to considering this method for the control of Lyme disease in public health programs. The efficacy of the impregnated tubes has to be tested under existing ecologic and meteorologic conditions of the endemic areas. Another question involves the role of transmission by other hosts and reservoirs and the migration of mice from untreated areas. A very crucial issue that cannot be overlooked is the expense that would be incurred by using this method in large endemic areas. The cost of the tubes and the labor involved may make this approach fiscally prohibitive for any municipality.[73]

At present, there is no stock formula that can be recommended for control of Lyme disease vectors. Every situation has to be evaluated on its own merits. Great caution, however, must be practiced in the formulation of any control program to avoid the indiscriminate area spraying of acaricides in endemic areas that depend entirely on groundwater, such as Suffolk County. Indiscriminate spraying to achieve control would not only be scientifically unjustified, but would also be environmentally unsafe.

Repellents. Currently, there are numerous repellents available which contain DEET (N, N diethyl-m-toluamide) or the Rutgers' formulation (2 ethyl-1,3-hexanediol). These chemicals repel flying-biting insects and ticks. However, they have serious drawbacks:

1. They are short lasting and easily removable by moisture or friction. Although flying-biting insects such as flies and mosquitoes are noticed when the effectiveness of the repellent is lost, ticks frequently go undetected.
2. They are not usually applied to clothing, although they may be used in this manner in an aerosol form. When sprayed on clothes they frequently provide only partial protection.

Individuals are encouraged to wear long pants and long-sleeved shirts or blouses when entering infested areas. Since ticks usually make initial contact with clothing, it is important that an effective repellent be applied at this point of entry.

At present, there is at least one effective repellent on the market, Permanone, which contains the synthetic pyrethroid compound, permethrin. It is applied to clothing where it can remain effective for several weeks or for one or more washings.[74] Permanone is not only a repellent, but is also an acaricide. It is currently being used by the military for treatment of uniforms. In addition, it is available for public use in 21 states. At present, it is not registered for use in New York State.

Host Reduction. Since the white-tailed deer is the principal host of the adult *Ixodes dammini*, attempts were made to reduce the tick population by reducing the deer population. Wilson et al[75] in 1984 found that the destruction of approximately 70% of the deer did not markedly reduce tick infestation.

Habitat Modification. Since ticks are abundant in wooded areas with excessive vegetation, attempts have been made in the past to mow and burn vegetation to reduce the tick population. This method was used several years ago for the control of Rocky Mountain spotted fever. Habitat modification can temporarily reduce the tick infestation in nonresidential areas. Destruction of vegetation sometimes leads to the migration of small mammals to denuded areas. In addition, the practice of burning brush and vegetation may be environmentally unsound and in violation of municipal ordinances.

Public Education. Since acaricides cannot be used for effective control and, lacking a vaccine, reliance must be placed on public and professional education. Public education is targeted toward reducing the chances of exposure to the tick population and informing the public of the clinical features of the disease so that they may seek early medical intervention. Several pamphlets have been developed during the past two years by health agencies and medical schools and are available on request.

Among the measures that can be recommended to those living in endemic areas are the following:[76]

- Try to avoid heavily infested areas where deer are known to exist, especially near water in wooded areas.
- Wear long-sleeved shirts, pull socks over pant legs, and wear closed shoes.
- Use tick/insect repellents. Some of the commercial products containing the chemical DEET can be helpful. Follow the label directions carefully.
- On returning from a wooded area, inspect yourself, your children, and your pets. Shower and wash your clothes immediately.

The public needs to be made aware of the fact that *Ixodes dammini* ticks are widely distributed, even in public parks with high annual attendance rates. A recent survey of eight recreational parks in Westchester County, New York, demonstrated that *Ixodes dammini* was present in all but one park. This species accounted for 580 (91.8%) of the 632 ticks collected. The majority of the ticks were nymphs (80%), 18% were larvae, and only 2% were adults.[77]

Professional Education. Professional education can be achieved through informing the practicing physicians of the pertinent features in the epidemiology of Lyme disease, characteristic clinical features, methods of diagnosis, and appropriate treatment regimens. The following approaches can be utilized:

- Presentations on tickborne infections to medical staffs in hospitals.
- Frequent personal communication with all practicing physicians in the community updating epidemiologic and clinical information.
- Educational conferences and seminars for practicing physicians in teaching institutions as part of the continuing medical education process.

Prophylaxis. Increased public awareness of Lyme disease in endemic areas has sensitized the residents to watch carefully for tick bites. When patients seek medical attention, the primary care physician is often faced with the question of whether or not to treat a tick bite with antibiotics. This was especially emphasized following the publication of a few studies that indicated a high infection rate of the *Ixodes dammini* tick with *Borrelia burgdorferi*.

Several issues have to be considered prior to adopting a practice of antibiotic prophylaxis. These are:

1. More than half of those who develop clinical Lyme disease give no history of tick bites.
2. Most endemic areas have in addition to the *Ixodes dammini* one or more of other tick species such as the *Dermacentor variabilis*, *Dermacentor andersoni*, *Amblyomma americanum*, and *Ripicephalus sanguineus.* It is true that the other ticks are larger than the *Ixodes*, but the difference in size cannot always be ascertained by the patient.
3. Even if a person is bitten by an *Ixodes dammini*, there is always the chance that the tick was free of infection.
4. Being bitten by an infected tick does not necessarily lead to human infection or clinical disease. Whether certain strains of the *Borrelia burgdorferi* have low infectivity or whether other factors play a role in transmission is unknown at present.

In a study conducted by Costello and coworkers,[78] 56 residents bitten or assumed to have been bitten by an *Ixodes dammini* were divided into two groups. One group of 27 was given penicillin for prophylaxis and one group of 29 received a placebo. One individual in the treated group developed an allergic reaction to penicillin, and one in the placebo group developed early Lyme disease. Of the 21 ticks examined, six (three from each group) were positive for *B burgdorferi*, an infection rate of 29%.[78] The above demonstrates the futility of the indiscriminate administration of antibiotics to those bitten or assumed to have been bitten by ticks. Such a practice is not only medically unjustified but is also unsafe.

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FROM THE LIBRARY

THE EPIDEMIOLOGY OF ROCKY MOUNTAIN SPOTTED FEVER

. . . Geographically, Rocky Mountain spotted fever is now located throughout the United States. All of the states with the exception of five, three of the New England States, Michigan, and Wisconsin, have reported cases contracted within their borders. Each year there seem to be an increasing number of cases reported, but we all feel that this is not a true increase in number, but rather an increase in recognition of the disease since doctors are becoming more familiar with it. Each year an additional county is put on our spot map. Even though the first reported case was in the 1930's, it probably existed in the locality for many years before that.

The disease is acquired where ticks are contacted. People on the streets of New York City do not contract Rocky Mountain spotted fever. On Long Island they do get the disease because they contact ticks. A simple example of the epidemiology of Rocky Mountain spotted fever could be given if we had a sufficient number of cases in golfers. I am sure we could correlate the incidence of spotted fever with the quality of the golf. The good golfer would not contract Rocky Mountain spotted fever, but the poor golfer would, because the better the golfer, the more he sticks to the fairway, and the poorer the golfer, the more he is in the rough. In a city such as Washington, where we see a considerable number of cases of Rocky Mountain spotted fever each year, it has always been observed that the case usually occurs in the house at the end of the street, where bushes and underbrush are close by where the children play. It is also a disease we see in people engaging in recreation. . . .

NORMAN H. TOPPING, MD
(*NY State J Med* 1947; 47:1585–1587)

Acute renal failure in children: Etiology, diagnosis, and management

MELANIE SWEET, MD; KANWAL K. KHER, MD; SUDESH P. MAKKER, MD

Acute renal failure (ARF) is defined as a sudden deterioration in renal function that results in an inability to excrete nitrogenous wastes. ARF may be a significant cause of morbidity, particularly when it accompanies involvement of other organ systems, as is often the case in the pediatric and neonatal intensive care patient. Despite major advances in techniques of supportive care, including dialysis and nutritional support, mortality remains high for such patients.

The purpose of this review is to provide an approach for treating the child with acute renal insufficiency and to outline the diagnostic studies that may aid in the determination of the cause of the renal injury. The pathophysiology of ARF is explored, and the principles of supportive care are reviewed.

CAUSES OF ACUTE RENAL FAILURE IN CHILDREN

The kidney is susceptible to a number of acute insults that are capable of producing renal injury and failure of glomerular filtration: ischemia, toxins, immunologic disorders, and obstruction to urine flow. The underlying cause of the injury is influenced by the age of the patient, the setting in which renal failure occurs, and also by environmental factors. In the premature infant, for example, hypovolemia, perinatal asphyxia, and congenital disorders are frequent causes of ARF.[1-4] In the older infant, acute renal insufficiency may develop as a result of severe dehydration or as a consequence of the hemolytic-uremic syndrome.[5,6] ARF due to acute glomerulonephritis is usually seen in the older child and adolescent.[5] In addition to age, other factors may determine the relative frequency of various causes of ARF in any given reported series. In underdeveloped nations, for example, renal ischemic injury secondary to gastrointestinal disease and dehydration is common.[7] At The Hospital for Sick Children in London, a large referral center for children with congenital heart disease, cardiopulmonary bypass surgery is the single most frequent cause of ARF.[8] Other epidemiologic factors influence the types of illnesses that cause ARF. Diseases that may be relatively infrequent in some geographic areas may be endemic in others, such as hemolytic-uremic syndrome in Argentina[9] and in Utah.[10]

It is helpful to categorize acute renal failure in terms of the primary site of the disorder. As such, the problem may be due to inadequate perfusion of the kidneys ("prerenal azotemia"), or may be the result of blockage of urine flow (obstructive uropathy or "postrenal azotemia"), or may be caused by a process that occurs within the renal parenchyma itself ("intrinsic renal failure"). Table I outlines various causes of ARF within these categories.

TABLE I. Causes of Acute Renal Failure

- Prerenal azotemia
  - Dehydration
  - Hemorrhage
  - Septic shock
  - Cardiac failure
  - Hypoalbuminemia with resultant decrease in intravascular volume—nephrosis, liver disease
  - "Third space" losses—peritonitis, pancreatitis, burns
- Postrenal azotemia (obstructive uropathy)
  - Bilateral renal obstruction—stones, clots, tumor, congenital anomaly
  - Bladder outlet obstruction—urethral valves, neuropathic bladder, phimosis, stricture
- Intrinsic renal disease
  - Glomerulonephritis (GN)
    - Acute poststreptococcal GN
    - Membranoproliferative GN
    - Rapidly progressive GN
    - Systemic lupus erythematosis with nephritis
    - Henoch-Schönlein purpura nephritis
- Vascular
  - Renal artery thrombosis or embolus
  - Renal venous obstruction
    - Vasculitis
    - Hemolytic-uremic syndrome
    - Disseminated intravascular coagulation
- Acute tubular necrosis/vasomotor nephropathy
  - Nephrotoxic
  - Pigment-associated
  - Ischemic
- Acute interstitial nephritis
  - Drug-related (penicillins, cephalosporins, thiazides, non-steroidals, allopurinol, others)
  - Infection-induced
  - Other—tumor infiltration, uric acid, hypercalcemia

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In prerenal azotemia, the decrease in glomerular filtration rate (GFR) is due to an inadequate circulation to the kidneys—be that from dehydration, hemorrhagic shock, cardiac insufficiency, or inadequate circulating volume secondary to hypoalbuminemia. Prerenal azotemia is probably the most common cause of acute renal failure in children, if the true incidence could be known. If the circulation can be restored with sufficient rapidity, renal function will normalize. Thus, this is sometimes called "functional" renal failure.

Obstructive uropathy in childhood is usually due to a congenital abnormality and is often more of a chronic nature than an acute one. For azotemia to occur, there must either be obstruction of a solitary kidney or bilateral renal obstruction. Severe phimosis, urethral stricture, congenital urethral valves, and neuropathic bladder will lead to obstruction at the level of the bladder outlet. Retroperitoneal processes such as tumor, hemorrhage, and fibrosis can obstruct both ureters and result in acute renal failure. As in prerenal azotemia, correction of the obstruction will result in normalization of renal function, assuming that significant intrinsic renal damage has not already occurred.

A number of disorders with varying pathophysiologic mechanisms can lead to intrinsic renal failure. Such disorders may primarily involve the glomerulus, as in glomerulonephritis, the vasculature of the kidney, as in renal vascular thromboses and vasculitis, or the tubules and/or interstitium of the kidney, as in acute tubular necrosis and acute interstitial nephritis. The kidneys are particularly susceptible to injury from ischemia and toxins. This is due to the large portion (20–25%) of the cardiac output that is received by the kidneys, as well as the ability of the kidney to absorb solutes and attain a high medullary tonicity, which in turn allows high concentrations of potentially toxic substances to build up in the renal parenchyma. Table II lists exogenous and endogenous toxins capable of causing renal injury and giving rise to acute tubular necrosis. The term "acute tubular necrosis" has fallen out of favor in recent years and has been replaced by "vasomotor nephropathy," for reasons which will be explained.

PATHOPHYSIOLOGY

Given the diversity of the causes of ARF, it is evident that no single physiologic alteration or abnormality can explain the failure of filtration in all cases. Our understanding of the pathophysiology of ARF has been greatly expanded in recent years, particularly in the area of vasomotor nephropathy. It is on this particular aspect of ARF that this discussion will focus. In addition, several excellent reviews of this subject have recently appeared in the literature.[11–13]

TABLE II. Potential Nephrotoxins

Acetaminophen	Heavy metals
Aminoglycosides	Hemoglobin
Amphotericin B	Methotrexate
Calcium	Methoxyfluorane
Captopril	Myoglobin
Cis-platinum	Nonsteroidal anti-inflammatory drugs
Contrast agents	Organic solvents
Cyclosporin	Uric acid

The formation of an ultrafiltrate of plasma and the ultimate production of the final urine are determined by a number of variables. Adequate perfusion of the kidneys, and thus plasma flow, through the glomerular capillaries provides the main driving force for ultrafiltration. Under normal circumstances, renal blood flow is controlled over a wide range of mean arterial pressures, a process called autoregulation.[14] If the lower limits of autoregulation are exceeded, renal blood flow will fall. An additional factor affecting ultrafiltration is the hydrostatic pressure in the Bowman space. If this pressure were to become sufficiently elevated, as might be seen in intratubular obstruction, the net force for ultrafiltration would be reduced. Other variables influencing glomerular filtration and excretion of the final urine are the filtering properties and surface area of the glomerular capillary wall (called the ultrafiltration coefficient), and the overall integrity of the renal tubular epithelium. Therefore, alterations in any of these factors may result in impairment of excretory function.

The contributory role of tubular abnormalities in the decline in GFR was the initial focus of clinical and experimental work in ARF. Minami, in 1923,[15] emphasized the prominent finding of tubular injury and intraluminal debris in autopsy specimens of patients dying of ARF, as is illustrated in Figure 1. It was then felt that tubular obstruction with resultant increase in intratubular pressure, and thus a decrease in net filtration pressure, was the principal cause of the decline in renal function. It was later emphasized that tubular injury could also lead to disruption of tubular integrity with resultant abnormal permeability of this barrier, in such a manner that while glomerular filtration might be occurring, "backleak" of water and solute would lead to oliguria and retention of waste products. This theory was proposed by Richards in 1929[16] and has subsequently been confirmed as a contributory factor in some experimental animal models and in human ARF.[17,18]

As suggested earlier, however, in many cases of ARF, significant tubular damage is not histologically evident. For this reason, the term acute tubular necrosis is not always suitable, and the term vasomotor nephropathy is preferred. Most recent research in the field of ARF has

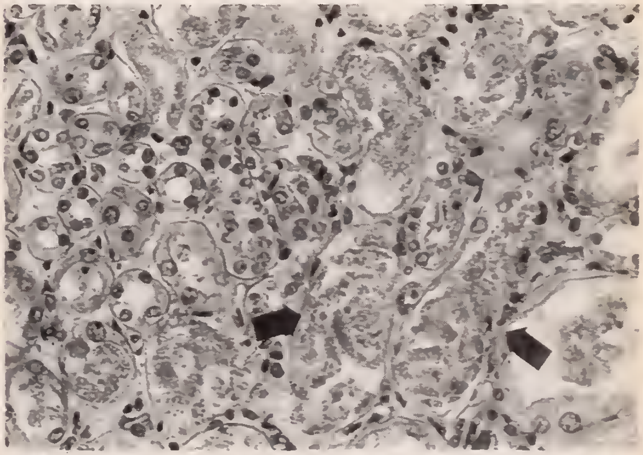

FIGURE 1. Post-mortem specimen from a child in whom acute renal failure developed following open heart surgery. Large amounts of debris (arrows) can be seen within the tubular lumens (hematoxylin-eosin stain, original magnification × 1250). Courtesy of Dr Victor Saldivar, Santa Rosa Medical Center, San Antonio, Texas.

focused on hemodynamic alterations and changes in glomerular capillary permeability as underlying the defect in GFR.[19–21] Such changes might be mediated by the renin-angiotensin system,[22] by prostaglandins,[23] by adenosine,[24] or by other vasoactive compounds. In addition, alterations in renal energy metabolism have been demonstrated in experimental ARF.[13] The situation is made more complex by the finding that different pathologic processes may be operant in the initiation phase of ARF from those in the maintenance phase.[25] What has been clearly demonstrated is that a multiplicity of factors are capable of altering GFR, and that these can contribute to varying extents depending on the nature, severity, and the time course of the renal injury. Research in this field is ongoing, and it is evident that a clearer picture of the factors affecting glomerular filtration will allow a more logical approach to the prevention and treatment of ARF.

EVALUATION OF THE CHILD WITH ACUTE RENAL FAILURE

Several important points should be emphasized in the evaluation of a patient with a suspected decrease in renal function. It should first be realized that not all patients with acute renal failure have diminished urine volumes. Nonoliguric ARF is being recognized with increasing frequency in recent years.[2,26,27] It is therefore inadequate to rely on urine output alone as an indicator of renal function; biochemical determinations are essential in patients at risk for renal injury.

Since biochemical determinations are so important in assessing renal function, it should be recognized that in certain settings, creatinine and blood urea nitrogen (BUN) concentrations may be elevated without a decline in GFR. The administration of certain drugs, notably some of the cephalosporins[28] and co-trimoxazole,[29] as well as the presence of ketoacidosis,[30] may produce false elevations in serum creatinine concentration due either to interference with the assay for creatinine or to altered tubular secretion. BUN levels may be elevated because of an excessive intake of protein or an increased catabolic rate, as seen in sepsis and with corticosteroid treatment. These factors must be taken into account when evaluating the patient.

Finally, in the absence of prior knowledge of the patient's renal function, it may not always be clear whether the renal insufficiency is acute or chronic in nature, since the presentation may be exactly the same. In addition, many patients with underlying chronic renal disease will develop superimposed acute worsening of their renal function when stressed, as by dehydration or infection. This should be borne in mind throughout the evaluation.

History. In most settings, elucidating the cause of damage to the kidneys will be a relatively easy task based on history alone. An infant with a three-day history of vomiting and diarrhea who presents with oliguria and an elevated blood urea nitrogen level most likely has prerenal azotemia secondary to dehydration or perhaps vasomotor nephropathy. A six-year-old with a recent history of sore throat who presents with periorbital edema, hypertension, and gross hematuria is likely to be suffering from acute glomerulonephritis. Paradoxically, the most difficult patients to evaluate are often those who develop acute renal failure while under close observation in a hospital. Many of these patients have severe multisystemic disease, have been treated with a variety of nephrotoxic drugs, and have been subjected to numerous diagnostic and therapeutic procedures. In such cases, the records must be scrutinized for risk factors such as volume depletion, use of nephrotoxic antibiotics, recent radiographic studies using intravenous contrast, and other potential causes of renal injury.

A history of chronic renal disease may be suggested by previous abnormal urinalyses, poor growth, recurrent urinary tract infections, or alterations in urine volume. Many systemic diseases may involve the kidney and lead to ARF. A history of fever, rash, arthralgias, or gastrointestinal or pulmonary symptoms may be present in such patients, suggesting multisystemic involvement.

Examination. Physical examination must be thorough, with special attention to an assessment of the volume status. Tachycardia, dry mucous membranes, lack of tearing, poor peripheral perfusion—all suggest inadequate circulating volume and the possibility of prerenal azotemia. In older children, postural changes in blood pressure should be assessed. The patient who presents with acute renal insufficiency but who clinically appears to be experiencing volume overload may either have intrinsic renal disease such as acute glomerulonephritis or impaired myocardial function with inadequate renal perfusion. An intravenous fluid "challenge" might be appropriate in the oliguric, volume-depleted child, but not in the oliguric child with signs and symptoms of volume overload.

Other important findings on physical examination are those that might suggest a systemic disease as previously mentioned, notably unexplained fever, rash, or arthritis. Such findings might suggest nephritis related to systemic lupus erythematosis or Henoch-Schönlein purpura, or may be due to a drug-induced interstitial nephritis. The presence of enlarged kidneys or a palpable bladder would indicate an obstructive process of the urinary tract. Pallor might be indicative of poor perfusion or of severe anemia, as would be seen in hemolytic uremic syndrome or in underlying chronic renal disease. Chronic renal disease would be further suggested by abnormal growth parameters or visual evidence of rachitic bony deformities.

Urinalysis. Examination of the urine both microscopically and biochemically is valuable to aid in assessing the cause of acute renal failure. Urine dipsticks are useful as a screen but should be followed by microscopic examination of urine sediment. A positive reaction for blood may be due to hematuria but could also indicate the presence of free hemoglobin or of myoglobin. Mild proteinuria is common with renal injury of any sort, but heavy proteinuria should suggest glomerulonephritis. This would be further supported by a finding of granular and red blood cell casts on microscopy. In ischemic and nephrotoxic renal injury, a variety of cellular casts and debris may be seen, and eosinophiluria may be present in interstitial nephritis.[31] Crystals of calcium oxalate or of urate, when present in significant quantities, may be indicative of urolithiasis or of urate nephropathy.

"Urinary indices," as described by Miller et al,[32] may prove useful, particularly with an oliguric patient who may have either prerenal azotemia or vasomotor nephropathy. When presented with a threat to perfusion, normal kidneys respond in a well-defined manner. The urine produced is highly concentrated, and reabsorption of sodium is maximized. Urea clearance is impaired to a greater extent than is creatinine clearance, resulting in a disproportionately high urea nitrogen relative to serum creatinine concentration. With intrinsic damage to the kidneys, on the other hand, renal concentration ability is impaired, with the urine produced often being isotonic to plasma. Tubular absorption of sodium is impaired, resulting in an elevated fractional excretion of sodium (FENa). Both creatinine and urea clearance are severely impaired. Table III summarizes the urinary findings in prerenal azotemia and intrinsic renal failure. Several important points must be noted, however. In adults, a BUN to creatinine ratio of greater than 20 is considered evidence of prerenal azotemia. In children, however, because of their smaller muscle mass, normal serum creatinine may be 0.5 mg/dL or less; therefore, this ratio may be less valid. In addition, when evaluating urinary sodium excretion, it should be noted that the premature infant may have greater urinary sodium losses because of renal immaturity.[33] Ellis et al[34] and Mathew et al[35] have suggested that a FENa of less than 2.5% to 3% be employed as the cut-off in these infants, as opposed to 1.0% in term babies and older children. Zarich and coworkers[36] have also emphasized other exceptions to the usefulness of the FENa in separating prerenal and intrinsic ARF, most notably in patients with acute glomerulonephritis. Lastly, use of diuretics such as furosemide makes interpretation of FENa impossible, as urinary sodium will be elevated despite intravascular volume depletion.

Renal Imaging. Radiographic and ultrasonographic imaging techniques are sometimes useful in the diagnosis of acute renal failure in helping to confirm the clinical impression. The purpose of such studies is to obtain an estimate of renal size, determine if obstruction is present, and assess renal perfusion and function.

A plain film of the abdomen may provide information concerning renal size, if the outlines of the kidney are visible. Small, contracted kidneys suggest chronic renal failure, while large kidneys may be seen with obstruction or renal inflammation or infiltration. Calcifications along the path of the ureter may be seen, although they do not necessarily imply obstruction. Renal sonography is a particularly good means of screening for abnormalities such as hydronephrosis, as it is noninvasive and is easily performed at the bedside if necessary (Fig 2). Increased echogenicity of the renal parenchyma may be seen in a variety of intrinsic renal disorders but is a relatively nonspecific finding.[37] Intravenous pyelography adds little information in the evaluation of the patient with acute renal insufficiency. Poor renal function results in inadequate renal visualization, and there is a risk of further renal injury because of the nephrotoxicity of the contrast agent itself.[38] Radionuclide renal scans can add information that cannot be obtained via sonography, most notably information concerning adequacy of renal perfusion and an estimate of function.[39] Technetium 99m-diethylenetriamine pentacetic acid (DTPA) is used most often. With the exception of obstructive uropathy, however, these studies will not provide a definitive diagnosis. On a rare occasion, renal angiography may prove to be necessary in the event of a suspected renal venous or arterial lesion.

TABLE III. Diagnostic Indices in ARF*

Parameter	Prerenal	Renal
Urine osmolality (mOSm/kg H_2O)	>500	<350
Urine/plasma osmolality	>1.0	<1.0
BUN/plasma creatinine†	>20	<20
Urine/plasma urea†	>8	<3
Urine/plasma creatinine†	>40	<20
Urine sodium (mEq/L)‡	<20	>40
FENa‡§		
(U/P NA)/(U/P CREAT) × 100	<1%	>1%

* Adapted from ref 1, 32, 34, 35.
† Less valid in infants and small children.
‡ Urine sodium <20, FENa <1% in acute nephritis.
§ In neonates, FENa <2.5% in prerenal azotemia.

Renal Biopsy. Biopsy of the kidney can provide a definitive diagnosis of the cause of renal failure and may be indicated in certain circumstances. Definitive diagnosis may be important in the child in whom the diagnosis is obscure despite other investigations, in cases of acute glomerulonephritis not clearly related to streptococcal infections, and in cases in which resolution of the renal failure does not occur in the expected time frame. In each of these cases, biopsy may provide information to guide specific therapy or for prognosis.

MANAGEMENT OF THE CHILD WITH ACUTE RENAL FAILURE

Successful management of the child with acute renal failure requires meticulous attention to details of fluid and electrolyte balance, as well as nutritional and sometimes dialytic support. Potential complications should be anticipated and aggressively managed. When specific therapy exists for the underlying disease, this should be rapidly instituted, but it is not within the scope of this article to

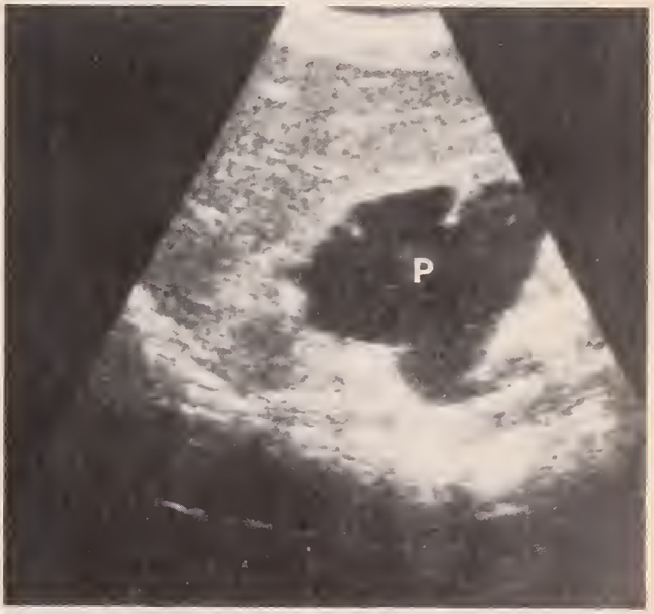

FIGURE 2. Renal ultrasound examination in a male infant who presented with urinary tract infection and an elevated serum creatinine. The renal pelvis (P) is hugely dilated. Renal function improved following antibiotic treatment and surgery for relief of obstruction.

explore this aspect of ARF. The same general principles of supportive care apply to all cases of ARF, regardless of the cause, and will be our focus here.

Fluids and Electrolytes. Close monitoring of fluid balance is crucial in the patient with ARF. The goal is to maintain the patient in a euvolemic state. Dehydration should be avoided to prevent further insult to kidneys that have already been injured, and fluid overload guarded against to decrease the risks of hypertension and circulatory overload. Replacement of insensible losses (an estimated 400–500 mL/m^2/day) in addition to replacement of ongoing losses (urine, nasogastric tube drainage, etc) will prevent excessive fluid gains. The patient who already has signs and symptoms of fluid overload will need more severe restriction. One should aim not for a stable weight but rather should expect a weight loss equivalent to 0.5% to 1.0% of the body weight in a patient who is consuming inadequate calories. If central venous pressure measurements are available, assessment of intravascular volume will be facilitated.

Diuretics may be helpful in some patients in the management of oliguria. While there continues to be controversy over the use of diuretics in ARF, it is clear that the oliguric patient can sometimes be rendered nonoliguric with the use of potent diuretics such as furosemide and mannitol.[40] The increased urine output makes the management of such patients less complex, but whether or not renal function or ultimate outcome is improved remains unclear. It would appear from animal studies as well as limited research in humans that prophylactic use of diuretics in high-risk settings (such as open heart surgery) may have a protective effect on renal function.[41–43]

Insensible losses contain little or no electrolytes and thus can be provided as 5% or 10% dextrose in water. Electrolyte losses in other body fluids may be more difficult to predict, and it may be helpful to measure the electrolyte content of such fluids and replace them with an intravenous fluid of similar composition. Hyponatremia in patients with ARF usually implies an excess of total body water rather than a deficiency of sodium. Further fluid restriction will usually suffice to correct the problem. Hypernatremia is not common in the absence of overzealous attempts to correct acidosis.

Hyperkalemia. This is a serious threat to the patient with ARF and may be present early in the course. Acidosis and an overall catabolic state contribute to the development of hyperkalemia. Potassium intake should be drastically restricted, and serum potassium levels monitored frequently. Moderate elevations of serum potassium that do not respond to restriction can be treated with sodium polystyrene sulfonate (Kayexalate®). A dose of one gram/kg body weight can be expected to lower serum potassium by about 1 mEq/L. Kayexalate® is provided as a suspension in sorbitol and can be given as a retention enema or by mouth or nasogastric tube. More severe elevations of serum potassium, especially if accompanied by changes in the electrocardiogram, require more urgent therapy. Calcium infusion (1–2 mL/kg of 10% calcium gluconate, given slowly with continuous electrocardiographic (ECG) monitoring), use of glucose and insulin (0.5–2.0 g/kg of glucose as a 50% solution followed by 0.2 units/kg of insulin), and sodium bicarbonate (1–2 mEq/kg) can be used to treat hyperkalemia. It is important to recognize that none of these treatments serves to remove potassium from the body; Kayexalate® must be given as well. Persistent hyperkalemia that is poorly responsive to these maneuvers should be treated with dialysis.

Acidosis. Acidosis is a common accompaniment of ARF. If mild, no treatment is necessary. Severe acidosis can be treated cautiously with sodium bicarbonate. Such therapy may be problematic in the patient with volume overload because of the concomitant administration of sodium. This again may be an indication for institution of dialysis.

Abnormalities of Calcium and Phosphorus. Hypocalcemia and hyperphosphatemia may occur early in the course of acute renal failure. Oral aluminum hydroxide therapy, 50 mg/kg/day in three or four divided doses, will lower the serum phosphorus level and usually lead to an increase in serum calcium as well. If hypocalcemia is severe or symptomatic, intravenous calcium gluconate may be administered cautiously.

Hypertension. Hypertension is often a problem in patients with ARF, particularly those with acute glomerulonephritis or the hemolytic uremic syndrome. If fluid overload is evident, water and salt intake must be decreased. Diuretics may be helpful in some patients. Severe elevations in blood pressure, particularly if associated with symptoms such as headache or vomiting, should be treated promptly with parenteral agents such as diazoxide, 3–5 mg/kg/dose intravenously, or hydralazine, 0.1–0.2 mg/kg/dose as an intravenous or intramuscular injection. If hypertension persists beyond two to three days, the patient can be switched to oral agents such as propranolol or hydralazine, starting at a dose of 1 mg/kg/day for either agent.

Nutritional Support. There is increased emphasis on nutrition in the recovery from ARF. Patients with ARF may be significantly catabolic, especially those in the postoperative period or following major injury. This will aggravate hyperkalemia, acidosis, and possibly increase the risk of serious sepsis. Adequate carbohydrate intake to allow for protein sparing must be provided. This amount is approximately 100–200 grams of carbohydrate/m^2/day. Proteins are usually restricted to about 0.5 g/kg/day in order to avoid worsening of azotemia, and that protein which is provided should be of high biologic value. It is obvious that providing adequate nutrition to the severely oliguric child will be a monumental if not impossible task. In the more critically ill child, such as the postoperative patient or those with infectious complications, it may be prudent to opt for earlier institution of dialytic support in order that more adequate nutrition can be provided.

Infections. Probably the most serious complication occurring during the course of ARF is infection. Patients with acute renal failure are at increased risk for serious infection due to prior surgical procedures, frequent utilization of invasive monitoring devices and urinary catheters, and possibly also to a depression of the immune system related to renal failure and an overall state of catabolism. Prevention is of course most crucial, with avoidance of unnecessary introduction of foreign bodies and careful sterile techniques. Suspected or documented infections should be treated vigorously. Potentially nephrotoxic antibiotics should be avoided if possible, and doses of all drugs adjusted if renal excretion is the principal means of drug elimination. Sepsis is a major if not the principal cause of death in patients with ARF.[44]

Dialysis. Indications for dialysis have been alluded to in the previous discussion, and these are listed in Table IV. In the small infant, peritoneal dialysis will be the simplest option and can in fact be performed without the need for specialized pediatric dialysis facilities. Hemodialysis will require suitable equipment and dialysis staff and thus is less widely available.

PROGNOSIS FOR THE CHILD WITH ACUTE RENAL FAILURE

It is not possible to assess an overall prognosis for children with ARF, since ARF is not a single entity. The prognosis will be influenced by a number of variables, most notably the natural history of the underlying condition, the setting in which the renal failure occurs, and the complications that ensue during the course of the illness. As an example of this, the child with ARF relating to acute postinfectious glomerulonephritis can be expected to recover fully in more than 90% of cases, whereas the development of ARF following surgical procedures or accompanying other serious medical illnesses carries a more ominous prognosis. It has repeatedly been noted in the adult literature that despite major advances in supportive care, mortality remains high and essentially unchanged from years past. This may partially be artifactual, in that critically ill patients are being kept alive longer, only subsequently to develop ARF in addition to multiple other complications.

In the pediatric age group, mortality shows a wide range, from about 8% to as high as 78%[1-3,5,6,45-47] depending on the population studied. It should be emphasized, however, that death in patients with renal failure is not always due to renal failure itself. In many cases, mortality is the result of other related complications such as sepsis or multiorgan failure. As an example of this, Rigden and coworkers[46] found an overall mortality of 63% in patients in whom acute renal failure developed following cardiopulmonary bypass surgery. Of these children, however, only two (8%) died of causes directly related to the renal failure itself.

The ultimate outcome for survivors of ARF will depend on the underlying disease process. Complete recovery, mild to moderate chronic renal insufficiency, and severe renal insufficiency requiring maintenance dialysis are all possible outcomes. Given the present advances in techniques for dialysis and transplantation in even small children, it is not unreasonable to be optimistic regarding long term outcome, even for those with residual renal injury.

The development of acute renal failure in a child presents an enormous diagnostic and management challenge to the physician. With the loss of renal function comes a vast array of biochemical and hemodynamic disturbances that underly the high morbidity and mortality of this condition. The impairment in renal excretory capacity further complicates the treatment of any coexisting disorders of other organ systems. An understanding of these disturbances and their consequences should enable the physician to anticipate and appropriately manage the patient with ARF, and thereby optimize the outcome for these children.

TABLE IV. Indications for Dialysis in Acute Renal Failure

Hyperkalemia unresponsive to conservative measures.
Fluid overload, especially if accompanied by hypertensive or congestive heart failure.
Severe hyponatremia or hypernatremia.
Severe acidosis.
Symptomatic uremia, with altered mentation, lethargy, nausea, vomiting.
Prolonged oligoanuria with inability to provide adequate nutrition.

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EXTRARENAL EXCRETORY METHODS IN KIDNEY FAILURE

Since renal failure is due to the retention of substances normally found in the urine, it is understandable that attempts to remove these noxious chemicals should be made by means other than through the damaged kidneys. All such mechanical efforts have been based on the use of some natural or artificial semipermeable membrane which retains threshold substances while permitting excretion of those materials inimical to the body's economy. Any technic for the removal of circulating diffusable substances should not only be effective but safe. Uncertainty as to safety, however, is not as great a contraindication to the employment of extrarenal excretory methods as might be supposed at first. Patients with renal failure are often desperately ill and in many instances doomed if treatment is not instituted. This justifies some calculated risk. The many individuals who have benefited, sometimes dramatically, by the various modalities to be discussed make it imperative that clinicians, regardless of training and specialty, be aware of the possibilities and limitations of these therapeutic technics. . . .

One advances the concept of therapy in renal failure with considerable trepidation. It is important that the physician define his objectives in each case. This demands not only clinical evaluation of the patient's condition and degree of distress—in many instances only symptomatic treatment is called for—but also, careful deliberation as to the advisability of prolonging life in hopeless terminal renal failure.

Recently I have employed the modalities under discussion more frequently than before and have experienced several, often unexpected, instances of gratification. Chronic nephritics who, often to my amazement, develop a degree of compensation which render them clinically well again, sometimes for years, are no rarity. The appreciation of patients and families for this attempt to help, even when the attempt is ultimately unsuccessful, is sufficient recompense for all the effort expended. . . .

HERBERT BERGER, MD
(*NY State J Med* 1958; 58:2663-2666)

The history of Kashin-Beck disease

LEON SOKOLOFF, MD

Kashin-Beck disease is an endemic osteoarticular disorder that incapacitates some two million persons in regions of northern China. In certain villages, the prevalence has been as high as 90% of the population. The condition first appears in the pre-adolescent or adolescent years, and leads to varying degrees of disability throughout adult life. Aside from the fact that it is thought to be of environmental origin, the etiology remains an enigma. The impact on public health and the economy of affected regions has been recognized by the Central Committee of the Chinese Communist Party and the Ministry of Public Health.[1] Many hundreds of papers about this disease have been published in the Russian, Chinese, and Japanese literature. Several reviews of the pathology, clinical manifestations, and current research are also available in English.[2–5] However, the subject has generated interest in the West only recently.

Kashin-Beck disease claims attention not only as a scientific challenge in its own right, but also for insights it may offer into more ubiquitous types of osteoarthritis. The latter are mechanically driven degenerative processes, but there are reasons to believe that they also have important biological dimensions.[6] The morbid anatomy of Kashin-Beck disease is unique: A single cell type in the affected tissues is selectively damaged either by an exogenous agent or by lack of a needed specific metabolite. The existence of a singular target implies that it may also operate in a broader reach of pathophysiology. At stake is a pragmatic question: Do environmental factors significantly contribute to banal forms of degenerative joint disease? This paper traces the discovery and early investigations of Kashin-Beck disease and probes historical factors involved in the slow pace of its elucidation.

HISTORICAL BACKGROUND

Skeletal remains indicate that the disorder goes back at least to the 16th century[7] and perhaps as far as the Bronze Age.[8] However, recognition of Kashin-Beck disease as an entity evolved gradually only after 1849. Its discovery is closely bound to the political and economic history of the area.

The disorder is most prevalent in Shaanxi and Heilongjiang provinces, but it also is found in a band that extends obliquely from the Siberian side of the Amur River in the northeast to as far as Tibet in the southwest (Fig 1). Most of our knowledge of the disorder unfolded in Siberia and Manchuria north of Shaanxi Province. Hegemony over these areas was long contested by Russia, China, and Japan,[9–11] but the tempo of these conflicts greatly accelerated during the second half of the 19th century.

Economic Geography. Southeastern Siberia, including what is now called the Soviet Far East or Maritime Province, is a vast territory that extends some 1,800 miles from Lake Baikal to the Sea of Japan. The region to the immediate east of the lake is called the Transbaikal. The terrain is largely mountainous and is rich in mineral, forest, and water resources.[12] The Amur River forms more than 1,000 miles of the frontier between Siberia and China. It arises from the confluence of the Argun and Shilka rivers approximately 800 miles east of Irkutsk, the historical city near the southwest edge of Lake Baikal. The Urov River is a smaller tributary of the Argun. All run in a generally eastward direction, and the Amur empties into the sea far north of Vladivostok. Vladivostok, the principal Pacific port, lies close to the southernmost end of the Maritime Territory. The latter projects into Manchuria and abuts the Korean peninsula.

Large deposits of coal, silver, gold, and other metals are present in this region and in time the ores became suspects in Kashin-Beck disease. A permafrost underlies the area

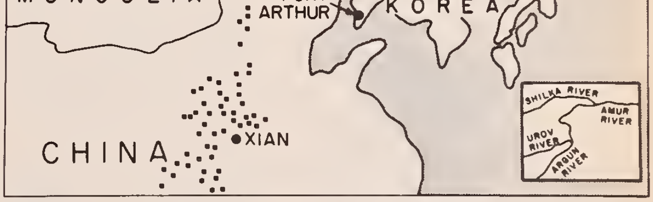

FIGURE 1. Distribution of Kashin-Beck disease. The representation is based on different periods of known incidence rather than a single time frame.

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and in warm weather only 1.5 meters is thawed. Although relatively little snow falls, winters are long and severe (October to mid-April). Summers are hot and humid. The banks of some of the rivers are marshy, the stagnation congenial to the malaria that once affected the area, and along the valleys, there are variably arable lands.

Some of the now large cities relevant to this story—Chita, Nerchinsk, and Blagoveshchensk—were founded in the mid-17th century, principally to process the ores. They were quite small for the next 200 years. Vladivostok, the principal port and industrial center, developed in the late 19th century. The Transsiberian railway ends here.

The aborigines of this portion of Siberia were largely nomadic tribesmen: Tungus, Orochen, and Buryat. Incursions of Manchus and Mongols are known to have taken place for many centuries and it must be presumed that admixtures of blood existed among them.

Manchuria is also largely mountainous and has huge timber and mineral reserves. Agriculture is possible mainly in a large central plain 1,000 feet above sea level. Harbin, the capital of Heilongjiang Province, is now the principal industrial and trading center. Although Harbin was an old city, it did not become important until 1896 when Russia was granted concessions and built it up. At the southern extremity of the Liaodang peninsula are two seaports often coveted because they are open all year: Dairen (now Lu-Ta) and nearby Port Arthur (now Lu-Shun). Extensive railway systems radiated from Harbin and Mukden (Shen-Yang) to these cities and also communicated with the Transsiberian Railway (Fig 2). The population of Manchuria is now largely Chinese but the Manchu sector was historically quite separate by language and tradition. There also were many Tungus inhabitants.

The Korean peninsula is delimited from Manchuria by the Yalu River and by the Maritime Province of the Soviet Union on the north. Like the immediate neighboring land, it is mountainous and rich in minerals. Arable land is sparse and largely confined to the more southern portions, remote from endemic areas of Kashin-Beck disease.

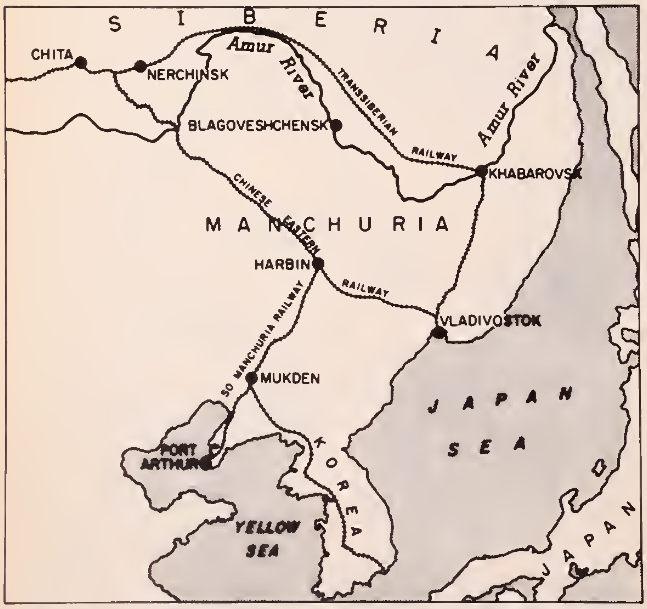


FIGURE 2. Political geography of Kashin-Beck disease (redrawn and simplified from Clyde[9]).

Shaanxi Province in north-central China has highland and plain areas. Xian, the ancient capital, is in the latter and is surrounded by farmland spared by the disease. The hilly regions are the seat of endemic foci. The soil there is a poor loess; the economy also is agricultural and the population distributed in villages rather than cities.

Imperialist History. Russian fur traders and Cossacks invaded eastern Siberia during the 16th and 17th centuries.[9,10] Before that time, the area along the Amur River was as unknown to the Chinese as to the Russians. Peter the Great sent Greek miners east of the Argun River to exploit the silver deposits in 1691. The tradition of exiling prisoners for forced labor there was begun by Catherine the Great. Major settlement was led by General Muraviyev in 1856. He was able to extract the Maritime Province from the Chinese in the 1860 Treaty of Peking. The treaty established the Amur River as the permanent boundary between China and the remainder of Siberia. Freeing of the serfs by Czar Alexander II in 1861 accelerated movement of large numbers of land-hungry peasants to the region from more western portions of Russia. The frontiers were guarded by Cossacks.

Construction of the Transsiberian Railway began in 1891.[9,11] Five years later, an agreement was struck between China and Russia to build a branch (the Chinese Eastern Railway) that would traverse Manchuria from Chita in Siberia and greatly reduce the distance to Vladivostok. Economic penetration of Manchuria increased and in 1899, the Liaodang peninsula of Manchuria and its ports were "leased" to Russia. In a short time, Russia had 20,000 men at Port Arthur, and Cossacks patrolled the railways under construction. By 1903, the railroads were extended to the southern ports and became more important than those to Vladivostok.

Meanwhile the imperialist ambitions of Japan in the area were being expressed. A war between China and Japan in 1894–1895 resulted in suzerainty of the Liaodang peninsula being awarded to the latter, but international pressure forced the Japanese to evacuate all of Manchuria several months later. There was also competition between Russia and Japan for military and economic control of Korea.

In 1904, Czarist troops seized all of Manchuria. The Russo-Japanese War of 1905 resulted in the nominal restoration of sovereignty over Manchuria to China but an "open-door policy" allowed Japanese control of the southern railways. The South Manchurian Railway Company created and maintained an educational system thereafter. One component was the Manchuria Medical College at Mukden. Its first tally in 1926 listed 118 Chinese, in addition to Japanese, medical students. The professors were Japanese and instruction was carried out in Japanese. The puppet state of Manchukuo was created in 1932, based on a putative Manchu as distinct from the Chinese constituency. Japan invaded further and further south until it was expelled from China, Manchuria, and Korea at the conclusion of World War II. Although Harbin was then briefly occupied by Soviet forces, there were close medical and political ties between the USSR and the People's Republic of China between 1950 and 1960.

The Cossacks. The earliest accounts describe Kashin-Beck disease in Cossack settlements along the Urov River (hence the frequently used name, "Urov disease"). It was not seen in other Cossack regions. Who were the Cossacks? Why were they affected?

Cossacks are not a linguistic or distinctive ethnic group. The earliest Cossacks were of Russian origins with a strong admixture of Tatar and other European stock.[10] They did not farm, but subsisted by fishing and fighting, either as mercenaries or as marauders. Later they were harnessed by the Czars as frontiersmen for the eastward expansion. Cossack status was given to people of other classes to hold new territories. In 1851, the Transbaikal Host was grafted off from older Siberian Cossacks west of Irkutsk. Twelve thousand of the Transbaikal Cossacks were settled along the Chinese border in 1857 and these gave rise to the Amur Cossacks. They were soon joined by 29,000 peasants and a small number of Tungus and Buryats. Living conditions were very difficult and many communities were decimated by starvation and malaria. Cossacks constituted one third of the Transbaikal population.

By the turn of the century, the Transbaikal Cossacks numbered some 60,000. They were not confined to military outposts; a proportion was distributed in villages with Russian and some non-Russian inhabitants. They were predominantly Slavic and had Russian names, but Beck[13] noted that many had Oriental features. Although there was considerable variation according to economic status, the general state of nutrition was superior to that of the average Russian. Horses and cattle did not thrive and served primarily as work animals. The manure was deposited close to the already stagnant waters (sometimes it was used for building dams to power the mills[14]), a custom that Beck thought might have causative significance.

Most persons affected by Kashin-Beck disease are Oriental. The possibility of a genetic basis was expressed by one Soviet investigator,[15] but another[16] pointed out the familial aggregation was being confused with heredity. Although a genetic predisposition to Kashin-Beck disease has recently been tested by class I histocompatibility typing,[17] there are no immunogenetic data relevant to shared Cossack and Asian ancestry. The already noted high frequency of Kashin-Beck disease in many villages constitutes *prima facie* evidence against its being inherited. Ethnic differences are sometimes observed in the prevalence in China today. In one village, Chinese inhabitants showed a high prevalence while Koreans escaped, despite the fact that they possessed a common water supply.[18] The preferred use of rice by the Koreans, rather than racial differences, was offered as an explanation for the phenomenon.

EARLY RUSSIAN INVESTIGATIONS

The *Transactions of the Imperial Free Economic Society* was a distinguished periodical founded in the late 18th century, and its successor is published to this day in Leningrad. During the 19th century, it regularly carried news about agricultural and other economic productivity in various parts of the world, including Russian settlements in Siberia and America. In 1849[19] a brief unsigned communication cited observations by corresponding member Yurenskiy of skeletal deformities in people living on the shore of the Urov River (Fig 3). Ivan M. Yurenskiy was a land surveyor in Nerchinsk, not a physician.[14]

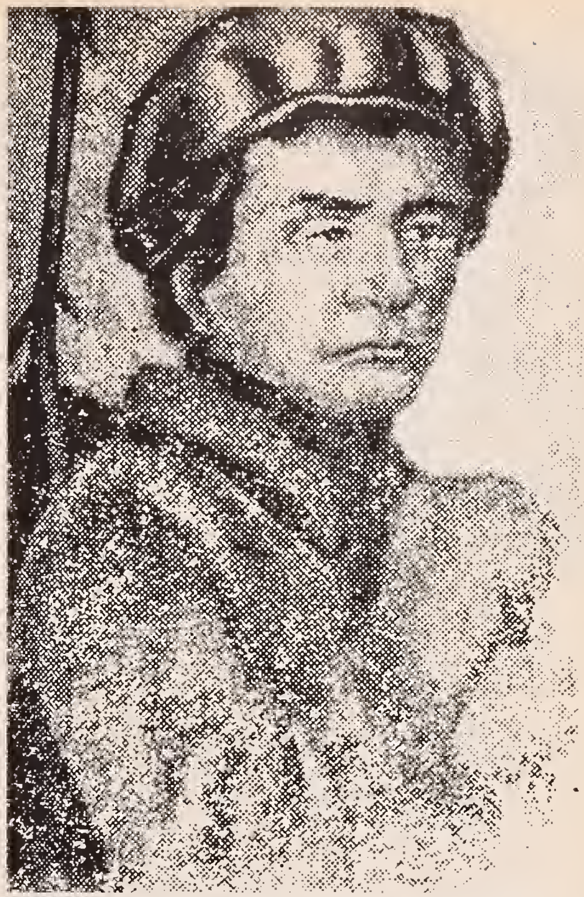


FIGURE 3. Ivan M. Yurenskiy (reproduced from Kravchenko,[14] courtesy of the National Library of Medicine).

In those afflicted, fingers were short, and bowing of upper and lower extremities and protuberances of the joints made work and ambulation difficult. These abnormalities were seen by the age of 20 years, and sometimes earlier. Longevity was not compromised. Unaffected girls from the Urov area, marrying at a young age and moving to other villages along the river, remained unaffected. By contrast, previously healthy persons developed deformities, albeit milder, several years after migrating to endemic foci. The pastureland in these areas was poor and the marshes were covered with rust. Sheep and a few horses were the principal domestic animals. They, too, had deformed extremities, and often were born without hooves; the appearance suggested what the English called rickets. The editorial commentator believed that goiter, endemic to the area, was the likely etiological factor, but acknowledged that Yurenskiy made no mention of mental deficiency as one would expect in cretinism. Speculation about the role of water dissolved from the snow in the higher portions of the mountains and stagnation of the air in the valleys was drawn from contemporary thinking about cretinism in the Alps. In any event, poverty, poor agricultural conditions, and disease made the Urov shores uninhabitable.

The first physician to investigate the condition was Nicholas I. Kashin (1825–1872) (Fig 4). (The name often is misspelled *Kaschin*, an inappropriate carry-over from the German transliteration of the Cyrillic.) He was born in central Russia but spent most of his professional career

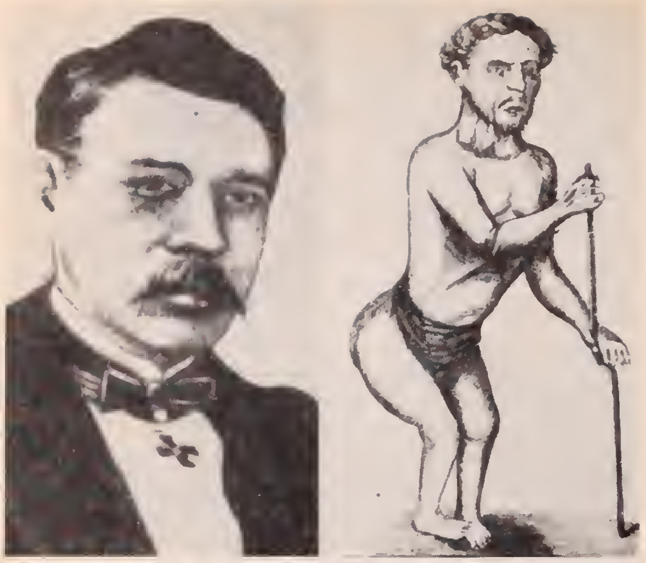


FIGURE 4. **Left,** Nikolai I. Kashin (reproduced with permission from S. G. Filatov, Ortopediya, *Travmatologiya i Protezirovaniye* 1963; 24(11):78, courtesy of the New York Academy of Medicine Library). **Right,** Kashin's drawing of Vassily Ivanov, a 50-year-old native Cossack, dated 1856 (reproduced with permission from N. N. Malinovskiy[20]).

in eastern Siberia. Kashin received his medical training in Moscow (1846-1851). His father, a feldsher, was unable to afford the tuition and Kashin served in the military for ten years to repay the costs of his education. He was the author of some 60 publications, two of which dealt with osteoarthropathy. It has not been possible to examine these original publications and the following account is reconstructed from secondary sources.[13,19-21]

Kashin was assigned to a Cossack brigade at Olochinsk on the Argun River. The administrative authorities were terrified by the crippling skeletal deformities in the Cossack villages. On April 24, 1856, he was directed to investigate the problem and relocate communities if necessary. The possibility that this might be an adult form of the English disease rickets was considered.[22] A month later, Kashin issued a report to the chief ataman of the Cossacks entitled "Description of endemic goiter and other diseases prevailing along the Urov River." Several villages were moved to other sites along the Argun River in 1857 but the medical consequences were lost to follow-up.

Following service in Siberia, Kashin worked at the Military Academy in Moscow in 1861 and wrote a doctoral thesis on a subject unrelated to Urov disease. Three years later, he returned to Irkutsk as a civilian medical inspector. There he dedicated himself to the health conditions of indigenous and settler communities of the Transbaikal. According to one biographer, he was a sympathizer of the Decembrist exile community in Irkutsk. Prominent among the diseases he encountered in Irkutsk were goiter and malaria; indeed, Kashin's analysis of the skeletal disorder was subsumed within his reports on endemic goiter.

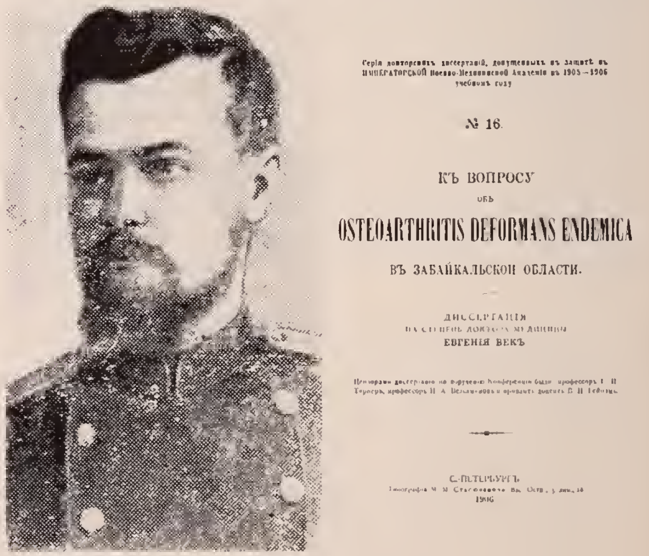
Kashin recognized that the deformities were distinct from those of goiter. One of his drawings shows a Cossack who had both a large goiter and short lower extremities, leaning on a walking stick (Fig 4). He regarded the skeletal abnormalities as one manifestation of complex systemic disturbances following prolonged fevers, scurvy, syphilis, and hereditary predispositions. Contributory factors included stagnation of the water, the high mineral and oxide content of the latter, and poor ventilation related to the still air of the valleys.

Following Kashin, there was a hiatus in investigation of Urov disease. It was rediscovered quite independently by Eugene V. Beck (1985-1916, Fig 5). Beck was educated in St Petersburg and became a military physician.[13] Graduated from the Medical Academy in 1893, he served at several posts and was assigned to east Siberia in 1895. The following year he went to the Transbaikal, first as junior physician in Chita, then as district officer of the Fourth Medical Department of the Transbaikal Cossack Army. There he was the sole physician to a population of 60,000, aided only by a staff of six feldshers and six practical nurses. A ten-bed hospital was available to him, but much of his effort was dedicated to a systematic epidemiologic survey of the endemic skeletal disorder.

The survey was carried out in 11 villages near Nerchinsk between 1899 and 1902. To avoid causing embarrassment, children were examined along with their entire families. Information elicited included the place of birth and time at which the individual migrated to the affected villages. Beck was assisted in the survey by his wife, A. N. Beck. Among 3,153 inhabitants examined, Beck observed 1,009 who had deformed bodies, thickened joints, and limping gait. He termed this *osteoarthritis deformans endemica*. The prevalence was as high as 46.5% in one village.

In October 1903, Beck received permission from the Chief Military Inspector to transfer six Cossack subjects, 16 to 23 years of age, to the Clinical Military Hospital in St Petersburg for detailed study. Systematic anthropological, radiological, and neuropsychiatric examinations were carried out.

The war with Japan intervened and Beck served as medical officer in the First Siberian Infantry Division. Following the war, he returned to the St Petersburg Academy and wrote his doctoral thesis based on the above investigations (Fig 5). It is a remarkable account of the entity as we



Серія докторскихъ диссертацій, допущенныхъ къ защитѣ въ
ИМПЕРАТОРСКОЙ Военно-Медицинской Академіи въ 1905–1906
учебномъ году

№ 16.

КЪ ВОПРОСУ

ОБЪ

OSTEOARTHRITIS DEFORMANS ENDEMICA

ВЪ ЗАБАЙКАЛЬСКОЙ ОБЛАСТИ.

ДИССЕРТАЦІЯ

ЕВГЕНІЯ ВЕКЪ

С.-ПЕТЕРБУРГЪ

1906

FIGURE 5. **Left,** Eugene V. Beck (reproduced from Kravchenko,[14] courtesy of the National Library of Medicine). **Right,** Title page of Beck's thesis, 1906 (reproduced with permission of the State Central Scientific Medical Library, Moscow).

now know it.[13] A precis of Beck's thesis was published in a Russian journal in 1906,[23] and a fuller version in a German one two years later.[24] Among the features distinguishing the lesions from the epiphyseal dysplasia of endemic cretinism were: inconstant association with goiter, relatively late onset, absence of delayed epiphyseal union, and the infrequency of arthritis in endemic hypothyroidism. Beck attributed the disease to poor drinking water but had no idea what the noxious agent might be. He became aware of Kashin's earlier work from old-timers who recalled the relocation of 1857. Only then was his wife able to track down the original reports of Kashin. A fundamental point of departure from Kashin's thinking was that the skeletal deformity was a specific entity rather than a manifestation of a multifactorial systemic disorder.

For many years following Beck's untimely death, Kashin-Beck disease was investigated only sporadically. The first conference of physicians of eastern Siberia attached to the University of Irkutsk created a commission to study the disease in 1925. Mrs Beck carried out anthropometric studies in two villages that summer and the following one.[25] They were designed to determine whether Kashin's view (systemic disease), or Beck's (specific skeletal disorder) was correct. She also initiated experiments on white mice to determine whether boiling water from endemic areas was an effective preventive measure. The thesis was that boiling would eliminate a possible infectious agent, but that if the agent were not infective, repeated evaporation of water in the same container would necessarily concentrate the causative factor and increase the risk. She did not complete the experimental study, stating in 1927 that it would be reported later by others.

Professor V. G. Shipachev of Irkutsk carried out epidemiological and experimental studies from 1926 to 1928.[26,27] He noted a dissociation of the frequency of goiter and the arthropathy as one moved from community to community along the Undi River. He viewed Kashin-Beck disease as a metabolic-endocrine disturbance of which the osteoarticular deformities were one manifestation. In his papers, one finds reference to the then recent discoveries of American nutritionists, particularly those of Elmer V. McCollum. Shipachev attached special importance to deficiencies of vitamins A and C and to mineral intake. Experiments were carried out on rabbits by feeding them vegetables from Irkutsk and water from the Undi River. Widespread endocrine and visceral abnormalities were found and interpreted to be like those in human tissues. Growth of the rabbits was retarded. Excessive porosity of bones and "vacuolization" of the epiphyses were reported but not articular abnormalities.

The fourth major period of investigation came with the establishment of the Urov Research Station in Chita in 1931. At that time, one third of the Transbaikal-Amur population was afflicted with Kashin-Beck disease, and 11–17% of recruits were rejected for military service because of it.[22] Creation of a Urov unit was authorized by the All-Republic Soviet of People's Commissars (now the Supreme Soviet) on May 15, 1931. The first director of the Chita Station was N. I. Damperov who wrote extensively on the subject from 1931 to 1939.[14] Collaborations were established with central research facilities in the Soviet Union. Elemental analyses of water, soil, foods, and plants were begun in 1935 at the Vernadsky Institute of Geochemistry and Analytical Chemistry in Moscow.[14] Low levels of calcium were found, as were excesses of strontium and barium, in studies reported through 1959.

Before 1932, knowledge of the pathologic anatomy of Kashin-Beck disease was confined to macerated bones and imperfectly fixed—usually surreptitiously obtained—postmortem specimens. In 1932, A. I. Nodov was assigned to examine the 50 skeletons in the collection of the Chita station.[21] His photographs reveal knee specimens with extensive eburnation and scoring characteristic of advanced osteoarthritis. Nodov obtained permission to carry out autopsies on several individuals in the presence of their families. His anatomic examinations were supervised by A. I. Abrikossoff, Director of the Institute of Pathology of the First Moscow Medical School. Abrikossoff was the best known Soviet pathologist of his day.

Nodov described the earliest changes in the joint cartilage as occurring in the matrix. The latter appeared swollen and pale. In these foci, the nuclei failed to take up stains. This chondrocytic necrosis was particularly marked at the junction with the bone. It was in these areas that fissures developed. Nonspecific fatty changes were present in the liver and other splanchnic organs. Nodov also carried out autopsies on 11 calves and dogs from Nerchinsk because livestock did poorly in endemic areas of Kashin-Beck disease. Articular manifestations were found in two age groups. In newborn calves, overt purulent arthritis, which we now recognize as infectious "joint ill," was found. In calves at the age of five or six months, failure to grow, associated with lameness, was found. Visceral lesions, like those of Kashin-Beck disease, were present, but in themselves nonspecific; chondronecrosis was not described. Nodules were seen along the ribs in some calves and suggested secondary avitaminosis to Nodov.

The comparative pathology of Kashin-Beck disease was—and still is—being pursued. Investigators at the Far Eastern Veterinary Institute in Blagoveshchensk were persuaded from 1951 to 1955 that Kashin-Beck disease does occur in many farm animals and barnyard fowl.[28] The skeletal lesions were marked by osteopenia and retarded growth, and were attributed to mineral deficiency. However, the characteristic zonal necrosis of Kashin-Beck disease was not described.

A new direction in research was initiated by F. P. Sergiyevskiy, the second director of the Chita station, in 1943. Attention was focused on the possibility that rotted grain might be the culprit. Because summers were short, grain was harvested early while it was green and moist, and therefore particularly susceptible to mold. In wet years, the incidence of Kashin-Beck disease increased; in dry years, it declined.[14] Yu. A. Rubenshtein, a research fellow in biochemistry and physiology at the Institute of Nutrition of the Academy of Medical Science in Moscow,[29] isolated toxigenic strains of *Fusarium sporotrichiella* Bilai var. *poae*. The organism is known in the western literature as *F poae* (Peck) Wollenwr. Experiments conducted from 1949 to 1959 in which grain infected with *F poae* from endemic areas fed to rats resulted in atrophy of the growth plate and osteopenic skeletons but not in the characteristic articular lesions of Kashin-Beck disease.[30,31] The mycotoxin theory held sway in the Soviet

Union at the time of Nesterov's review in 1964,[2] but now has no following there.

THE JAPANESE EXPERIENCE

Another pathway of discovery came with the Japanese entry into the area. In 1916, an army surgeon, Tomozo Okano, observed many people in the northernmost part of the Korean peninsula who had a chronic, progressive, deforming polyarthritis.[32] He was told by a local practitioner named Pak that the condition, called "Tojiru," had been known there for 300 years. The area was mountainous and famous for its pine forests. During the previous half-century, as the forests were cut down and housing improved, the arthritis had become less frequent. The cause was unknown, but suspicions centered about some harmful factor in the soil or contamination of the drinking water by resin from the fallen pine needles.

Okano, assisted by a military policeman and nurse, examined 603 inhabitants of two villages. He found that 62 had enlarged and painful knees, fingers, or elbows. All were farmers, often debilitated by prolonged fevers or weak constitutions. Finger grasp was poor, legs sometimes bowed, and pain forced some subjects to use a walking stick. In most instances, the initial manifestations dated back to the first or second decade of life. Other Japanese physicians visited the region on several occasions through 1928. However, most of the investigations were carried out in Manchuria.

Tokio Takamori was seconded from Kyoto in 1927 to become Professor of Medicine at the Manchuria Medical College in Mukden (Fig 6). Systematic studies of Kashin-Beck disease were carried out there until the Japanese were expelled from China in 1945. Their documents, other than what had already been published in the scientific press, were confiscated. Years later, Takamori's work appeared again in a posthumous volume that was published in English.[33] It noted that Kashin-Beck disease occurred in very limited localities in remote mountainous, impoverished areas. The condition was more often encountered in the elderly than in young people—evidence, perhaps, that construction of the railway improved living conditions and reduced the incidence. Takamori's term "Dysostosis enchondralis endemica" recognized the centrality of cartilage in the precocious union of epiphyses and deformity of joints. The bibliography includes references to 86 Japanese articles on the subject.

Numerous analytical reports on the composition of the water and soil of endemic Manchurian areas appeared in the Japanese literature during this period. Kentaro Hiyeda, professor of pathology at the Manchuria Medical College, found high concentrations of inorganic iron in the drinking water of endemic areas.[34,35] On the basis of experiments carried out on rabbits, he concluded that excessive intake of iron was the cause of Kashin-Beck disease. Although this hypothesis was often cited in the past, it has no standing currently.

Nobujiro Takizawa, professor of pathology at Chiba University, often visited Manchuria from 1941 to 1945. He was impressed by enlargement of the parotid glands of people living in the endemic regions. The histologic appearances of the salivary ducts suggested that aptyalism might be the pathogenetic mechanism of Kashin-Beck disease. Professor T. Ogata of Tokyo Medical School developed the idea that the parotid gland has endocrine as well as exocrine functions. Extracts of several "parotins" had certain biological activities including alteration of serum calcium levels. Takizawa sought to link the aptyalism to organic matter in the drinking water derived from decayed vegetable matter or putrid carcasses. Professor Noguchi, a geological chemist, examined water from 200 sources in Manchuria in this regard. The content of organic materials, gauged by biological oxygen demand, paralleled the morbidity of Kashin-Beck disease. He purified two substances from decayed plants, feruric and p-hydrocinnamic acids, and proposed them as causative agents of Kashin-Beck disease in 1967.

FIGURE 6. Tokio Takamori (reproduced from T. Takamori,[33] courtesy of the National Library of Medicine).

Takizawa believed that Kashin-Beck disease also occurred in Tokyo and the Aso volcanic district of Kyushu Island in southern Japan. His monograph[36] was published in Japanese in 1970, but his views are available to English readers in an earlier paper.[37] Takizawa's proposition caused considerable anxiety in Japan although it was rebutted by Takamori. The Ministry of Social Welfare created an ad hoc committee, chaired by K. Shigamitsu, to investigate the controversy in 1973. It supported Takamori.[38] The term *Aso volcanic disease* still is found in older bibliographies but no one now supports the idea that Kashin-Beck disease exists in Japan.

THE CHINESE EXPERIENCE

There was little Chinese literature on Kashin-Beck disease prior to World War II. According to one source,[39] it was first reported in 1908 in the Chang Bai Mountain area near Korea. Another[40] attributes it to Zhang Fengshu in Jilin Province in Manchuria in 1934. A monograph by a Chinese, Hung Pao-Yuan, emanated from Takamori's department and was published in Japan in 1945.[4] Hung ascribed the skeletal disease to acidosis resulting from excessive carbon monoxide levels in homes poorly ventilated during the long winters. He recommended the administration of a proprietary vitamin A preparation for prophylaxis and treatment of the condition.

A Chinese pathologist, Lin Chia-Chin, made the acquaintance of Takizawa when the latter visited Taiwan in 1957.[42] At Takizawa's urging, he surveyed 5,593 primary school children in Nantau Prefecture and concluded, on the basis of radiologic findings, that 5% of them had Kashin-Beck disease. Only youngsters born in the area or those living there at least four years were affected. Lin's report appeared in a Japanese journal rather than a Taiwanese one.[43] As in the case of Takizawa, Lin's idea of ectopic Kashin-Beck disease did not gain acceptance in his country.

International health programs of the USSR with its allies in the post-World War II era included research into Kashin-Beck disease. The People's Republic of China came into existence in 1950. The last document dealing with the collaboration of the two countries on Kashin-Beck disease appeared in 1956.[44] It had ten chapters by Soviet and Chinese authors, and went over much the same ground previously covered in each country. Particular emphasis was placed on the mycotoxicologic theories of Rubenshtein. A Bulgarian-Vietnamese team reported in 1971 that Kashin-Beck disease occurred in North Vietnam,[45] a semitropical country. The findings were atypical and there has been no further corroboration of the claim.

Since that time, there has been an enormous indigenous effort on the part of Chinese authorities to handle the problem.

CURRENT STATUS OF KASHIN-BECK DISEASE

Kashin-Beck disease still represents a serious impediment to the development of the affected area. The incidence in eastern Siberia diminished from 32% in 1906 to 8% in 1983.[3,46] The decline has been attributed to improved hygiene and the importation of food from other regions.[14] In China, the incidence has declined in many areas[47] but apparently has increased in some others.[48] In addition to the already mentioned apocryphal reports of Kashin-Beck disease in other parts of the Orient, one occasionally sees undocumented statements about its occurrence in Scandinavia. According to Bick,[49] Kashin-Beck disease was seen by American military physicians in Korea during the occupation after World War II. Its occurrence south of the 38th parallel may well have resulted from the massive uprooting of people from the northern regions during 1945 to 1947. So far as is presently known, Kashin-Beck disease is confined to limited areas of China, Siberia, and North Korea.

There no longer is any doubt that Kashin-Beck disease is an entity *sui generis*. Hidden in ongoing research, however, are two quite different concepts of how it comes about. One envisions the lesions arising from a defect in the quantity or quality of osseous tissue. The next phase of the process has not been spelled out. The implication is that the joint surface is compromised secondarily. The more tenable view is that the initial insult causes selective zonal necrosis of chondrocytes in the articular and epiphyseal plate cartilages.[3,48,50] Variable changes in the joints and growth disturbance are the consequence. In this scenario, elemental abnormalities would operate at some precise cellular target in the cartilages or the blood vessels that nourish them, rather than as components of mineralized bone matrix. Assuming that the second interpretation is correct, the bulk of evidence for the experimental reproduction of Kashin-Beck disease is not persuasive. A few impressive photomicrographs of chondrocytic necrosis in experimental dogs* have been published,[51,52] but most of the experimental lesions more closely resemble nutritional secondary hyperparathyroidism than Kashin-Beck disease.[53]

Two hypotheses have dominated research in recent decades: 1) Kashin-Beck disease results from abnormalities of trace elements or organic matter of soil and water; 2) mycotoxins, elaborated by rotted grains, act selectively on the cartilages. With few exceptions, these avenues were set out by early investigators. Epidemiological, geochemical, and laboratory studies have examined, with varying degrees of rigor, topics as diverse as carbohydrate metabolism, vasomotor tone, endocrine status, and cell membrane integrity. A notable exception to the preceding statement has been a large effort expended on the possibility that selenium deficiency is the major etiologic factor. This thought was derived from a partial overlap in the distribution of Kashin-Beck and Keshan disease. The latter is a cardiomyopathy first described in the Keshan region of Heilongjiang in 1935. It has only recently been discovered in the Transbaikal. The evidence for a relationship to seasonal lack of selenium is more firmly established for Keshan than for Kashin-Beck disease.

In China, three medical schools have Kashin-Beck disease research units: Harbin, Xian, and the Bethune school in Changchun. In addition, there are provincial medical and geochemical institutes wholly or partly dedicated to the subject, and special sections in 110 public health stations.[54] The premise of the Xian school is that selenium deficiency is the major culprit. The Harbin investigators pursue the view that toxins of *Fusarium oxysporum* are responsible for Kashin-Beck disease.[18] Attention has also been given to the role of humic acid in the ground water. Humic and fulvic acids are phenolic degradation products of decaying vegetable matter, particularly its lignin. Elevated levels are present in the well water of endemic areas according to Lin[55] and synergism with selenium deficiency has been postulated by one group.[56]

The main Soviet center of research is in Chita and clinical reports also appear from Vladivostok. There, emphasis is placed on abnormalities of trace elements. Rosin et al[57] reported the level of calcium in bone ash to be significantly low, while the relative content of strontium, copper, lead, zinc, manganese, iron, and silver was elevated in Kashin-Beck disease. Chronic phosphate intoxication, exaggerated by phytic acid, has been proposed by Voshchenko.[58]

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* Spontaneous chondronecrosis, similar to these experimental findings and resembling Kashin-Beck disease to varying degree, is common in growing dogs and livestock in America and western Europe.[3] The condition is well known in veterinary pathology under the term "osteochondrosis" but is not presently recognized in human medicine.

International agencies have recently entered the field. In 1985, four years after a preliminary review by two Japanese physicians,[54] the World Health Organization sponsored a conference on the subject along with the Ministry of Public Health in Beijing. Negotiations between China and UNICEF for field research were initiated in 1986, and final arrangements have now been concluded.[59]

SOME HISTORICAL PERSPECTIVES

It is puzzling that the cause of so massive an environmental disease has eluded detection for such a long time. The period of discovery coincided with the birth of concepts and scientific knowledge that revolutionized medicine throughout Europe, Japan, and America. The main academic centers of Russian medicine kept abreast of relevant advances in Western Europe. Beck's patients were examined radiologically just seven years after Roentgen's discovery of x-rays. The name Beck coined for the condition was in keeping with the primitive nosology of chronic joint diseases of the times. Introduction of the term "osteoarthritis" is attributed to J. K. Spender of Bath in 1888, but it did not then have its present connotation. Rudolf Virchow grouped both rheumatoid arthritis and osteoarthritis as "arthritis deformans." The first clear delineation of the two was made by the younger Garrod only in 1907,[6,60] shortly after Beck's thesis was published. Takamori's term, dysostosis enchondralis endemica, came closer to the mark insofar as it focused on the cartilage as the seat of the disorder.

Many of the early speculations about the etiology of Kashin-Beck disease—eg, impure water, stagnant air, unventilated housing—had previously been entertained in two other endemic disorders that also affected children, caused deformities, and restricted growth of the skeleton: cretinism and rickets. Russian thinking about rickets was similar to that elsewhere in Europe. Lack of sunlight was only one of several climatic factors regarded as important. The use of cod liver oil as a therapeutic measure had a considerable following elsewhere since the beginning of the 19th century and won its advocates in Russia by 1880.[61,62] The pediatrician N. Lunin pioneered research into mineral metabolism the same year. It took several more decades to establish that specific skeletal diseases might have a defined nutritional basis. The success of the germ theory during this period had a regressive effect in this field.[63] It favored the view that exogenous agents, rather than deficiency states, were the causes. Like beriberi, pellagra, and scurvy, rickets was sometimes considered an infectious disease. Beck differentiated osteoarthritis deformans endemica from rickets on clinical grounds. Rickets was frequent in the Transbaikal among the Buryats even though their nomadic existence necessitated exposure to the outdoors. The explanation now offered for this phenomenon was their tradition of keeping children within the yourta-tents and hence away from sunlight until they were two years old.[61,62]

Peculiarities of the water supply had been invoked as the cause of endemic goiter in different parts of the world for two millenia. A noxious agent in the vapor was considered likely even by Virchow. The definitive studies of David Marine on the requirement of the thyroid gland for iodine (1921) and of Elmer McCollum on vitamin D (1926) for the first time established nutritional deficiencies as the cause of specific types of dwarfism and skeletal deformity.

The moldy grain theory that dominated Soviet research during the 1940s must be seen in the context of the development of the science of mycotoxicology. Although ergotism had been known for many centuries, the possibility that other products of fungi might cause human disease was first suggested by Russian and Japanese scientists in the late 19th century. The field did not come into its own until mid-century with the growth of the antibiotics industry and recognition of the carcinogenic properties of aflatoxins. Yellowed rice toxicosis became known in Japan in 1891.[64] Alimentary aleukia was described in Russia in the late 19th century and a severe outbreak occurred in the Transbaikal and Amur regions in 1913.[65] It was a devastating endemic disorder of agricultural villages where overwintered cereals were consumed. Later studies showed that wheat and millet, harvested after the spring thaw, became contaminated by toxigenic strains of *F poae* and *F sporotrichiella*. Investigations of the same organisms were then extrapolated to Kashin-Beck disease.

Aside from the intrinsic scientific difficulties involved, three social themes suggest themselves in explaining the slow progress in unraveling Kashin-Beck disease. First, the affected areas were rural, impoverished, and remote from the main centers of medical knowledge. Unlike the aforementioned deficiency diseases that were solved, Kashin-Beck disease never came under the scrutiny of a larger medical research world. Early Japanese workers[32,33] were told by local residents that the incidence had decreased as living conditions improved, but systematic attention was given to Kashin-Beck disease only as more enlightened public health services became available and the economy advanced in the affected areas. Second, several observers commented that, despite the obtrusiveness of the deformities, affected persons did not regard themselves as diseased.[19,32,48] There may be an analogy here to the suggestion that rickets and osteomalacia were so common in medieval Europe that they were taken as normal.[63] Third, recognition of zonal chondronecrosis as the critical pathogenetic phenomenon in Kashin-Beck disease was delayed by the paucity of specimens for histologic examination at an early stage, unadulterated by secondary changes. Cultural proscriptions against necropsy were at play here. To this must be added the tenacity of old ways of thinking that concentrated on the bone rather than the cartilage.

There are indications of a dichotomy between empirical and rational strategies for the elimination of Kashin-Beck disease. Instances of the former started with Kashin's relocation of communities and extend most recently to hygienic measures contemplated by international health organizations. At the opposite pole is the reductionist gearing of contemporary medical science to the idea that precise understanding of cellular and molecular mechanisms leads to control of disease. It is too soon to predict which approach will succeed here. At times, environmental diseases, including another endemic form of osteoarthritis, Mseleni hip disease, disappear as societies change before their biological cause is elaborated.[66]

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Prevention of perinatal transmission of hepatitis B virus: Prenatal screening of all pregnant women for hepatitis B surface antigen*

Transmission of hepatitis B virus (HBV) from mother to infant during the perinatal period represents one of the most efficient modes of HBV infection and often leads to severe long-term sequelae. Infants born to mothers positive for hepatitis B surface antigen (HBsAg) and hepatitis B "e" antigen (HBeAg) have a 70%–90% chance of acquiring perinatal HBV infection, and 85%–90% of infected infants will become chronic HBV carriers.[1,2] It has been estimated that more than 25% of these carriers will die from primary hepatocellular carcinoma or cirrhosis of the liver.[3] These deaths usually occur during adulthood, when familial and financial responsibilities make them particularly devastating. In the United States, an estimated 16,500 births occur to HBsAg-positive women each year (about 4,300 of whom are also HbeAg-positive), and approximately 3,500 of these infants become chronic HBV carriers. Prenatal screening of all pregnant women would identify those who are HbsAg-positive and thus would allow treatment of their newborns with hepatitis B immune globulin (HBIG) and hepatitis B (HB) vaccine, a regimen that is 85%–95% effective in preventing the development of the HBV chronic carrier state.[2,5-6]

In 1984, the Immunization Practices Advisory Committee (ACIP) recommended that pregnant women in certain groups at high risk for HBV infection be screened for HBsAg during a prenatal visit and, if found to be HBsAg-positive, that their newborns receive HBIG and HB vaccine at birth.[7] No data are available regarding the proportion of high-risk women currently being screened in clinical practice, but several studies and the experience of public health workers indicate that major problems have been encountered in implementing these recommendations.[8-12] These include 1) concerns about the sensitivity, specificity, and practicality of the current ACIP guidelines for identifying HBV carrier mothers; 2) lack of knowledge among prenatal health-care providers about the risks of perinatal transmission of HBV and about recommended screening and treatment procedures; 3) poor coordination among medical-care workers who provide treatment and follow-up of mothers and infants; and 4) refusal of some public and private third-party payers to reimburse for HBV screening of pregnant women and treatment of their infants. In addition, concern has been expressed that these recommendations may not be practical or applicable in some US jurisdictions where HBV infection is highly endemic, such as parts of Alaska and certain Pacific Islands.

The problems encountered in implementing the currently recommended strategy of screening high-risk women have been examined by a number of investigators. Recent studies in several large inner-city hospitals, where all pregnant women were tested for HBsAg, have found that only about 35%–65% of HBsAg-positive mothers would have been identified by following the current ACIP guidelines.[8-12] In these studies, the prevalence of HbsAg in inner-city black (0.4%–1.5%) and Hispanic women was higher than expected. Several investigators expressed concern that many health-care providers are too busy or may be reluctant to obtain the sexual and drug-use history necessary to identify high-risk patients for screening. In addition, persons providing health care to pregnant women often are not aware of the risks of perinatal transmission of HBV and of the recommended screening and treatment guidelines. In one study, 40% of obstetricians could name no more than two groups at high risk for HBV infection, and only 28% knew the recommended treatment for infants born to HBV carrier mothers (CDC, unpublished data).

Given these limitations, it is now evident that routine screening of all pregnant women is the only strategy that will provide acceptable control of perinatal transmission of HBV infection in the United States. Screening the approximately 3.5 million pregnant women per year for HBsAg would identify 16,500 positive women and allow treatment that would prevent about 3,500 infants from becoming HBV carriers. Recent studies also indicate that the costs and benefits of universal testing of mothers are comparable to those encountered in other widely implemented programs of prenatal and blood-donor screening.[13,14] The cost of an HBsAg test ranges from an estimated $3.50 per test in blood-bank laboratories to $21.00 per test in private commercial laboratories. If one assumes an average screening cost ranging from $12.00 to $20.00 per test plus $150.00 for the HBIG and vaccine needed to treat each infant of an HBsAg-positive mother, the cost to prevent one newborn infant from becoming a chronic HBC carrier would be between $12,700 and $20,700.

HBsAG testing should be done early in pregnancy when other routine prenatal testing is done. The HBsAg test is widely available and can be added to the routine prenatal "panel" of tests without requiring additional patient visits. The advantages of making HBsAg testing routine during early pregnancy include 1) the ability to identify HBV carrier mothers that is not dependent on the health-care provider's identifying high-risk women or ordering HBsAg as a special test; 2) the availability of test results before delivery so that infants can receive HBIG and vaccine without delay after birth; and 3) appropriate counseling of families before delivery.[15]

Because more than 90% of women found to be HBsAg-positive on routine screening will be HBV carriers, routine follow-up testing later in pregnancy is not necessary for the purpose of screening. In special situations, such as when the mother is thought to have acute hepatitis, when there has been a history of exposure to hepatitis, or when particularly high-risk behavior such as parenteral drug abuse has occurred during the pregnancy, an additional HBsAg test can be ordered during the third trimester. Few women in populations at low risk for HBV infection will have a change in HBsAg status during subsequent pregnancies. However, because of the expected benefits of making HBsAg testing a routine part of each prenatal panel, testing should be done during each pregnancy.

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* These guidelines are reprinted from *Morbidity and Mortality Weekly Report* 1988; 37:341-346. The Council of the Medical Society of the State of New York (MSSNY) has adopted these guidelines as MSSNY policy on the recommendation of the society's Committee on Preventive Medicine.

Women who present for delivery without prenatal care or without medical records documenting the results of HBsAg screening should have the HBsAg test done as soon as possible after admission, since delay in administration of HBIG to infants of carrier mothers will decrease the efficacy of therapy. In the studies that demonstrated the highest efficacy (85%–95%) of combined HBIG and HB vaccine prophylaxis, HBIG was administered within 2–12 hours after birth.[2,4-6] In one study in which only HBIG was used for prophylaxis, no efficacy was found if HBIG was given more than seven days after birth, and a significant decrease in efficacy was observed if it was given more than 48 hours after birth.[16] Only one-third of US hospitals currently perform the HBsAg test as an in-house procedure, and many of these have technicians who are trained to do the test available on only one shift. Hospitals that cannot rapidly test for HBsAg should either develop this capability or arrange for testing to be done at a local laboratory or blood bank where test results can be obtained within 24 hours.

The commercially available HBsAg tests have an extremely high sensitivity and specificity if positive tests are repeated and confirmed by neutralization as recommended by the manufacturers of the reagent kits. Testing for other markers of HBV infection, such as HBeAg, is not necessary for maternal screening. Mothers who are positive for both HBsAg and HBeAg have the highest likelihood of transmitting HBV to their newborns. However, infants of mothers who are HBsAg-positive but HBeAg-negative may become infected and develop severe, even fatal, fulminant hepatitis B during infancy.[17,18] For this reason, HBIG and HB vaccine treatment of all babies born to HBsAG-positive women is recommended.

HBsAg-positive mothers identified during screening may have HBV-related acute or chronic liver disease and should be evaluated by a physician. Identification of women who are HBV carriers through prenatal screening presents an opportunity to vaccinate susceptible household members and sexual partners of HBV carriers, as previously recommended.[19] Screening and vaccination of susceptible contacts should be done by the family's pediatrician, primary health-care provider, or the physician evaluating the clinical status of the HBsAg-positive pregnant women.

Implementation of the recommendations to prevent perinatal transmission requires maternal screening, treatment of the newborn in the hospital, and administration of subsequent doses of HB vaccine to the infant during pediatric visits at one and six months of age. This multistep process requires effective transfer of information among several groups of health-care providers, knowledge of recommended treatment, and availability of HBIG and vaccine at separate facilities. Treatment failures due to lack of communication among health-care providers can occur, especially in situations where prenatal, obstetric, and pediatric care are provided in different facilities.[20] Central coordination of the treatment of these infants by city, county, or state health departments would improve the education of the health-care providers involved and increase the likelihood that proper treatment is provided.

In certain populations under US jurisdiction, including Alaskan Natives and Pacific Islanders, as well as in many other parts of the world, HBV infection is highly endemic in the general population, and transmission occurs primarily during childhood.[21] In such groups, universal vaccination of newborns with HB vaccine is recommended to prevent disease transmission both during the perinatal period and during childhood. Several studies have shown that HB vaccine given without HBIG will prevent 70%–85% of perinatal HBV infections and 95% of early childhood infections.[22,23] In many of these areas with highly endemic HBV infection, prenatal screening is impractical because the population is isolated, laboratory facilities are not available, and/or health-care budgets and personnel are limited. In these areas, control of HBV infection can be better achieved by directing available resources into programs to vaccinate all children with HB vaccine. Programs for screening all mothers for HBsAg and providing HBIG to infants born to carrier mothers are costly and will add only modestly to disease prevention. They should be considered only after the program for universal vaccination of children has been implemented.

RECOMMENDATIONS

All pregnant women should be routinely tested for HBsAg during an early prenatal visit in each pregnancy. This testing should be done at the same time that other routine prenatal screening tests are ordered. In special situations, such as when acute hepatitis is suspected, when there has been a history of exposure to hepatitis, or when the mother has a particularly high-risk behavior such as intravenous drug abuse, an additional HBsAg test can be ordered later in the pregnancy.

If a woman has not been screened prenatally or if test results are not available at the time of admission for delivery, HBsAg testing should be done at the time of admission, or as soon as possible thereafter. If the mother is identified as HbsAg-positive more than one month after giving birth, the infant should first be tested for HBsAg; if negative, the infant should be treated with HBIG and HB vaccine. Hospitals where infants are delivered should have HBsAg testing capabilities or should be able to obtain HBsAg results within 24 hours from a local laboratory.

If a serum specimen is positive for HBsAg, the same specimen should be tested again, and then the test results should be confirmed by neutralization. It is unnecessary to test for other HBV markers during maternal screening, although HBsAg-positive mothers identified during screening may have HBV-related acute or chronic liver disease and should be evaluated by their physician.

Infants born to HBsAg-positive mothers should receive HBIG (0.5 mL) intramuscularly (IM) once they are physiologically stable, preferably within 12 hours after birth. HB vaccine, either plasma-derived (10 μg per dose) or recombinant (5 μg per dose), should be administered IM in three doses of 0.5 mL each. The first dose should be given concurrently with HBIG but at a different site. If vaccine is not immediately available, the first dose can be given within seven days after birth. The second and third doses should be given one month and six months after the first. Testing the infant for HGsAg and its antibody (anti-HBs) is recommended at 12–15 months of age to monitor the effectiveness of therapy. If HBsAg is not detectable and anti-HBs is present, the child can be considered protected. Testing for antibody to hepatitis B core antigen (anti-HBc) is not useful, since maternal anti-HBc can persist for more than a year. HBIG and HB vaccination do not interfere with the routine childhood immunizations.

Household members and sexual partners of HBV carriers identified through prenatal screening should be tested to determine susceptibility to HBV infection and, if susceptible, should receive HB vaccine. Screening and vaccination of susceptible contacts should be done by the family's pediatrician, primary health-care provider, or the physician evaluating the clinical status of the HBsAg-positive pregnant women.

Obstetric and pediatric staff should be notified directly about HBsAg-positive mothers so that the neonate can receive therapy without delay after birth and follow-up doses of vaccine can be given. Hospitals, as well as state, county, and city health departments, should establish programs to educate appropriate health-care providers about perinatal transmission of HBV and its control through maternal screening, treatment of infants, and vaccination of susceptible household and sexual contacts of HBV carrier women.

Programs to coordinate the activities of those providing prenatal care, hospital-based obstetrical services, and pediatric well-baby care must be established to assure proper follow-up and treatment of infants born to HBsAg-positive mothers and other susceptible household and sexual contacts.

In populations under US jurisdiction in which hepatitis B infection is highly endemic, including certain Alaskan Native and Pacific Island groups, vaccination of all newborns with HB vaccine is the most effective strategy for HB control. In these populations, such vaccination programs should be given highest priority. In areas where HBsAg screening of mothers and use of HBIG in infants born to HBV carrier mothers are not practical, the vaccination of all newborns with HB vaccine should be considered the appropriate treatment.

Editorial Note: Hepatitis B vaccine is the first human vaccine that can prevent both serious chronic disease and a uniformly fatal type of cancer. These recommendations, developed in consultation with representatives of the American College of Obstetricians and Gynecologists and the American Academy of Pediatrics, represent a major step toward control of perinatal hepatitis B transmission in the United States. Programs for universal screening of pregnant women are currently in progress in Hawaii, certain Canadian provinces, Italy, West Germany, New Zealand, Australia, and Japan. More extensive infant HB vaccination programs are in progress in Alaska, American Samoa, Korea, Taiwan, Singapore, and the People's Republic of China. A number of US health-care facilities have already begun to screen all pregnant women for HbsAg.

State and local health departments can facilitate implementation of these recommendations by 1) working to assure that all women receiving prenatal care in both public and private sector programs are offered screening and appropriate treatment; 2) working to assure that costs of screening and treatment are covered by public and private third-party payers; 3) establishing programs to coordinate the transfer of information between prenatal, obstetric, and pediatric health-care providers; and 4) providing health education about hepatitis B to the public and to health-care providers. CDC will continue to work with state and local health agenices and professional associations in hepatitis B prevention and control.

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Cholestatic hepatitis after therapy with amoxicillin/clavulanate potassium

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Hepatotoxicity is an unusual complication of penicillin therapy. The pencillinase-resistant semisynthetic penicillins have been the most common form associated with liver injury, particularly oxicillin.[1-4] Carbenicillin has been reported to cause an anicteric hepatitis.[5] We describe a patient in whom cholestatic hepatitis resulted from therapy with amoxicillin-clavulanate potassium (Augmentin).

CASE REPORT

A 44-year-old woman was evaluated for abdominal pain, diarrhea, and jaundice. Thirty days earlier she had been administered a course of amoxicillin, 500 mg/clavulanate acid, 125 mg, for sinusitis. She completed a 12-day regimen. Eight days later burning epigastric discomfort developed that radiated to the right infrascapular area. The discomfort was unresponsive to antacids. Five days after the onset of the pain, dark urine and diarrhea appeared. Her physician noted jaundice.

The patient's medical history included three normal pregnancies and one dilation and curettage. She had a history of intermittent "heartburn," rarely used alcohol, had no history of transfusions, hepatitis, exposure to jaundiced persons, recent sexual activity, or intravenous drug use. She had a history of sulfa allergy, but no history of penicillin allergy. Previous screening blood tests one year earlier demonstrated no abnormalities.

On examination she appeared comfortable. She was slightly obese and icteric. Examination of the heart and lungs was within normal limits. The abdomen was soft with normal bowel sounds. The liver was palpated 4 cm below the right costal margin in the mid-clavicular line and was mildly tender. There was no splenomegaly. The rectal examination was unremarkable, and the stool was guaiac negative.

The patient's general course is represented in Figure 1. Her initial blood studies were as follows: total bilirubin, 1.9 mg/mL; alkaline phosphatase, 259 U/mL, aspartate aminotransferase, 129 U/mL; cholesterol, 304 mg/dL; white blood count, 6,700 mm^3; hemoglobin, 14.0 g/dL; hematocrit, 42.2%. She had no IgG antibody to hepatitis A virus. She was Hb_sAg, Hb_sAb, and hepatitis Hb_cAg negative. A heterophile test was negative. A cytomegalovirus titer measured one month after the onset of jaundice was less than 4. An abdominal ultrasound revealed a normal-sized liver without dilated ducts. No stones were seen in the gallbladder. A computed tomographic scan of the abdomen did not reveal hepatosplenomegaly or other structural abnormalities. An HIDA scan demonstrated no filling of the common bile duct. A repeat abdominal ultrasound performed two weeks after the onset of jaundice remained normal.

The patient's bilirubin and alkaline phosphatase counts peaked 38 days after she began taking the amoxicillin/clavulanate potassium at 4.6 mg/dL and 361 U/mL, respectively. Within four weeks the patient's bilirubin, alkaline phosphatase, and aspartate aminotransferase levels were within normal limits. The clinical picture was thought to be consistent with a drug-induced cholestatic hepatitis. The only drug to which the patient was exposed was amoxicillin/clavulanate potassium. It was not thought that additional studies or drug rechallenge would be appropriate.

DISCUSSION

Zimmerman[6] noted the rarity of liver damage from penicillins and semisynthetic penicillins. Oxacillin has been the most commonly implicated agent. A case of oxacillin-induced cholestatic hepatitis was reported by Pas and Quinn[7] in 1965, and 18 other cases of anicteric hepatitis have been reported.[1-4] A case of dicloxacillin causing cholestatic hepatitis was reported in 1986.[8] Two cases of cloxacillin and one case of floxacillin causing cholestatic hepatitis have been reported.[9-11] These agents have similar chemical structures, and are classified in the isoxazolyl penicillin group.[12] Carbenicillin has resulted in anicteric hepatocellular injury, but has not been reported to cause cholestatic hepatitis.[5]

Although cholestatic hepatitis or symptomatic liver injury has not previously been reported in relation to amoxicillin/clavulanate potassium, the striking clinical picture presented by this patient leads us to believe that this represents the first reported case of amoxicillin/clavulanate potassium-induced cholestatic jaundice. The onset of jaundice and duration of chemical changes resemble the occurrences with oxacillin, dicloxacillin, cloxacillin, and floxacillin. This report is particularly timely, as the use of amoxicillin/clavulanate potassium as a broad spectrum antibiotic has found great acceptance among primary care physicians.

Amoxicillin/clavulanate potassium is a new combination antibiotic of amoxicillin with the beta-lactamase inhibitor potassium clavulanate. Modest increases in the aspartate aminotransferase level occurs in less than 1% of patients using this antibiotic.[13] Both amoxicillin and the chemically similar ampicillin also have been reported to cause elevations of aminotransaminases.[6] There have been no reported cases of cholestatic hepatitis to our

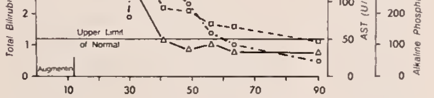

FIGURE 1. Patient's total bilirubin, aspartate aminotransferase, and alkaline phosphatase levels during a 60-day period.

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knowledge. Ampicillin has been associated with one case of fulminant hepatic failure in a patient with Stevens-Johnson syndrome. However, cephalexin was being used concurrently.[14] Clavulanate has not been reported to alter liver enzymes.

The mechanism by which penicillin-related antibiotics cause hepatic injury remains unclear. The rare occurrence in patients taking oxacillin and the other isoxazolyl penicillins, carbenicillin, and now amoxicillin/clavulanate potassium suggests that these reactions are idiosyncratic. The lack of fever, rash, and eosinophilia argue against this being a hypersensitivity reaction, and suggest it may be due to a hepatotoxic metabolite.[6] The occurrence in the combination antibiotic amoxicillin/clavulanate potassium may have particular significance. Clavulanic acid has a ring structure similar to that of penicillin (Fig 2), and this may enhance hepatic toxicity.[15]

This is the first reported case, to our knowledge, of amoxicillin/clavulanate potassium-induced cholestatic hepatitis. Further evaluations will determine if amoxicillin/clavulanate potassium has an increased likelihood of causing this side effect as compared to other penicillins.

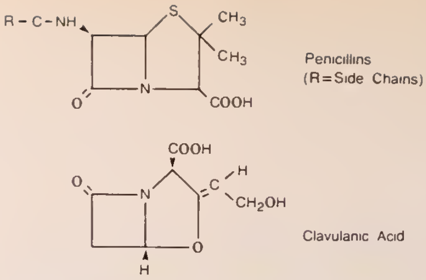

FIGURE 2. The molecular structure of penicillin and clavulanic acid.

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Use of gallium citrate Ga 67 scintigraphy in recurrent Merkel cell tumor

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Merkel cell carcinoma, a neuroendocrine carcinoma of the skin first described by Toker[1] in 1972, has been known to be particularly aggressive, with a 35% local recurrence.[2,3]

We report a case of Merkel cell carcinoma of the left elbow, diagnosed by excisional biopsy, managed with radiation therapy, and followed with gallium citrate Ga 67 (gallium 67) scintigraphy—documenting the usefulness of this method in detecting recurrence.

CASE REPORT

A 66-year-old woman was referred to the Department of Radiation Oncology for management of Merkel cell carcinoma affecting the left elbow. The patient was initially seen with an elevated, reddish-purple, smooth nodule measuring 2.2 × 3.2 cm. An excisional biopsy was carried out, and the histologic findings were of skin cancer arising from Merkel cells. On initial presentation, a wide, excisional defect with a healing skin graft was apparent with no evidence of tumefaction or regional adenopathy. Two months later, local swelling occurred and gallium 67 computerized scintigraphy was carried out; this disclosed several discrete, elevated, purplish tumefactions along the extensor surface of the proximal third of the forearm as well as avid deposits in the axilla and pectoral region (Figs 1, 2). The remainder of the scan displayed normal uptake throughout the thorax and mediastinum. There was no evidence of abnormal paralumbar or pelvic labelling. Roentgenograms of the left shoulder, humerus, and elbow failed to disclose any evidence of bone destruction. Skeletal scintigraphy performed with 99mTc hydroxydiphosphonate disclosed arthritic changes of the knees, vertebral column, and right sacroiliac joint; otherwise, there was no evidence of metastatic disease.

The patient was referred to the Department of Radiation Oncology. A rectangular, 9×7 cm^2 field with an equivalent square of 7.9 cm^2 was utilized with wet bolus technique maximizing the dose at the skin surface. Three thousand one hundred rads were delivered in ten fractions. On the last treatment day, the skin lesions were practically ablated. Within six weeks, the patient returned with recurrent tumor above the treatment site.

DISCUSSION

The Merkel cell is a nondendritic, nonkeratinocytic, epithelial, clear cell located in the basal layer of the epidermis. The neurotactile cell is capable of malignant transformation and has been

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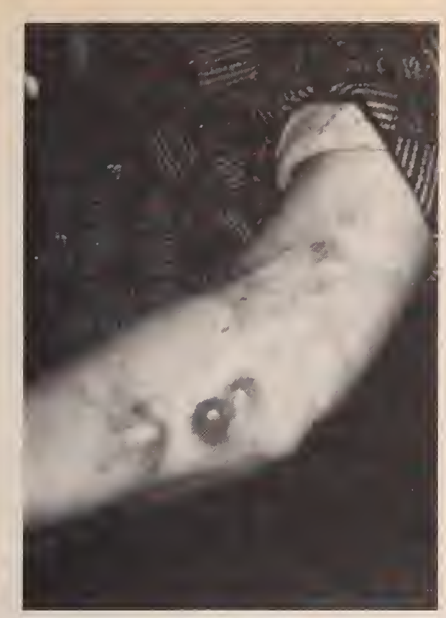

FIGURE 1. Discrete, elevated, purplish nodular lesions along the extensor surface of the left elbow.

reported as "trabecular cell carcinoma."[3] In 1972, Toker[1] first described this primary skin tumor as having a microscopic appearance of irregular anastamosing trabeculae and rosette arrangements of deeply basophilic tumor cells. These irregular sheets or trabeculae frequently were observed to infiltrate the collagen fibers of the dermis and the subcutaneous adipose tissue.[4] The richest source of Merkel cells is the skin of the neck and abdomen. The tumors are cellular, with a high mitotic rate.

The histologic features of this tumor can be difficult to differentiate from other small-cell undifferentiated tumors that occur in the skin, either primary or metastatic. These tumors can resemble lymphoma, carcinoid, oat cell carcinoma, sweat gland carcinoma, and neuroblastoma on light microscopy[3]; electron microscopy is therefore essential for the diagnostic confirmation of the Merkel cell tumor. Examination of the ultrastructure of Merkel cell tumor cells shows membrane-bound, dense-core, neurosecretory granules peripherally in the cytoplasm.[3] There is some variation in the size of these granules. However, they are morphologically identical to the membrane-bound granules seen in cells differentiated from neurocrest or amine precursor uptake decarboxylase (APUD) cells, and thus support the concept that trabecular cell carcinoma is derived from neurocrest origin.[4]

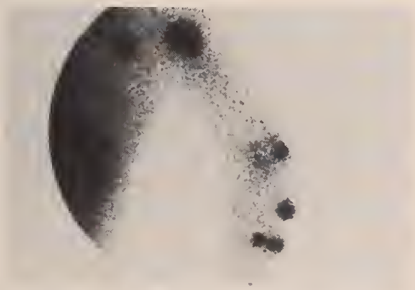

FIGURE 2. Seventy-two hour gallium citrate Ga 67 scan showing avid deposits corresponding to the nodules as well as metastatic deposits in lymph nodes in axilla and pectoral region.

Since Edwards and Hayes,[5] in 1969, first introduced the clinical use of gallium 67 for the detection of soft tissue tumors by imaging techniques, there have been numerous reports on the use of this radiopharmaceutical in the demonstration of a variety of tumors, and their radiochemistry and cellular localization. Gallium 67 has been the most widely used tumor seeking agent, and the literature has documented its effective role in the demonstration and management of lymphomas, occult primary lesions, and metastatic lesions.[6-8] The precise mechanism of gallium 67's localization in malignant lesions is unknown. Considerable evidence suggests that the mechanism of gallium 67 in tumors is complex. Multiple factors contribute to its localization in tumors based on adequate blood supply, the presence of specific binding proteins (lactoferrin or ferritin), increased tumor membrane porosity, local pH change, and histiocytic content of tumor, to mention a few.

Gallium 67 is predominantly localized as amorphous electron dense bodies in single membrane-bound, lysosome-like cytoplasmic organelles and to a lesser extent in the nuclei and cytoplasm.[9] A relatively specific ultrastructural finding for Merkel cell tumor is the presence of perinuclear fibrillar whorls on electron microscopy (Fig 3). Identification of these perinuclear structures allows differentiation of trabecular carcinoma from the most common neuroendocrine tumors that metastasize to the skin of the head and neck and have similar morphology to the Merkel cell carcinoma.[10] Additionally, a notable cytologic feature of the Merkel cell tumor is a finely dispersed chromatin in round or oval nuclei with a sharply defined nuclear membrane.[2] Trabecular cell carcinoma usually occurs as a solitary tumor in middle-aged to elderly individuals and is found equally in men and women.[11] Meland and associates[3] have reported that in 23 patients the tumor occurred most often in the head and neck region and lower extremities. The upper extremity tumor location in this case has been reported to be a less common site, along with the region of the trunk. Characteristically, the Merkel cell tumor involves dermis, with sparing of the epidermis. Lymphatics were the most common metastatic site, with liver and lung being frequently affected visceral areas.[3] On microscopic evaluation, the histologic features of the skin tumor and the metastatic tumors in the lymph nodes are identical.[4]

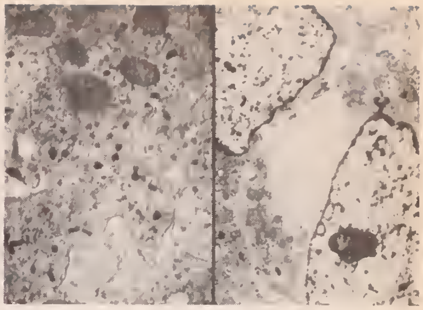

FIGURE 3. Left, low power (X13,500); Right, high power (X33,750) electron microscopy showing membrane bound granules with dense core in tumor cells.

No method of treatment is entirely successful, although local excision and regional lymph node dissection for palpable nodes have been used most often. Chemotherapy has not prevented mortality in extensive disease, although adjunctive radiotherapy has sometimes been helpful.[11]

Since this neoplasm has a high propensity for local as well as lymphatic and hematogenous metastases, follow-up studies using gallium citrate Ga 67 scintigraphy could lead to earlier diagnosis of recurrence, with improved survival.

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Perforation of a gastrojejunal ulcer into the pericardium

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Pneumopericardium most commonly results from penetrating or blunt chest trauma, spontaneous formation of gas from anaerobic bacteria, extension of pericardial disease into the neighboring organs, involvement of the pericardium by diseases of adjacent organs, and iatrogenic causes. Pneumopericardium rarely results from fistula formation from the gastrointestinal tract below the level of the diaphragm. We describe here a patient with a gastrojejunal anastomotic ulcer complicated by pneumopyopericardium and discuss the diagnosis and therapy of this entity.

CASE REPORT

A 31-year-old man was admitted to the University of Istanbul Cerrahpasa Hospital complaining of dyspnea, weakness, nausea, vomiting, and a sharp, constant pain between his scapulae. The patient had a surgical history of an antrectomy and Roux-en-Y gastrojejunostomy for peptic ulcer disease three years prior to admission. He had complained of mild mid-epigastric pain during the ensuing period. However, he had not needed medical treatment. Four days prior to admission, he had had a three-hour episode of severe chest pain with associated dyspnea and frothy vomitus. The patient denied any history of blunt trauma, fever, or chills.

On examination, the patient was afebrile with a regular pulse of 120/min and a bilateral blood pressure of 90/70 mm Hg. His skin was cold and his jugular veins were distended to the angle of the mandible. The heart sounds were diminished, and a sound of fluid turbulence was audible over the precordium. The liver was palpable four centimeters below the right costal margin in the mid-clavicular line.

The hematocrit was 35% with a leukocyte count of 6,800 cells/cm^2. The remaining laboratory results were remarkable for a serum aspartate transferase (SGOT) of 2,755 U/L, a lactate dehydrogenase (LDH) of 2,725 U/L, and a creatinine phosphokinase (CPK) of 82 U/L. An electrocardiogram demonstrated a sinus tachycardia, low voltage in all leads, and ST segment elevation in leads I, II, aV$_L$, and V$_{2-6}$. The chest roentgenogram (Fig 1) revealed an air-fluid level within the pericardial space.

The central venous pressure was found to be 21 mm H_2O. The patient's blood pressure remained at 90/70 mm Hg without evidence of deterioration. Therefore, further investigation into the etiology of the pericardial effusion was conducted. An echocardiogram showed fluid and air anterior and posterior to the heart. An abdominal ultrasound demonstrated hepatomegaly, gallstones, and dilation of the intrahepatic veins and the inferior vena cava. An upper gastrointestinal study with meglumine diatrizoate failed to demonstrate the pericardial fistula. A computed tomographic (CT) scan of the region showed air in the pericardium with associated posterior displacement of the heart. No fistula was identified.

On the third hospital day, a superventricular tachycardia was observed and controlled with verapamil. On the eighth hospital day, a fever developed, with a temperature of 38.5°C, and leukocytosis of 16,700 cells/cm^2. Physical examination was notable only for increased dullness over the left hemithorax. A left thoracentesis yielded 300 mL of yellow-green, foul-smelling fluid. The white cell count of the fluid was 12,500 cells/cm^2, and examination of the smear showed gram-positive and negative bacilli and gram-positive diplococci. Cytologic examination revealed no malignant cells. Cultures of the fluid grew *Proteus mirabilis.* Cefazolin and tobramycin therapy ensued, with a resultant decline in the temperature and leukocyte count. On the 11th hospital day, profuse gastrointestinal bleeding developed which necessitated an emergency celiotomy. A perforated ulceration at the site of the gastrojejunal anastomosis was found penetrating through the pericardium and into the left ventricular myocardium. Bleeding was noted at this site, and following an unsuccessful attempt at primary repair, the patient died.

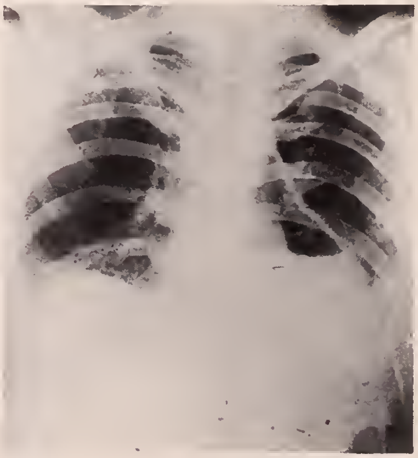

FIGURE 1. Air-fluid level within the pericardium demonstrated on a posteroanterior chest roentgenogram.

At autopsy, a gastrojejunal anastomotic ulcer which had eroded into the diaphragm, liver, and pericardium was identified. The pericardial space was filled with blood and pus, and there was a peptic ulceration through the left ventricular wall leading to the fatal gastrointestinal and intraperitoneal hemorrhage.

DISCUSSION

Pneumopericardium is rarely caused by a fistula from the gastrointestinal tract below the diaphragm. Gastropericardial fistulas formed as a result of perforated benign gastric ulcers[1] and gastric carcinomas[2,3] have been reported. Due to the nature of fistula contents, these patients usually present with pneumopyopericardium. Two cases of gastrojejunal anastomotic ulcers complicated by pneumopyopericardium have been reported previously.[4,5] Both patients had Zollinger-Ellison syndrome, and both died as a direct result of the pneumopyopericar-

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dium. It has been suggested that a high gastric resection may result in a gastrojejunopericardial fistula secondary to adhesions around an old anastomotic ulcer.[5]

The patient reported here fits this pattern. Adhesions were encountered between the gastrojejunal anastomosis, diaphragm, and liver, which apparently channeled the contents of the perforated ulcer into the diaphragm and pericardium. A fibrinopurulent pericarditis resulted, which led to left ventricular peptic lysis and myocardial perforation. The peritoneum was bypassed, thus accounting for the remarkable lack of peritonitis.

On physical examination, a millwheel murmur is often audible in pneumopericardium complicated by a pericardial effusion.[6,7] Some authors have noted that in the absence of a substantial amount of fluid, a metallic crackling cardiac sound is often heard.[8] Roentgenographic findings include air within the pericardium with or without an air-fluid level, enlargement of the heart shadow, and evidence, with appropriate contrast studies, of a fistula between the pericardium and neighboring organs.[5,6]

The important lesson to be learned from this case is that the presence of an air fluid level within the pericardial cavity must be treated as a surgical emergency. Inability to demonstrate a fistula on roentgenographic investigation should not preclude the diagnosis of this entity, since only early and aggressive surgical therapy will allow successful resolution of the problem. Additional diagnostic studies which, in this case, were not performed, including upper endoscopy, could help define the pathologic process. If the etiology of the pericardial fluid is not clearly diagnosed by available means, then a pericardial window should be performed for diagnostic as well for therapeutic reasons. As with the patient described here, a delay in performing this procedure because a clear etiology for the pyopneumopericardium is not found can result in a poor outcome. Once a fistula is recognized, exploration should be initiated to debride the region and repair any perforations. The pericardial sac should be irrigated and fluid drainage ensured. If necessary, the wound should be left open. Secondary closure of the wound can be attempted after the suppuration subsides.[9,10]

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Postoperative fistula following insertion of a penile prosthesis in a diabetic patient

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Infection following the surgical implantation of a prosthetic device is an ominous event. Meticulous attention to detail and strict sterile technique are mandatory to prevent contamination.

When infection occurs in the presence of a penile implant, the prosthesis must be removed. If reinsertion is desired, this can only be done after subsequent healing with scar formation and fibrosis. In addition, the second surgical procedure is usually met with extreme technical difficulties, as the tissue planes have been obliterated by excessive scarring.

We present a case of bilateral cavernoso-cutaneous fistulas caused by infection following the implantation of an inflatable penile prosthesis, which was successfully treated without removal of the prosthesis.

CASE REPORT

A 42-year-old patient with diabetes mellitus, who required 30 units of NPH insulin daily, received an inflatable penile prosthesis (AMS 700) in March 1981, for the management of organic impotence. The prosthesis had functioned well for approximately five years, but it then failed to inflate.

On surgical exploration through a suprapubic incision, the right cylinder was found to have a 2-cm longitudinal tear. New replacement cylinders were inserted without difficulty, after which the prosthesis inflated easily and functioned properly. During the procedure, both the wound and the replacement parts were irrigated with copious amounts of kanamycin and bacitracin solution. The wound was closed in several layers. In the postoperative period, the patient received ampicillin, 1 g every six hours, and gentamicin, 80 mg every eight hours, for three days. He was discharged on the fourth day on oral ampicillin, 500 mg every six hours for ten days, after an uneventful recovery except for minimal ecchymosis in the suprapubic region.

Three weeks later, the patient returned for his first follow-up visit with a 5-mm separation present in the middle of the suprapubic incision. There was a purulent discharge from this opening. He was immediately placed on oral cephradine, 500 mg every six hours. Once a day irrigation of the wound by the physician was started on an outpatient basis using a solution of gentamicin (80 mg), polymixin B (100 mg), and neomycin

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(500 mg) per liter of sterile water. Culture of the purulent material grew *Pseudomonas* and *E coli*. After each daily irrigation, the wound was packed with iodoform gauze.

The purulent drainage gradually became clear and decreased in amount with continued irrigation. Repeat cultures were negative. While irrigating the wound, the penis was noted to become engorged and erect. A sinogram was obtained with contrast surrounding both cylinders and the valve mechanism (Fig 1). The daily irrigations continued for about eight weeks as well as the oral cephradine regimen until eventually the wound completely healed. Twenty months after complete closure of the fistulas, the patient was using the prosthesis with complete satisfaction. He is not maintained on suppressive antibiotics, but is examined on a regular basis for any signs of recurrent infection.

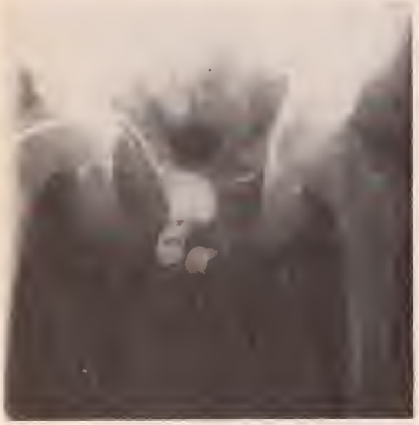

FIGURE 1. Sinogram demonstrating contrast surrounding both cylinders and valve mechanism.

Discussion

Mechanical failures still account for most of the replacements of penile prosthetic devices, with reports stating an 12% to 80% incidence.[1-3] Wound infections account for an additional 0.5% to 3.1% of complications.[1-3] In most cases, when infection is present the prosthesis must be removed for the healing to proceed.[4]

Occasionally, the corpora may be drained to promote healing.[5] Replacing the devices later requires extensive sharp dissection, which is usually met with extreme technical difficulty.[2] Most recently a rescue procedure, involving removal of the infected prosthesis and simultaneous replacement with a new system, has been reported with a success rate of 83.7%.[6] Only patients with a non-eroded, infected device may be considered for this alternative form of salvage.

Patients prone to infection, especially diabetics, should be warned of this possibility. Diabetic patients are known to have slow healing wounds and decreased blood flow due to small vessel disease. Gentle handling of tissue, meticulous hemostasis, and antibiotic therapy are required to prevent complications. Surgical complications include scrotal hematomas, penile ecchymosis, and extrusions due to necrosis from tissue ischemia by a prosthesis of incorrect or too large a size.

This case represents the first published report to our knowledge of conservative management of a postoperative fistula that occurred following reinsertion of a penile prosthesis in a diabetic patient. In certain cases, an alternative form of conservative therapy with daily wound care by the patient and surgeon is required to avoid a potentially disastrous result that is frustrating to both the patient and physician.

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Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Buttocks blow barotrauma and pneumomediastinum

TO THE EDITOR: The case report in the August 1988 issue of the *Journal* of cocaine-induced pneumomediastinum possibly associated with a Valsalva maneuver[1] reminded us of a patient who had also experienced pneumomediastinum by an unusual mechanism.

A 16-year-old female, without any prior cardiac or pulmonary history, came to the emergency room with progressive substernal pain which had begun 12 hours before while she was sitting on her bed. It worsened with deep inspiration. She denied any history of cigarette or drug use and was on no medications. She had had no recent coughing or hemoptysis.

The initial examiners thought a pericardial rub was present and a cardiology consultation was requested. The Hamman sign (mediastinal crunch with cardiac systole) was promptly detected over the anterior precordium with minimal subcutaneous emphysema below the left breast. The electrocardiogram revealed sinus rhythm and normal morphology. The electrolytes and complete blood count were normal. A chest roentgenogram was obtained, confirming the presence of pneumomediastinum but showing no pneumothorax. An additional history revealed that the patient had experienced an unexpected mishap 24 hours previously. She suddenly slipped while walking on ice, causing her to land directly on her buttocks. She then experienced a sudden forcible exhalation and subsequently had the gradual onset of pain.

The patient's chest pain resolved within 36 hours on bed rest and conservative management. She had no recurrence of symptoms over the following year.

Based on the patient's description, a Valsalva mechanism is postulated as the etiology of her pneumomediastinum. Although falls on ice would seem an infrequent cause of pneumomediastinum, this case reinforces Valsalva as a possible mechanism. Thus, we would add sudden blows to the buttocks with resultant Valsalva as another cause of chest pain and pneumomediastinum.

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Needle localization of occult breast cancer and subsequent management

TO THE EDITOR: The paper by Abel and Deitch[1] in the November 1988 issue of the *Journal* on needle localization of occult breast cancer makes several valid and important points. We agree with its central themes, and would like to see most of the diagnostic methods described enter widespread practice, both to save lives, and also to minimize cosmetically unpleasant effects from the biopsy of lesions that are ultimately proven histologically to be noncancerous. However, two areas require rebuttal.

First, modified radical mastectomy is not the only treatment of choice for breast cancer. Careful prospective studies have established that the combination of breast segmentectomy (wide-margin excisional biopsy), axillary dissection, and radiotherapy to all residual ipsilateral breast parenchyma is associated with as good results as mastectomy in the patient who meets selection criteria.[2–4] Many women with newly diagnosed breast cancer meet these criteria. For them, the probability of curing cancer is not jeopardized, and the physical, social, and psychologic after-effects of surgery are substantially lessened. Thus, the authors' appropriate action on behalf of cosmesis can be safely extended to many patients with malignant diseases as well.

Second, we object to the end of the statement, "the best hope for cure remains early detection by mammography and appropriate local treatment." The benefits obtained by following local surgery/radiotherapy with adjuvant (systemic) therapy (well-tolerated hormone therapy, or tolerable multiagent chemotherapy) have been well established in even the best prognostic subgroups of node-negative women.[5–8] The historic impetus to trials of adjuvant therapy was an unacceptable rate of treatment failure, ie, development of metastases and death from breast cancer, in patients whose local disease was well treated but whose (initially undiagnosable) systemic disease ultimately spread. This sequence occurred in some three-fourths of node-positive women treated with surgery alone, and in a fourth of node-negative patients.

Patients with positive nodes are statistically unlikely to be cured by surgery alone. The significant frequency of lymph node metastases in occult (subclinical, impalpable) breast cancer in the series cited by Abel and Deitch[1] underscores the need to add systemic treatment for patients with occult breast cancer as well as for others. Patients with positive nodes have even more to gain by adjuvant therapy, ie, the largest benefit compared to patients who receive local therapy alone. Thus there is abundant reason for offering a potentially curative combined treatment modality program to patients with occult breast cancer.

It would be most unfortunate if readers of this study diagnosed their patients' breast cancers earlier or more efficaciously, only to offer them less than the most current and effective options for limited surgery and for systemic preventive therapy.

On another issue, the need to freeze a portion of breast biopsy tissue requires re-emphasis. We agree that permanent histologic sections are preferable for definitive pathologic diagnosis, but material must be preserved for assays for estrogen and progesterone receptors in the event of a final diagnosis of carcinoma. The results of such assays clearly assist selection of an optimal breast cancer management plan.

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In reply. The comments in the letter by Drs Schneiderman, Sporn, and Deckers are well taken and we agree generally, but we would like to address a few specific points. The article was not intended to reflect our complete approach to breast cancer management but to report our results on needle localization of occult breast cancer which included the treatment given. We recognize that alternatives exist to a modified radical mastectomy and in New York State we are required by law to discuss these with the patient. One of the major trials,[1] upon which current conservative surgery is largely based, is the National Surgical Adjuvant Breast Project B-06, which published its results in March 1985 after a mean follow-up of 39 months with five-year projections based on life-table estimates. We all hope that the projections of equivocal results with mastectomy or segmentectomy hold up, but in the meantime it is important to remember what the B-06 trial actually revealed. Twenty-five percent of patients originally assigned to one of the segmental arms required immediate mastectomy because of tumor involvement of a margin. The group assigned to segmentectomy plus radiation therapy had a 10% local recurrence within five years, and those with segmentectomy alone had a 30% local recurrence. Within five years, approximately 35% of those treated by segmental resection plus radiation had undergone mastectomy, and over 50% of those who had segmental resection alone underwent mastectomy. Whether survival will be affected following treatment of these recurrences is too early to tell, but if adequate follow-up reveals equivalency, then a giant step forward will have been made.

We are grateful to Drs Schneiderman, Sporn, and Deckers for pointing out the benefits of appropriate adjuvant therapy, and agree entirely with their remarks. A full discussion of the pros and cons generated by the May 1988 Clinical Alert regarding treatment of Stage I patients is not possible here, but the implication that all node-negative patients be advised to undergo therapy when 65-70% are cured by surgery alone should be of some concern. The benefits, as measured by three to four years' mean disease-free survival, though real, are modest, and in our opinion, should be used in any high-risk subsets. However, good local control needs to be emphasized. Hayward,[2] reporting on the Second Guy's Hospital Trial, provided "unequivocal evidence of the importance of achieving local control in the primary treatment of this disease." This was most important in T_1 lesions, the group most appropriate for conservative surgery. Peterson,[3] in a well designed trial, reported a 20-year follow-up of patients treated by radical mastectomy vs ultraradical mastectomy with *no* adjuvant treatment given to either group, which further indicates what may be accomplished by surgery alone. Robbins and Berg[4] a decade ago reported a 30-year follow-up of patients treated with radical mastectomy alone, which showed that 12.6% were alive and free of disease, 24% were dead of other causes, 6.8% were lost to follow-up, and 56% had died of breast cancer. It may be concluded that radical mastectomy "is capable of controlling local disease and curing a substantial number of patients." We hasten to add that we have not performed a radical mastectomy in ten years at our institution.

We agree with the necessity for preserving tissue for receptor studies and this is routine. We are now seeing many tumors too small to provide enough tissue for the dextran-charcoal method of analysis. Additionally, with increasing use of fine needle aspiration, another method for detecting receptor status was needed. We are currently using monoclonal antibodies (ER-EIA) on both fine needle aspirates and formalin-fixed paraffin blocks with good correlation with dextran-charcoal method.

A brief mention should be made of ductal carcinoma in situ, a disease that is in transition awaiting a final treatment recommendation. Authors should report whether they are dealing with gross or microscopic disease, and the histologic type, as this is important to the clinician agonizing over what is the "best" treatment for his patient. As reported by Gump,[5] "aggressive local treatment may be justified when one encounters breast cancer at a time when the possibility of systemic involvement is remote" and the chance of cure approaches 100%.

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1. Fisher B, Bauer M, Margolese R, et al: Five-year results of a randomized clinical trial comparing total mastectomy and segmental mastectomy with or without radiation in the treatment of breast cancer. *N Engl J Med* 1985; 312:665-673.
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Universal precautions

TO THE EDITOR: Thank you for the clear and succinct review of universal precautions, the Center for Disease Control's (CDC) guidelines for controlling the transmission of bloodborne pathogens in health care settings. By helping to raise the level of awareness, the Medical Society of the State of New York and the *Journal* have made a significant contribution to infection control.

There is, however, one very important fact which the article overlooked, and that is that the Occupational Safety and Health Administration (OSHA) has mandated and is vigorously enforcing compliance with the CDC's guidelines. Many health care employers, including some very prominent ones, have already been cited and fined by OSHA.

Under the Occupational Safety and Health Act of 1970, employers have an obligation to provide a workplace free from recognized hazards. This obligation, known as the "General Duty Clause," was the basis for OSHA's Compliance Directive of August 15, 1988, which mandated the implementation of an infection control program encompassing universal precautions by every health care employer in the United States. Private employers in New York State fall under OSHA jurisdiction. Public employers must comply with the Public Employees Health and Safety Act. However, PEHS Act "standards" must be at least as stringent as the federal OSHA standards. Administrators of state and municipal facilities in New York State may wish to contact the New York State Department of Labor for more information.

The OSHA Compliance Directive mandates a comprehensive effort on the part of health care employers—physicians with a single assistant and large academic medical centers alike—to train employees, provide personal protective equipment, offer hepatitis B vaccine and appropriate medical counseling to employees at risk, and monitor their establishments to ensure compliance. Employers not in compliance may risk civil liability in addition to citations and fines from OSHA.

Recently, OSHA released a proposed Standard for Bloodborne Pathogens which further defines employers' responsibilities in this area. OSHA will accept comments and suggestions from interested parties before publishing a final standard. Some of your readers may wish to take advantage of this opportunity to participate in the rulemaking process. In the meantime, they should know that they are required to comply with the "rules" already on the books.

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1. Update: Universal precautions for prevention of transmission of human immunodeficiency virus, hepatitis B virus, and other bloodborne pathogens in health-care settings. *NY State J Med* 1988; 88:649–651.

In reply. The Council of the Medical Society of the State of New York adopted the Centers for Disease Control's (CDC) guidelines on the recommendation of the Society's Committee on Preventive Medicine. These guidelines, originally published in *Morbidity and Mortality Report* (1988; 37:377–382, 387–388), were reprinted in the *Journal*,[1] and it is to this reprinting that Ms Van Wagenen is referring. Since this was an exact reprinting of the CDC guidelines, it was not appropriate to modify it with the inclusion of other information.

We very much appreciate Ms Van Wagenen's bringing to our attention the fact that the Occupational Safety and Health Administration has mandated and is enforcing compliance with the CDC's guidelines on universal precautions as previously described. This information will be useful to our readers.

PASCAL JAMES IMPERATO, MD
Editor

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1. Update: Universal precautions for prevention of transmission of human immunodeficiency virus, hepatitis B virus, and other bloodborne pathogens in health-care settings. *NY State J Med* 1988; 88:649–651.

Penile prosthesis as an adjunct to intermittent self-catheterization in patients with neuropathic bladders

TO THE EDITOR: The use of the penile prosthesis to manage organic erectile impotence has become commonplace. Less well known but equally important is its use to facilitate urinary control in neurologically impaired patients. Insertion of a semirigid prosthesis to maintain a condom collection device has been described.[1] We have found the same prosthesis useful as an aid to intermittent self-catheterization in patients with neuropathic bladders and hemiplegia, as it obviates use of a second hand.

Case Reports. **Case 1.** A 35-year-old T_4 paraplegic man suffered from left hemiplegia secondary to autonomic dysreflexia. He was unable to benefit from a condom catheter due to a retractile penis and was being managed with an indwelling Foley catheter because of inability to do intermittent catheterization with his right hand alone. Urodynamic evaluation demonstrated a hyperreflexic bladder with autonomic sphincter dyssynergia. Adequate capacity with low intravesicular pressure was achieved with anticholinergic drugs, and intermittent catheterization was recommended. To make this possible, a semirigid (Mentor Malleable) penile prosthesis was inserted, making it unnecessary for the patient to hold his penis while catheterizing himself. He is now dry between catheterizations and also uses the implant for sexual activity.

Case 2. A debilitated 79-year-old man was hemiplegic after a right-sided cerebrovascular accident. He was being managed with continuous condom drainage but suffered frequently from urinary tract infections and excoriation of the penile shaft. Urodynamic evaluation demonstrated above-average bladder capacity. After insertion of a semirigid prosthesis, the patient uses intermittent self-catheterization and has been free of infection.

Discussion. Management of an external collecting device is cumbersome and difficult for the bedridden or wheelchair-bound patient. The complications include recurrent excoriation and lacerations of the penile shaft secondary to reflex erections, too-tight application of the appliance, and inability to secure the device adequately against vigorous voiding contractions. In patients unable to achieve an erection, the penis tends to retract and shorten, resulting in slippage of the collecting device and subsequent wetness. Intermittent self-catheterization can also be difficult because of the short length of the atrophic penis. Moreover, the need to hold the penis with one hand and insert the catheter with the other makes this method of urinary management impossible for the hemiplegic patient without aid.

Insertion of a semirigid penile prosthesis circumvents many of these problems. The prosthesis straightens the penis and adds length and girth. The resulting rigidity eliminates buckling and therefore the necessity to hold the penis for catheterization, making it possible for a patient with only one functioning hand to inject local anesthetic and insert the catheter. The patient remains dry, and the frequency of infection and penile excoriation is markedly reduced.

Before prosthesis insertion, all patients should have excretory urography and urodynamic evaluations to be certain they have stabilized bladder function and are good candidates for intermittent self-catheterization. The device can be inserted under local anesthesia. Possible complications are wound infection or poor fit of the appliance; inserting only one cylinder of the prosthesis may help avoid these problems. The incidence of prosthesis erosion is significant in patients with spinal cord injuries but is less of a problem in the hemiplegic, since the problem of decreased sensation is not present.

In summary, insertion of one or both cylinders of the semirigid penile prosthesis makes it possible for many hemiplegic patients to use intermittent self-catheterization with little morbidity.

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Family portraits from a family practice

If You Can't Remember Please Don't Forget. By Gerald W. Deas, MD. 44 pp. Hollis, New York, IDEAS, 1988. $5.00 (softcover)

Dr Gerald W. Deas is a black family practitioner who treats patients in Queens, New York, where housecalls are an integral part of his medical practice. He places a high value on caring for his patients in their homes and this has provided him with unique insights not only into their ills, but also into the larger canvas of their lives. This was a perspective once held by most practicing physicians in the United States, but one that has long since been lost by many as housecalls have become a rare phenomenon in the practice of American medicine.

While in his patients' homes, Dr Deas has always been struck by the family pictures, many of them dating to the late 19th century, which hang on the walls. He has now taken 19 of these photographs and has matched them to verse in this volume which celebrates not only black ancestry and values in the United States, but also the special

"Working her hands to the bone, To keep together our home, 'Blessed are the peacemakers' was her refrain, Giving the glory to God's name" (portrait of the author's mother).

"The Black father was majestic, And she was the queen mother, Bringing into the world children of royalty, Who became the treasures of our blackness."

relationship that a dedicated black family practitioner has with his patients.

Dr Deas is a highly respected playwright and poet who has produced several off-Broadway plays and written a number of books of poetry. One of his major objectives in producing this book was to give young black people a sense of pride in their traditional values. As he says in his introductory notes, "It is evident that if we are to preserve our African dignity and spirituality, we must make an effort to dress up the inside rather than the outside. We must make it plain to our young brothers and sisters that it is not the gold and diamonds that they wear around their necks and in their ears but, it is the golden quality of their thoughts and the diamonds of ideas that will contribute to our survival." Dr Deas then goes on to say, "Whenever I make a housecall on my patients I have been impressed that even in the most humble of homes I could always observe one or more photographs of distant relatives on the walls. These photographs not only reflected dignity in appearance but also dignity in character . . . In this book . . . I will present some of these photographs with verses that I feel will appropriately express the innermost thoughts of our people at that time."

In his book, Dr Deas succeeds in honoring black values and historical accomplishments and in stimulating young American blacks to uphold their dignity and spirituality. However, the more subtle story is that of the family practitioner who has established a very close relationship with his patients. Through the metaphors of photography and verse, we can come to understand this physician-patient relationship and how Dr Deas' patients have responded to him by sharing some of their most precious possessions—images of their own cherished past. He has been able to build up this relationship over a period of close to a quarter of a century through dedicated service in a part of New York City where physical illness is greatly influenced by social and economic determinants and where poverty and crime are major burdens.

This is a moving volume which serves as a vivid reminder of how admirable the practice of medicine can be. Dr Deas has linked visual imagery with his verse and in so doing has given all of us a view of not only black heritage and values but also the physician-patient relationship. This book is highly recommended and will provide readers with pleasure while reminding them of the treasured values long established by family practitioners in this country.

If You Can't Remember Please Don't Forget may be obtained from IDEAS, 109-07 197th St, Hollis, Queens, NY 11412.

PASCAL JAMES IMPERATO, MD
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Foodborne illness in New York State 1980–1987

Background

Foodborne illness accounts for substantial morbidity in New York State and represents a public health problem that is largely preventable. During 1980–1987, 999 outbreaks involving 20,415 cases were reported to the New York State Department of Health. In 1987, there were 157 outbreaks consisting of 3,893 cases. Twelve deaths and 81 hospitalizations occurred in that year as a result of foodborne illness.

Types of Foodborne Illness

Foodborne illnesses are classified as either poisonings or infections according to how they originate. Food poisoning (intoxication) is the ingestion of toxins found in plants or animals, metabolic products produced by microorganisms and certain chemical food additives that result in illness.[1] Infection is the ingestion of microorganisms and the resultant bodily response that occurs either to the organism or to the toxins the organism may generate within the body.

Foodborne illness can also be classified based on the causative agent as bacterial, viral, chemical or parasitic. Illnesses caused by these agents vary from mild to severe intensity. Viral gastroenteritis usually manifests as a mild disease of short duration (24–48 hours) while bacterial gastroenteritis tends to be more severe and of longer duration (> 48 hours). Foodborne illness of bacterial etiology represented the largest number of confirmed and suspected cases in the state (7,531 during 1980–1987). Salmonella was the bacterial agent confirmed most frequently during this period. Foodborne illness of suspected or confirmed viral etiology also represented a major portion with 7,005 reported cases. Although viruses are believed to be the most common etiologic agent involved in foodborne illness, many cases of viral gastroenteritis remain unconfirmed due to the difficulty and limited availability of viral testing.[2]

Chemical agents accounted for a much smaller percentage of all foodborne illness; 402 suspected and confirmed cases were reported during 1980–1987. The most common chemical agent associated with foodborne illness was scombrotoxin. Scombrotoxin intoxication occurs when histidine in certain fish flesh is converted to a histamine-like compound by *Proteus vulgaris* and other species that produce histidine decarboxylase, resulting in an allergic response.[3] Other chemical agents that have caused sporadic cases of illness include both heavy metals and mushroom poisoning. Parasitic agents caused only 21 cases. A total of 5,456 cases were of unknown etiology.

Control Measures

Inadequate cooling or refrigeration is the most often reported contributing factor in foodborne outbreaks. Infected food handlers have been associated with foodborne illness. Other frequently reported factors include improper hot holding temperatures, inadequate cooking and food from an unapproved source (food that has not undergone a mandated inspection).

The following measures are recommended to prevent foodborne illness from occurring.

- Contact with food should be kept at a minimum through the use of utensils and plastic gloves.
- Those handling food should follow strict handwashing procedures before and after food handling and should not work while ill.
- Food should be kept at the proper hot holding temperatures (>140°F) and refrigerated (<45°F) promptly after serving.
- Consumption of raw or undercooked meats, eggs and shellfish should be avoided.
- Surfaces used for food preparation should be washed, rinsed and sanitized before and after use in order to avoid cross contamination with other foods.

The surveillance data were compiled by the New York State Department of Health Foodborne Disease Surveillance Program. This program assists in prevention and control activities, and seeks to find the causes of foodborne illness. The number of reported foodborne outbreaks are believed to represent a fraction of the total outbreaks that actually occur. Outbreaks resulting in mild illness or those associated with a private family meal are much less likely to be reported. Those associated with public food services and when serious illness or death occurs are reported more frequently.[3]

Reporting

The State Sanitary Code requires that outbreaks involving two or more individuals be reported as soon as possible to the local health unit so that an investigation can be promptly initiated. In instances of botulism or chemical poisoning, one case constitutes an outbreak due to the more serious nature of such poisonings.

Laboratory testing in outbreak situations is conducted by the New York State Department of Health through the local health unit. For further information regarding foodborne illness, contact the Bureau of Community Sanitation and Food Protection at (518) 458-6706.

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TABLE 1. Common Foodborne Illnesses[1-8]

Disease/Causative Agent	Symptoms	Foods Often Implicated	Control Measures	Specimens for Lab Testing*
Salmonellosis *Salmonella* (all serotypes)	Onset: 6-72 hours (usually 12-36 hours) Abdominal pain, diarrhea, chills, fever, nausea, vomiting, malaise (Duration: several days)	Meat, poultry, eggs and egg products, other suspected foods	• Proper cooking temperatures of meat, poultry and eggs • Prompt refrigeration of food • Handwashing before and after food preparation • Proper cooking of eggs and egg products	Feces (Cary-Blair or glycerol transport medium), suspect foods, environmental swabs
Viral Gastroenteritis Norwalk and Norwalk-like agents Rotaviruses Other unknown viruses	Onset: 5-72 hours Fever, malaise, nausea, vomiting, abdominal pain, diarrhea (Duration: 1-2 days)	Clams, oysters, any food handled by infected food handlers	• Careful handwashing • Use of gloves when handling food • Avoid raw oysters, clams or other shellfish from polluted waters • Proper food handling and storage	Feces (glycerol) • Specimens should be delivered immediately or shipped on ice • Paired serum sometimes needed for confirmation
Perfringens Gastroenteritis *Clostridium perfringens*	Onset: 6-24 hours (usually 10-12) Most cases have watery diarrhea with nausea, intestinal cramps, few cases have fever or vomiting (Duration: 1 day or less)	Cooked meat or poultry, gravy, stew, meat pies	• Proper cooking and storage of foods, especially meat and meat gravies prepared in bulk quantities (Organisms destroyed by cooking but spores are generally heat resistant and able to withstand prolonged boiling)	Feces (Cary-Blair transport medium), suspect foods shipped on ice
Staphylococcal Intoxication *Staphylococcus aureus* Enterotoxin Toxins A-F	30 minutes-7 hours (usually 2-4 hours) Sudden onset of nausea, vomiting, diarrhea, abdominal cramps, dehydration (Duration: 1-2 days)	Cooked ham, meat, poultry, sauces and gravy, egg and dairy products	• Prompt refrigeration of foods, sanitary food handling • Food handlers with infected wounds or burns should not handle foods (Toxins are generally heat stable and not inactivated by reheating/cooking)	Feces (Cary-Blair transport medium), vomitus, nasal swab, suspect foods • If evident, swab purulent lesions or wounds of implicated food handlers
***Bacillus cereus* food poisoning** *Bacillus cereus* 2 enterotoxins	Vomiting toxin (heat stable) Onset: 1-6 hours Vomiting with some cases having diarrhea Diarrheal toxin (heat labile) Onset: 6-16 hours diarrhea, abdominal cramps with some vomiting	Boiled and fried rice, salads, high protein leftovers, custards, cereals, puddings, sauces, soups, meat	• Sanitary food handling, proper cooking and storage of foods (*B. cereus* spores are not killed during cooking and germinate when left unrefrigerated. Brief warming or flash frying will not destroy toxins)	Feces (Cary-Blair transport medium), vomitus, suspect foods
Campylobacteriosis *Campylobacter jejuni* and other species	Onset: 2-10 days (usually 3-5 days) Abdominal pain, often severe bloody diarrhea with foul-smelling loose stools, headache, malaise, fever (Duration: 1-7 days)	Handling raw poultry, raw milk, raw beef and liver	• Avoid cross contamination from raw poultry • Proper cooking of food from animal sources • Pasteurization of milk and milk products • Water chlorination • Proper food handling, use of plastic disposable gloves	Blood, feces (Cary-Blair transport medium), suspect foods
Hepatitis A Hepatitis A Virus	Onset: 10-50 days (average 25 days) Fever, malaise, lassitude, anorexia, nausea, abdominal cramps, dark urine, jaundice (Duration: few weeks to several months)	Oysters, clams, sandwiches, any food handled by infected food handlers	• Careful handwashing is the single most effective control measure • Do not work when sick • Close contacts of a case should obtain immune globulin • Use plastic disposable gloves and minimize hand contact with foods	Serum for anti-HAV IgM
Scombrotoxin Intoxication Scombrotoxin (Histidine in certain fish converted to a histamine-like product by certain bacteria) *Proteus vulgaris* and other bacteria	Onset: 1 minute-3 hours (average one hour) Flushing, headache, dizziness, burning of mouth and throat, upper and lower GI symptoms, uticaria and generalized pruritis	Scombroid fish, mahi-mahi (blue dolphin), tuna, bonito, mackeral, bluefish, skipjack	• Prompt refrigeration of fish soon after capture • Maintain cold temperatures until fish is cooked • Be wary of fish with sharp, metallic or peppery taste	Vomitus, feces, suspect fish
Shigellosis *S. sonnei*, *S. flexneri*, *S. dysenteriae*, *S. boydii*	Onset: 1-7 days (usually 1-3 days) Abdominal pain, fever, bloody, mucoid or purulent stools, tenesmus, pain on defecation	Moist mixed foods, salads, milk, beans, any food handled by infected food handlers	• Careful handwashing is the single most effective control measure • Do not work if sick with diarrheal illness • Minimize hand contact with foods and use plastic disposable gloves	Feces (Cary-Blair transport medium), suspect foods
***Escherichia coli* Diarrhea** *Escherichia coli*, generally 0157:H7	Onset: 12-72 hours Bloody diarrhea, hemolytic uremic syndrome, vomiting, dehydration, shock (Duration: 1-3 days)	Generally beef or raw milk	• Prompt refrigeration of foods • Proper cooking and reheating of foods, especially red meats • Good personal hygiene, proper sewage disposal	Feces (Cary-Blair transport medium), suspect foods
B-hemolytic streptococcal infection *Streptococcus pyogenes*	Onset: 1-3 days (rarely longer) Sore throat, painful swallowing, high fever, vomiting, malaise, occasionally a rash may occur	Milk, eggs and egg products, shrimp and tuna	• Prompt refrigeration of foods • Proper cooking of foods • Pasteurization of milk	Throat swabs, suspect foods, swab purulent lesions or wounds of implicated food handlers

Initial (diagnostic) specimens should be routed to the local hospital laboratory and remaining or reference specimens to the state laboratory.

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FROM THE LIBRARY

FOOD ADULTERATION

. . . Having given a brief resumé of the progress which has been made by the use of scientific methods toward answering the question as to what constitutes food values, we shall turn to the examination of the condition of our commercial food supplies. When science has failed to reach satisfactory conclusions, we can hardly expect to find that the business world has solved its problem.

While much of the underfeeding of the poorer classes is due to such lowering of food values, a still larger amount is due to ignorance of how to spend money so as to get the greatest amount of nourishment for a dollar, as well as ignorance of such methods of cooking as will utilize to the best advantage the food which is purchased.

A still greater danger is found in the presence of actually poisonous substances in foods. They may be divided into two classes:

1. Bacteria.
2. Inorganic poisons, such as preservatives and coloring matter.

Bacteria are probably the most dangerous of all impurities which are found in foods. This is very strikingly shown in the case of milk.

As lectures have recently been given on this subject I need not dwell upon it. Oysters grown in contaminated water may produce typhoid and other intestinal diseases.

Meats may be contaminated from two sources—the animal may be diseased, or on account of unsanitary methods of handling, disease bacteria may be introduced.

Vegetables and fruits, especially when eaten raw, may be infected from the fertilizer used. Gelatine is peculiarly liable to contamination on account of the material from which it is made and also because glue is manufactured from very similar materials. It is a comparatively simple matter to bleach the glue and sell it for gelatine. When the latter has been made up into candy, ice cream, jelly or other food products, it is almost impossible to determine by chemical analysis if a pure product has been used. On account of the keen competition in prices, bleached glues are often sold for use in foods. Not more than a year ago I had occasion to analyze a sample of glue used by one of our large ice cream manufacturers.

Not only is the consumer absolutely helpless to guard against food contaminated in this manner, but even the chemist and bacteriologist often find it difficult if not impossible to show by analysis of the finished product that unsanitary methods have been used in its production. The only effective method of protecting the consumer consists in an efficient system of inspection of all food factories. The only foods whose preparation are at present under such a system of inspection are meats and meat products, such as oleomargarine. This was brought about by a highly sensational exposure of the conditions existing in the packing industry. Even this would probably have failed to bring about any efficient remedy if the cudgel had not been taken up by that exceptionally enthusiastic and vigorous president, Theodore Roosevelt. I have no doubt that conditions quite as bad can be found to exist in many other of the food industries. With the present attempts at economy on the part of the National Government, not only will there be no extension of its activities in enforcing pure food laws but there is danger that the work of the pure food squad will be greatly curtailed unless emphatic expressions of public opinion are made. There are plenty of unscrupulous manufacturers who, at the first indication of inactivity of the enforcement of the pure food laws, are ready to resume the manufacture and sale of unwholesome foods. . . .

J. C. OLSEN
(*NY State J Med* 1910; 10:23–30)

MANAGEMENT OF THE FRAIL ELDERLY BY THE HEALTH CARE TEAM

By John R. Walsh, MD, Ruth Ann W. Tsukuda, RN, Judy Miller, RN. 206 pp, illustrated. St Louis, Mo, Warren H. Green, Inc, 1989. $32.50 (softcover)

This book is a concise, abbreviated text that addresses the care of the elderly from a primary caregiver's point of view. The authors describe basic interventions that can make an elderly person's life more fulfilling. The text summarizes clinical features, consequences, multi-dimensional management, drug therapy, and outcomes for problems faced by the elderly patient. Chapters are titled for presenting symptoms and go on to present brief differential diagnoses. This organization makes it particularly useful for the clinician.

The authors define the "frail elderly" as any patient over 75 years of age. This is unfortunately pejorative and perhaps "old" elderly would have been more descriptive. Other than this, the authors convey real respect for the geriatric patient. The book is directed to "health care providers" but seems most useful for the medical student, resident, or nurse practitioner being introduced to geriatrics. The authors state that essential elements in management are thoughtful evaluation, utilizing good judgment to avoid risks of drugs, and other interventions. They emphasize that maximized self-sufficiency should be the goal of a candid but optimistic assessment of limitations.

The early chapters present an authoritative synopsis of the aging process, making the point that the elderly are not a homogeneous group who have necessarily ceased to be productive, functional, or creative. Knowledge of aging effects on body systems promotes understanding of such simple problems as failure to recognize a nurse in a white uniform due to glare. The chapters on health promotion and assessment discuss the effects of nutrition, exercise, alcohol, and bed rest on the elderly patient. There are excellent guidelines for assessing mental status and activities of daily living skills. A point is made that recovery from even limited illness often takes more than six weeks, a fact that helps a family avoid premature placement decisions.

The chapter on rehabilitation points out that caregivers often focus on weakness and even reward deficiencies in their zeal to help. Chapters on dementia, incontinence, bowel problems, falls, pressure sores, osteoporosis, cerebral vascular disease, arthritis, Parkinson disease, and drugs are all brief, descriptive, and informative. A more complete discussion on the use of restraints and a more comprehensive list of adverse drug effects would have been useful. Good suggestions are made about chair exercises, antacids, calcium, constipation, and avoiding the use of aluminum-containing antacids in osteoporotic patients. The generous use of tables is helpful.

The authors seem to possess a wealth of practical experience that is well presented in the text. The book would seem most useful for the professional beginning a career in the care of the elderly and presents the most concise, easy-to-read, basic synopsis of geriatrics I have seen. The authors are willing to make specific practical recommendations. Any reader desiring more detail can refer to the bibliographies at the end of each chapter. This book should find its way to the shelves of many students in the several disciplines that deal with elderly patients and even into the homes of a few families with elderly loved ones.

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MEDICAL ETHICS AND THE ELDERLY: A CASE BOOK

By Mark H. Waymack, PhD, and George A. Taler, MD. 250 pp, illustrated. Chicago, Pluribus Press, Inc, 1988. $29.95 (clothbound)

This book provides an interesting and thorough overview of central ethical decisions arising in the care of aged persons. The book arose in concept from joint clinical case discussions conducted by the authors when introducing ethical issues into the teaching program of the family practice residency at the University of Maryland. A quick review of the table of contents will cue the reader to the breadth of subject material and comprehensiveness of the content. This is not a sample of "interesting episodes" from the clinic's experience, but a well constructed outline of the central issues arising in the whole range of clinical settings, each illustrated with a realistic if not entertaining clinical vignette.

The combined backgrounds of Dr Waymack in philosophy and medical humanities, and Dr Taler in family medicine and geriatrics, coupled with the experience-based origin of the work, lend immediate credibility. Experienced clinicians will readily identify with the situations presented and perhaps be disappointed that more definitive "answers" are not presented, though the delineation of specific issues and permutations of the cases may broaden their understanding. The primary value of the book appears to be for less experienced individuals or those who have never given serious thought to the less "scientific" aspects of their work, with immediate application for the introduction of ethics to health care students. The level of discussion and vocabulary used also make this an understandable work for lay audiences who may be surprised at the interplay of personal preferences and institutional or economic policies on the physician's freedom to act.

The text is arranged in four parts dealing with human interactions, levels of care, location of care, and reimbursement. Chapters in each section present a brief case followed by a discussion of the ethical issues focused on decisions made by the health care providers and their impact on the patient, family, and health care team members. Possible variations of the case and conclusions are presented. Each chapter is sufficiently brief to allow rapid reading and discussion in a realistic teaching setting. As mentioned, a sense of disappointment may arise from the appropriate absence of firm conclusions, although the authors do suggest their own beliefs through "should" or occasional "must" statements. Clinicians may respond with a sense of "hindsight is better than foresight" or "easier said than done," reflecting the reality of the discussions. The introductory discussions and the appendix offer a limited statement of basic ethical principles and vocabulary, substituting to some extent for the more comprehensive statement novices might desire.

As intended by the authors, this is a useful work at an introductory level, strengthened by its realism, immediate clinical application, and case-based approach. It could be strengthened or supplemented by an additional summary statement of basic ethical principles and terms apart from their inclusion in the case discussions. Readers may be frustrated by the inconclusive nature of the subject material itself. Those who feel sure they know the best future for health care in America and are ready to develop comprehensive but inflexible policies for the future may find the text disquieting.

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RESEARCH AND THE AGEING POPULATION

By Ciba Foundation Symposium 134. 264 pp, illustrated. New York, John Wiley & Sons, Inc, 1988. $49.95 (hardcover)

This 15-chapter volume of papers and interdisciplinary discussions by international authorities, presented at the 265th Ciba Foundation Symposium on Research and the Ageing Population, April 28–30, 1987, marks the centenary of the National Institutes of Health. It reviews the progress and research relevant to the elderly, including planning for the provision of care for elderly people, taking demographic, social, and economic factors into account. The book provides the reader with detailed research information and excellent insights into long term perspectives on future developments in aging. At the same time, the empathetic reader can enjoy the interdisciplinary creative and scientific thinking of the contributing research epidemiologists, demographers, physicians, health care planners, and economists.

This reviewer, currently concerned with psychogeriatrics, cardiology, and long term care, found of particular value Dr Tom Arie's chapter, "Questions in the Psychiatry of Old Age," Dr Nanette K. Wenger's chapter, "Cardiovascular Disease and the Elderly," and Stanley S. Wallack and Marc A. Cohen's chapter, "Costs of Long-Term Care: Distribution and Responsibility."

"Planning for Health Services for the Elderly" by Carel F. Hollander of France and Henk A. Becker of the Netherlands presented a most interesting three-contextual scenario research design, which offers policy makers a learning environment in which they can test strategies and then evaluate the circumstances in which they might be feasible. This method is said to eliminate much poor planning and waste that characterize projections and predictions for the future. It can also provide clues for making choices, clarify consequences and alternatives, and assist in establishing cost effective, long term strategy for health care for the elderly.

Expert editing, good typography, pertinent tables and charts, and comprehensive pertinent bibliographies render this volume an outstanding production. This book is a useful addition to the medical literature and will be of value to all interested in aging: researchers, practitioners, gerontologists, geriatricians, public policy planners and government agencies, epidemiologists, demographers, physicians, health care planners, and economists.

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CARING FOR THE DISABLED ELDERLY

By Alice M. Rivlin and Joshua M. Wiener. 318 pp. Washington, DC, The Brookings Institution, 1988. $29.95 (clothbound)

Health care professionals of today are confronted with an increasing number of obstacles in providing the type of health care they think is needed for all their patients, especially the disabled, chronically ill older adult. The usual explanation given by the providers of health care services is that of fiscal restraints imposed by the hospital, state, or federal government. This is especially true in the areas of home care and long term institutional care. For the practitioner who has little financial background in the health care field, there is a feeling of frustration over the inability to provide quality care and to understand the complexity of health care financing.

Caring for the Disabled Elderly was published at an appropriate time in the United States, when Congress has been struggling with a catastrophic health care bill that includes a type of "social" insurance to protect the assets of an older couple or individual confronted with a serious (and costly) *acute* illness. This legislation, however, does not protect the financial integrity of the older population confronted with long term institutional care. This book discusses who will pay for this type of long term care in the next 30–40 years. It also provides an in-depth discussion on the pros and cons of current existing models, and proposes newer and possibly more successful methods to finance this extremely costly form of health care.

The authors, Alice Rivlin and Joshua Weiner, are senior fellows in the Brookings Economic Studies program of the Brookings Institution, Washington, DC. Along with several other persons, also associated with the Brookings Institution, a model was developed entitled the Brookings-ICF Long Term Care Financing Model, to analyze various health care financing models in order to project their respective viability in the future. Their computerized model depends on predictions of changes in the demographics of the older adult population disability rates, use of long term care services, sources and levels of payment, and predicted death rates. Their predictions come from many government and private research sources. Interestingly, not taken into consideration in this model is the ability of the health care profession to significantly change the rate of long term disability or mortality in the year ahead. Currently existing models examined by the authors were Medicare, Medicaid, social/health maintenance organizations, and the still embryonic private long term care insurance programs covering long term care. In their current state all the models examined were found to be inadequate in scope for this population's rapidly expanding needs or else financially not viable. Future models are proposed by the authors which, in their opinion, could work to meet this type of health care requirement.

This book is written in a clear and concise manner that can be easily understood by the noneconomist. Sources and statistics for their calculations and predictions are well documented. The Brookings-ICF Long Term Care Financing model is described in both general and technical terms. The book is organized into sections dealing with public and private sector reform strategies.

The book will probably not have widespread appeal to the practitioner who struggles, often unsuccessfully, to keep up with medical literature in his or her area of expertise. This book and its concepts, however, should be part of the basic education of all health care providers who will practice geriatric medicine in the future. For this reason, this reviewer strongly recommends the book for administrators of hospitals and long term care institutions, legislators, and other practitioners who deal with budgeting issues in their respective places of employment. Also, this book is important reading for the gerontologist and geriatrician who are currently affected by fiscal restraints in their practices.

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THE PSYCHIATRIC TREATMENT OF ALZHEIMER'S DISEASE

By the Committee on Aging. Group for the Advancement of Psychiatry. 176 pp. New York, Brunner/Mazel Publishers, Inc, 1988. $20.00 (hardcover)

Our scientific understanding of Alzheimer disease has dramatically expanded in the past decade or so, unfortunately outpacing therapeutic advances. This disparity between knowledge and treatment is predominantly reflected in the many failed attempts to improve cognition. The authors of this book would have us look beyond these failures to recognize that patients with Alzheimer disease suffer not just from a disturbance of memory but from behavioral and psychosocial problems and, as such, may be treated. This is a compelling truth which is often doubted and which justifies the publication of such a book.

The concise text was written by a committee of 12 psychiatrists (Committee on Aging of the Group for the Advancement of Psychiatry). Many members of the committee are acknowledged leaders in the field of geriatric psychiatry. To my knowledge, the group has produced the first text exclusively emphasizing psychiatric interventions in Alzheimer disease.

The book is divided into five main sections: an introduction, sections on clinico-pathological correlations, diagnosis, and treatment, and a chapter containing case examples. The introductory chapter is a brief overview of the history and definition of Alzheimer disease, the issue of normal aging, theories of etiology, and approaches to treatment. This chapter is lean, but is apparently meant to be little more than a preamble. The discussion of normal aging at this point seems out of place, probably fitting better in the diagnosis chapter. The chapter as a whole does serve as a good introduction. The critical statement that "the concept of care can be as important as cure" is an excellent point of departure. The vital issue of Medicare policy is also addressed here.

The second section deals with the "excess disabilities," affective and personality changes, and their relationship to the stage of dementia. Caregiver reactions are briefly discussed. The chapter ends with a table describing the natural history of Alzheimer disease in stages; unfortunately, the streamlined organization of the table contrasts with the theme of clinical variability emphasized in the text. The chapter on diagnosis is straightforward and may serve as a quick reference on this subject.

The section on treatment, the central focus of the book, attempts to be comprehensive in a short span. This leads to a relative overemphasis on psychoanalytic issues and a somewhat sparse discussion of pharmacologic interventions. A more complete review of the use of psychotropic agents in Alzheimer disease would have been welcome. The authors do not convey the significant limitations of neuroleptic treatment for sundowning and other forms of agitation in Alzheimer disease, and there is no discussion of various agents which should be considered as alternatives (eg, carbamazepine). The concluding chapter comprises ten case examples illustrating relevant clinical points. Several important rating scales are appended; accompanying explanations might have aided the generalist in their use.

In toto, the book is a fine introduction to the subject for clinicians, students, and the multidisciplinary team. It is limited by an incomplete discussion of clinical management and pharmacologic treatment. Despite these drawbacks it effectively outlines the role of psychiatric intervention in Alzheimer disease.

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DOCTORS. THE BIOGRAPHY OF MEDICINE

By Dr Sherwin B. Nuland. 519 pp. New York, Alfred A. Knopf, Publisher, 1988. $24.95 (hardcover)

Medicine's great progress during the last 100 years makes it an attractive topic for writers. Recently, several books have been published covering portions of its history. Three of them used the biographies of key medical men to illustrate the advances made.

I started to review this particular book reluctantly. The 519 pages and the heaviness of the language in the introductory paragraphs intimidated me. However, challenged by the fact that the author is a surgeon and a member of the faculty of an outstanding medical school (Yale), my interest became aroused.

Using detailed and well researched accounts of the personalities and original contributions of pivotal physicians, the author succeeds in creating an interesting, albeit incomplete biography of medicine. He weaves a fascinating story that includes such medical "Hall of Famers" as Hippocrates, Galen, Vesalius, Pare, Harvey, Morgagni, Virchow, Lister, Halstead, and Taussig. The reader is escorted from Hippocrates' wonderful precepts through the beginnings of scientific research, the evolution of the germ theory of disease causation, antisepsis, and asepsis. The remarkable story of the discovery of anesthesia is retold in detail and the book ends with a very short chapter on heart transplantation. Having had the personal pleasure of meeting the magnificent Helen Taussig, and acting as moderator of a scientific program during which Dr Blalock introduced several of his (formerly) blue babies, I enjoyed reading this book. It requires deep concentration, and the pace is slow, but that is compensated for by the quality of the writing.

In words that my colleagues have heard many times, I recommend this book highly and without reservation.

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RATIONING MEDICINE

By Robert H. Blank. 290 pp. New York, Columbia University Press, 1988. $25.00 (hardcover)

Every physician in practice will acknowledge the fact that health care is expensive and will undoubtedly become more costly with time. However, until recently physicians have been insulated from the fiscal realities of medicine, as third parties have paid the bills for the care that we order. This insulation from dollars and cents reality and our professional commitment to the best interests of our patients have made rationing health care anathema to most physicians. After reading *Rationing Medicine* by Robert H. Blank, no health care professional will be able to ignore the central facts that rationing in many forms already exists, further rationing is inevitable, there is no overall national policy to address this issue, and the development of such a policy is going to be exceedingly difficult.

Robert H. Blank is a professor of political science and associate director of the Program for Biosocial Research at Northern Illinois University. A social scientist by training, he writes authoritatively in this area of medicine, using as examples organ transplantation, the treatment of seriously ill newborns, reproductive technologies, and fetal health. These are familiar illustrations that have been used by other authors to emphasize the high cost of medical care and the potential need for rationing. Dr Blank expands the usual superficial discussion of these problems by analyzing the problems and pitfalls of rationing mechanisms that could be utilized to deal with these issues. Chapters on the development and implementation of policy decisions as well as on the role of governmental institutions in providing this direction are particularly helpful to medical professionals who may be uninformed on how a national policy on this issue might be developed. Dr Blank's fundamental conclusion is that implementing cost constraining programs that may benefit society as a whole, but inadvertently harm individual patients, is extremely problematic. This is due to our dedication to the best interests of our patients and the commitment of society as a whole to the rights of individual citizens. However, in the final chapter he does outline his opinions on how we might make the hard choices that are necessary. As one might expect, Dr Blank emphasizes a shift from high technology, curative medicine to a broader, preventive approach and the alteration of public expectation and demand concerning health care with a change in basic values concerning individuals' rights and responsibilities for their own health. However, even using these methods to reduce the cost of health care and distribute it more equitably, Dr Blank sees numerous problems ahead in our attempt to avert financial disaster.

Although not stated explicitly, a distressing message is that medical professionals could, by and large, be left out of the decision making process involved in rationing health care. Perhaps a more constructive interpretation would be that unless we as health care professionals become directly involved in an attempt to develop a national policy to intelligently ration health care resources, all will suffer in the end.

While there are other books on rationing health care, I feel that Robert Blank has made a major contribution to this field. His scholarship is apparent in the clarity of his presentation and his arguments. In addition, the book is well referenced, which makes it an excellent source for individuals wishing to pursue further studies in this field.

In conclusion, *Rationing Medicine* should be considered compulsory reading for all practicing and aspiring physicians. While it may not be the book one would display prominently in the consultation room, it is a book which may provide all of us a foretaste of our future in medicine.

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BOOKS RECEIVED

AIDS

The Heterosexual Transmission of AIDS in Africa. Edited by Dieter Koch-Weser, MD, Hannelore Vanderschmidt, PHD. 293 pp, illustrated. Cambridge, Mass, Abt Books, Inc, 1988. $29.00 (paperback)

Toward a National Policy on Drug and AIDS Testing. Edited by Mathea Falco and Warren I. Cikins. 81 pp. Washington, DC, Brookings Books, 1989. $10.95 (paperback)

BIOCHEMISTRY

Inhibitors of Ribonucleoside Diphosphate Reductase Activity. Edited by J. G. Cory and A. H. Cory. 273 pp, illustrated. Elmsford, NY, Pergamon Press, Inc, 1989. $72.50 (hardcover)

CARDIOLOGY

Comprehensive Electrocardiology. Volumes 1, 2, 3. Edited by Dr Peter W. Macfarlane, Professor T. D. Veitch Lawrie. 1,786 pp, illustrated. Elmsford, NY, Pergamon Press, Inc, 1989. $495.00 (hardcover)

Synopsis of Cardiac Physical Diagnosis. Edited by Jonathan Abrams, MD. 253 pp, illustrated. Philadelphia, Pa, Lea & Febiger, 1989. $18.50 (paperback)

GASTROENTEROLOGY

Data Analysis for Clinical Medicine: The Quantitative Approach to Patient Care in Gastroenterology. Edited by Thomas C. Chalmers, MD. 217 pp, illustrated. Cheshire, England, International University Press, 1988. $49.00 (hardcover)

HISTORY OF MEDICINE

History of Medicine. Edited by Rebecca Greene, PHD. 133 pp. Binghamton, NY, The Haworth Press, Inc, 1988. $24.95 (hardcover)

INTERNAL MEDICINE

Bronchopulmonary Dysplasia. Edited by Eduardo Bancalari, MD, and J. Thomas Stocker, MD. 441 pp, illustrated. New York, Hemisphere Publishing Corp, 1988. $79.50 (hardcover)

Joslin Diabetes Manual. 12th Edition. By Leo P. Krall, MD, and Richard S. Beaser, MD. 406 pp, illustrated. Philadelphia, Pa, Lea & Febiger, 1989. $10.95 (paperback)

Biosignalling in Cardiac and Vascular Systems. Edited by M. Fujiwara, S. Narumiya, and S. Miwa. 441 pp, illustrated. Elmsford, NY, Pergamon Press, 1989. $80.00 (hardcover)

LEGAL MEDICINE

Preventing Emergency Malpractice. By Raymond M. Fish, MD, and Melvin E. Ehrhardt, MD. 179 pp. Oradell, NJ, Medical Economics Books, 1989. $31.95 (hardcover)

MEDICAL CARE

Rationing of Medical Care for the Critically Ill. Edited by Martin A. Strosberg, I. Alan Fein, and James D. Carroll. 97 pp. Washington, DC, Brookings Books, 1989. $11.95 (paperback)

MISCELLANEOUS

Health and Medicine in the Christian Science Tradition: Principle, Practice, and Challenge. By Robert Peel. 154 pp, New York, The Crossroad Publishing Co, 1988. $18.95 (hardcover)

Diagnosis and Treatment of Muscle Pain. Edited by Hans Kraus, MD. 116 pp, illustrated. Chicago, Ill, Quintessence Publishing Co, Inc, 1988. $32.00 (hardcover)

Controlling Cholesterol. By Kenneth H. Cooper, MD. 395 pp, illustrated. New York, Bantam Books, 1988. $4.95 (paperback)

The Responsible Physician: Standards of Excellence and the Critically Ill Patient. Edited by Stewart G. Wolf, Jr, MD, Milton W. Hamolsky, MD, Austin H. Kutscher, MD, and Karin Muraszko, MD. 102 pp. Brooklyn, NY, The Center for Thanatology Research, 1989. $14.95 (paperback)

The Intern Blues: The Private Ordeals of Three Young Doctors. By Robert Marion, MD. 352 pp. New York, William Morrow & Co, Inc, 1989. $19.95 (hardcover)

The Better Back Book: Simple Exercises for the Prevention and Care of Back Pain. By Constance A. Bean. 232 pp, illustrated. New York, William Morrow & Co, 1989. $17.95 (clothcover)

The Oxygen Breakthrough: 30 Days to an Illness-Free Life—The Natural Program. By Sheldon Saul Hendler, MD. 271 pp, illustrated. New York, William Morrow & Co, 1989. $18.95 (clothcover)

PE: How to Overcome Premature Ejaculation: By Helen Singer Kaplan, MD. 118 pp, illustrated. New York, Brunner/Mazel Publishers, 1989. $11.95 (paperback)

ONCOLOGY

Theories of Carcinogenesis. Edited by Olav Hilmar Iversen, MD. 327 pp, illustrated. New York, Hemisphere Publishing Corp, 1988. $59.95 (hardcover)

PEDIATRICS

Pediatric Pulmonary Disease. Edited by J. Thomas Stocker, MD. 273 pp, illustrated. New York, Hemisphere Publishing Corp, 1989. $45.00 (hardcover)

Toxicologic and Pharmacologic Principles in Pediatrics. Edited by Sam Kacew, MS, PHD, and Simon Lock, MS, PHD. 314 pp, illustrated. New York, Hemisphere Publishing Corp, 1988. $59.50 (hardcover)

Heart Disease in Infants, Children, and Adolescents. Fourth Edition. Edited by Forrest H. Adams, MD, George C. Emmanouilides, MD, and Thomas A. Riemenschneider, MD. 1,066 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1989. $149.95 (hardcover)

PUBLIC HEALTH

Methods for Biological Monitoring: A Manual for Assessing Human Exposure to Hazardous Substances. Edited by Theodore J. Kneip, PHD, and John V. Crable, BS. 496 pp, illustrated. Washington, DC, American Public Health Association, 1988. $59.25 (hardcover)

PSYCHIATRY

Treating Couples: The Intersystem Model of the Marriage Council of Philadelphia. Edited by Gerald R. Weeks, PHD. 353 pp. New York, Brunner/Mazel Publishers, 1989. $32.50 (hardcover)

The Treatment of Psychiatric Disorders. By William H. Reid, MD. 414 pp. New York, Brunner/Mazel Publishers, 1989. $35.00 (hardcover)

Sexual Trauma in Children and Adolescents: Dynamics and Treatment. By Diana Sullivan Everstine, PHD, and Louis Everstine, PHD. 206 pp, illustrated. New York, Brunner/Mazel Publishers, 1989. $25.00 (hardcover)

Moodswing. By Ronald R. Fieve, MD. 276 pp. New York, William Morrow & Co, Inc, 1989. $22.95 (clothcover)

SURGERY

Anorectal, Presacral and Sacral Tumors: Anatomy, Physiology, Pathogenesis and Management. By S. Arthur Localio, MD, Kenneth Eng, MD, and Gene Francis Coppa, MD. 356 pp, illustrated. Philadelphia, Pa, WB Saunders Co, 1987. $78.95 (hardcover)

Surgery for the Morbidly Obese Patient. Edited by Mervyn Deitel, MD. 400 pp, illustrated. Philadelphia, Pa, Lea & Febiger, 1989. $70.00 (hardcover)

The hunt for a vaccine against Lyme disease

Lyme disease, a multisystemic illness that over a period of weeks or years can cause a multitude of symptoms, including arthritis, neurologic abnormalities, and cardiac disease, is caused by the bacterium *Borrelia burgdorferi*. The most common means of transmission of *B burgdorferi* is via a tick bite, primarily that of a deer tick. Lyme disease has been isolated in 43 states in the United States, with Lyme disease-carrying ticks reported to be on the increase in particularly high risk areas of the northeast and upper midwest. In the United States, 90% of the cases of Lyme disease occur in either New York, New Jersey, Connecticut, Massachusetts, Rhode Island, California, Minnesota, or Wisconsin (*Science News* 1989; 135:184–187).

If infection with *B burgdorferi* is caught early in the course of the disease, most cases will respond to antibiotic therapy; however, tick bites often go unnoticed, and the early symptoms of Lyme disease may go undiagnosed. The prospects for a vaccine against Lyme disease are improving, with researchers exploring a variety of approaches for blocking *B burgdorferi* infection (*Science News* 1989; 135:186).

A major problem in vaccine development is that *B burgdorferi* produces only a weak immune response in humans. While some approaches to Lyme disease prevention involve boosting the host's immune response, others focus on how the tick disarms the host's immune system and how to inhibit the tick's anti-immune defense mechanisms.

Several theories of vaccine development against Lyme-infected ticks take advantage of the process of the tick's attachment to the host and feeding behavior, and the transmission of the bacteria to the host. Ticks can remain attached to the host for a period of days or weeks. During this time they secrete a spectrum of chemicals called "tick saliva"—which includes anticoagulants, digestive enzymes, and an "attachment cement"—that helps them resist the human immune response and aids in feeding. Several experimental vaccines are targeted against components of this saliva. Based on the belief that bacterial transmission is not likely during the first few hours of tick attachment, "Researchers hope a purified dose of the right tick proteins, combined with a chemical that amplifies the host's immune response, might add up to a vaccine that would interrupt a tick's blood meal before disease transmission occurs."

Efforts to develop a vaccine can be highly labor intensive. One group of researchers dissected nearly 3,000 salivary glands from ticks, and then used genetic engineering technology to purify and mass-produce components of the tick saliva for subsequent testing as potential vaccines. Another group is developing a vaccine to block completely the production of the tick saliva.

In Australia, a vaccine developed against a cattle tick reduced the number of ticks on vaccinated animals by 91%. The Australians isolated the digestive tracts of 50,000 ticks and extracted 0.18 mg of a protein specific to the tick gut. Following analysis of the protein, a synthetic, nearly identical substance was developed as the vaccine. While feeding on the blood of a vaccinated host, the tick ingests a lethal dose of the substance, which causes damage to the intestinal lining.

"Despite these and other encouraging trials—none of which has been applied to humans—some scientists question whether standard vaccination approaches will ever outwit the wily tick." Ticks have honed particularly effective means of out-maneuvering the human immune system.

Isotretinoin—an effective yet teratogenic drug

Isotretinoin, a derivative of vitamin A, is highly effective in the treatment of severe cystic acne. A therapeutic regimen with this oral medication typically lasts 20 weeks, after which the acne commonly enters remission for a prolonged period. Despite the success achieved with isotretinoin since it first came to market in the United States in 1982, the established teratogenic effects of the drug create a conflict with regard to dispensing, marketing, and regulating isotretinoin. In a recent article, Robert S. Stern, MD, detailed the efforts taken to assure safe usage of isotretinoin without restricting availability of the drug to patients who would benefit from its therapeutic effects (*N Engl J Med* 1989; 320:1007–1009).

Based on animal tests that demonstrated the drug's teratogenicity, the package insert stated from the beginning that pregnant women should not use isotretinoin. However, by 1983, there were reports of birth defects and spontaneous abortions after exposure to isotretinoin during the first trimester of pregnancy. Subsequent published reports described the affected infants and their birth defects.

In response, in 1983, the drug manufacturer and the Food and Drug Administration (FDA) escalated their educational program aimed at increasing physician and patient awareness of the teratogenic effects of isotretinoin. In 1984, the warning accompanying the drug's labeling information was reworded to present even more clearly the risk to potentially fertile women. Since 1985, the frequency of spontaneous abortions and malformed infants attributed to isotretinoin exposure appears to have decreased, based on the number of reports to the FDA.

Continued success with isotretinoin in the treatment of severe cystic acne and the potential for its effectiveness in treating other, less debilitating forms of acne, emphasize the benefits of this drug. In addition, although other reportedly less harmful forms of vitamin A are currently under development (see following news brief), their safety and therapeutic value have yet to be demonstrated in humans. As Dr Stern explains, failure to comply with the guidelines regarding dispensation and administration of isotretinoin and to minimize the frequency of exposure to isotretinoin among pregnant women could result in restricted distribution of the drug or its removal from the market.

NEWS BRIEFS are compiled and written by Vicki Glaser.

Working with the FDA, the manufacturer has developed changes in labeling and packaging as well as new educational programs. "Isotretinoin is now contraindicated in women with childbearing potential unless they have severe, disfiguring cystic acne that is recalcitrant to standard therapies, are considered by the physician to be reliable, can take contraceptive measures, have received oral and written warnings of the hazards of pregnancy, and have negative serum pregnancy tests within two weeks before starting therapy."

These guidelines do impose an increased burden on the physician, demanding more time for patient screening, counseling, and monitoring for compliance. However, this challenge to the medical and regulatory communities represents more than an attempt to sustain the availability of one drug. "Our success in facing the challenge of isotretinoin will be important in determining whether other such drugs become available and how they are used."

Water-soluble vitamin A proves less toxic

Derivatives of retinoic acid, such as tretinoin (trade name Retin-A) and isotretinoin (Accutane), are being used to treat cases of severe acne. Researchers are also testing tretinoin as an anti-wrinkle cream. However, these two potent medications produce serious complications. Tretinoin kills cells and burns skin, while isotretinoin is associated with an increased risk of birth defects when used by pregnant women.

Whereas retinol, the fat-soluble form of vitamin A, is toxic in large doses, water-soluble derivatives of vitamin A are less toxic. Scientists have recently performed animal tests with water-soluble forms of vitamin A that have proven effective in curing vitamin A deficiency in rats (*Science News* 1989; 135:204). Early results indicated that one form may even be safe for pregnant rats. While all the fetuses of pregnant rats administered comparable doses of retinoic acid died, the pregnant rats given water-soluble compounds all produced normal offspring. The researchers have suggested that the water-soluble derivatives do not cross the placenta. Studies did, however, demonstrate absorption of the water-soluble derivatives by the rats themselves.

In addition to finding use as a potential treatment for acne and wrinkling, these water-soluble compounds could be effective in preventing vitamin A deficiency in people with genetic disorders or diseases that predispose them to vitamin A deficiency.

Laser may improve early detection of cataracts

A joint effort between scientists at the Georgia Institute of Technology and the Joslin Diabetes Center has produced a laser instrument that the developers hope will help identify cataracts in the early stages of formation. By detecting warning symptoms of cataracts, this experimental instrument could allow physicians to initiate preventive and therapeutic measures earlier in the development of cataracts.

Based on the design of a conventional slit lamp used in routine eye examinations, this instrument incorporates a low-intensity laser beam instead of a filament light source. The helium-cadmium laser focuses a safe level of light onto the lens of the eye and yields Raman scattering signals, fluorescent emissions, and Rayleigh scattering signals.

The instrument uses this information to measure the concentrations of cataract-susceptible proteins in the lens, the chemical makeup of existing protein-forming bonds, metabolic rates that relate to protein growth, and rates of change in the sizes of protein particles. In the course of a two-year clinical trial set to begin this summer at the Joslin Center, researchers hope to define data parameters that correlate with subsequent cataract formation.

One million dollars in grants available for research on kidney failure

In response to cutbacks in government-sponsored medical research programs, the Renal Therapy Division of Baxter Healthcare Corporation has established a $1 million Extramural Grant Program. The grants are available to nephrology researchers who are studying kidney failure, and its complications and treatment.

Priority will be given to research proposals that address the cardiovascular response to kidney dialysis, including hypotension, hypertension, and myocardial infarction. Members of the international academic community will judge the scientific merit of each grant proposal.

EPA sets Medical Waste Tracking Act in motion

The Medical Waste Tracking Act, implemented by the Environmental Protection Agency (EPA), is a two-year demonstration project aimed at monitoring and controlling the disposal of medical waste in ten states. The states named in this law are New York, New Jersey, Connecticut, Illinois, Indiana, Minnesota, Michigan, Ohio, Pennsylvania, and Wisconsin. Any other state can participate in the project, as determined by each state's governor.

According to the law, specific types of medical waste generated by physicians' offices, clinics, and laboratories must be separated from other waste products, placed in special, leak-proof containers, and tracked through their final disposal or destruction. Records of all such waste are essential, including waste that is incinerated or treated and destroyed on site.

The emphasis on tracking medical waste stemmed from the many reports of medical waste washing up on beaches last summer in states including New York, New Jersey, and Connecticut.

MSSNY House of Delegates calls for the removal of David Axelrod, MD, as Commissioner of Health of New York State

The single action of the MSSNY's 183rd annual House of Delegates Meeting in Rochester that caused the most stir was the passage of Resolution 89-20, which called upon Governor Mario Cuomo to dismiss David Axelrod, MD, as Commissioner of Health for New York State. This resolution, said Charles D. Sherman, MD, then president of the Society, was "not a personal attack against David Axelrod, MD, himself. He is obviously an individual committed to doing what he *thinks* is best for the health care system. The problem is that he is often *wrong*!"

The resolution was submitted by Verne Marshall, MD, of Geneva, in upstate Ontario County. It passed by an overwhelming margin. Dr Marshall expressed the growing frustration with increasing rules and regulations promulgated by Dr Axelrod and his department without input from physicians in the field. As Dr Sherman later expressed it, "Dr Axelrod does not directly deliver clinical care to patients in New York State. Consequently, he simply does not realize the often disastrous consequences of the regulatory excesses initiated at his direction. These excesses often constitute a complete roadblock, preventing patients from receiving the care they need."

This resolution generated enormous publicity. Governor Cuomo responded by calling Commissioner Axelrod "the best Health Commissioner in the country." The Medical Society followed up the action of the House by sending a strongly worded letter to the governor expressing concern about the great problems that medicine is facing in the near future, and asking for more cooperation between the state and the physicians practicing in New York. Governor Cuomo has not yet responded.

In a separate action, but one which also showed physicians' uneasiness with the current bureaucracy of health care in New York, the Medical Society passed a resolution that called for support of legislation to establish an independent commission of impartial experts to immediately undertake a study of the conflicting demands for resources within the health care system and make recommendations on setting priorities for resolving the health care crisis in New York City and the rest of the state. The commission will also examine federal issues affecting the health care crisis and will include national health experts as well as representatives from the MSSNY, the Hospital Association of New York State, the legislature, and the office of the governor.

Said Dr Sherman, "We call for a new approach which will involve all interested segments of the health care delivery system. Doctors, nurses, hospitals, nursing homes, labor, business, and of course, health care consumers must all be given a legitimate role in the planning process now so dominated by the Department of Health under its present leadership."

The Medical Society held its annual meeting in an upstate location for the first time in six years when delegates gathered at the Rochester Stouffer and Holiday Inn hotels in April. The HOD, although somewhat cramped with more than 500 delegates, Auxilians, MSSNY staff members, and guests in the two hotels on the banks of the Genesee River, was a particularly productive meeting. More than 100 resolutions were discussed and voted on in four days of meeting. The resolutions ranged from proposing safety helmets for the occupants of convertible cars traveling with the tops down to recommending greater testing and vaccination for hepatitis B). Among the more significant resolutions were:

Reproductive rights: that the MSSNY affirm the position that reproductive choice, like any medical decision, is one of an informed consent between the patient and her physician. A minority report signed by 49 of the 301 delegates stated that the MSSNY should accept the legality of the *Roe v Wade* decision of the Supreme Court of the United States regarding abortion; and that any physician involved in such activity should provide quality medical care.

RU-486 abortion pill: that the MSSNY believes that testing and release of RU-486 and other medications should be based upon scientific evidence and should be free of influence of political pressure groups.

AIDS and HIV classification: that the New York delegation to the AMA introduce two resolutions at the Annual Meeting of the AMA House of Delegates in June 1989 calling on the AMA to: 1) Formally take the position that states should apply the same public health measures to contain the HIV epidemic as are used to control the spread of other communicable and sexually transmissable diseases; and 2) Formally take the position that physicians be permitted to test patients for the presence of HIV infection in the same manner as they currently test for other infections and conditions, ie, when such a test is warranted in the physician's exercise of his medical judgment, and that states impose no restrictions or obstacles on this ability.

HIV infection confidential reporting: that the state Medical Society urge the New York State Health Department to consider requiring confidential reporting of any form of HIV infection under the rubric of HIV disease or HIV infection, with the term AIDS to be added if needed for state or federal accounting purposes; and that individuals who prefer anonymous testing as opposed to confidential testing be allowed to use a pseudonym or other identifier so as to assure completeness in the state's data reporting systems; and that a similar resolution be introduced to the AMA House of Delegates by the New York delegation as a recommendation to health departments of other states.

Tobacco use as contributory cause of death: that the MSSNY request the Commissioner of Health of New York State to modify the death certificate form to include a check-off box regarding tobacco use as a contributory factor to the cause of death on death certificates issued in the State of New York.

Nursing shortage in New York State: that the Society work with the legislature to implement short and long range measures to address nursing shortages and other health care personnel shortages.

Nurse education programs: that the MSSNY continue strongly to support programs that will assist and encourage persons to enter the field of nursing; and that these programs be integrated to allow transfer of credits toward higher levels of education; and that recruitment efforts begin in high schools, and enlist the aid of high school guidance counselors.

Neurologically impaired infants: that the MSSNY introduce legislation to remove neurologically impaired infants from the civil adjudication process.

Information statement of the Emergency Medical Services Committee of the MSSNY on conflicts between physicians and the pre-hospital system

Physicians who wish to provide emergency medical care should be familiar with the pre-hospital care organization (fire company, ambulance corps, emergency technician) in their area. Specifically, physicians should know how sophisticated or how limited the pre-hospital system may be in terms of equipment, supervision, training, and the sort of written protocols the system uses as a standard for care. Although Emergency Medical Services (EMS) systems in New York State are as variable in sophistication and resources as the hospital systems they serve, certain recurring problems seem to be common to all.

Physicians interacting with the EMS in an emergency situation. Physicians who are unknown to the emergency medical technicians and who wish to assume the care of patients in an emergency situation must identify themselves to the technicians as a New York State licensed physician.

Physician direction of care during transport. Physicians wishing to continue care for their own patients or one whom they are treating in an emergency situation usually must accompany the patient to the hospital in the ambulance. Most systems do not allow technicians to carry out a physician's orders if the physician is not physically present at the scene. If the physician will not accompany the patient to the hospital, most systems provide that the medical technicians follow established protocols or contact Medical Control for continued direction in patient care. This is especially true if an acute emergency occurs in the ambulance enroute to the hospital.

Transport to the nearest hospital. In an attempt to decrease the time from onset of illness or injury until definitive hospital care can be provided, most pre-hospital emergency systems require that ambulances take patients to the nearest hospital. They do not permit or honor patient requests for transport to a more distant hospital. This has created some hardship for physicians and patients when patients are taken to hospitals where their physicians are not on staff or if the hospital has none of the patient's previous medical records. If a physician believes that the policy of transport to the nearest hospital is inappropriate because a particular hospital does not meet the patient's needs, the physician should petition the emergency medical system so appropriate transport decisions can be made.

Do not resuscitate and EMS. Emergency medical technicians are required to resuscitate all patients unless they have been decapitated, have rigor mortis, or show other unmistakable signs of having been dead for a considerable period of time. If a physician believes that CPR and resuscitation are inappropriate for a patient, it is best for them to instruct the family not to call the emergency medical services if cardiac arrest occurs. The new do-not-resuscitate (DNR) law does not cover the home situation. DNR orders are not valid for transportation situations from the home to the hospital. However, they are in effect for inter-institutional transfers, ie, from nursing homes to hospitals.

William Sharpe, MD, and Leonard Jan Bruce-Chwatt, MD, receive Medical Society's Redway Award

The Medical Society bestowed its annual award for excellence in medical writing upon William Sharpe, MD, and Leonard Jan Bruce-Chwatt, MD, for outstanding articles in the *New York State Journal of Medicine.*

Dr Sharpe received the award for his commentary "The morality of hospital stewardship," and Dr Bruce-Chwatt for his article "Cinchona and its alkaloids: 350 years."

Dr Sharpe is Director of Laboratories at the Cabrini Medical Center in New York City and Associate Professor of Pathology at the University of Medicine and Dentistry of New Jersey. He also serves as the editor of the *Bulletin of the New York Academy of Medicine.*

Dr Bruce-Chwatt is Emeritus Professor of Tropical Hygiene at the University of London, a former Director of the Ross Institute of the London School of Tropical Medicine and Hygiene, and a member of the World Health Organization Expert Panel on Malaria.

Robert D. Brandstetter, MD, awarded William Hammond Citation

Robert D. Brandstetter, MD, was awarded the William Hammond Citation for Distinguished Service to the *New York State Journal of Medicine.* Dr Brandstetter, who is Assistant Director of the Department of Medicine at New Rochelle Hospital Medical Center, has demonstrated exceptional service to the *Journal* as a peer reviewer and a member of the Associate Editorial Board since 1983.

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Dominick A. Ajello, MD, Loch Arbour, NJ. Died July 23, 1988; age 80. Dr Ajello was a 1933 graduate of Faculte de Medecine de l'Universite de Geneve, Geneva, Switzerland. He was a Diplomate of the American Board of Ophthalmology and a Fellow of the American College of Surgeons. He was a member of the American Academy of Ophthalmology and Otolaryngology, the Pan American Association of Ophthalmology, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

William Louis Bennett, MD, Bayville. Died March 3, 1989; age 83. Dr Bennett was a 1934 graduate of the Albany Medical College of Union University. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

Marvin Avram Block, MD, Buffalo. Died February 28, 1989; age 87. Dr Block was a 1925 graduate of the State University of New York at Buffalo School of Medicine. He was a member of the New York Academy of Medicine, the New York State Society for Medical Research, the American Geriatrics Society, the American Public Health Association, the Academy of Psychosomatic Medicine, the American Medical Society for Alcoholism, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Coburn A. L. Campbell, MD, Ormond Beach, Florida. Died February 16, 1989; age 89. Dr Campbell was a 1928 graduate of the University of Toronto Faculty of Medicine, Toronto, Canada. He was a member of the Steuben County Medical Society and the Medical Society of the State of New York.

William Hynes Conway, MD, Larchmont. Died March 15, 1989; age 77. Dr Conway was a 1936 graduate of the George Washington University School of Medicine, Washington, DC. He was a Diplomate of the American Board of Internal Medicine. His memberships included the New York Academy of Medicine, the Clinical Society of the New York Diabetes Association, the New York State Society of Internal Medicine, the American Geriatrics Society, the Pan American Medical Association, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Frank J. Curran, MD, New York. Died February 7, 1989; age 85. Dr Curran was a 1929 graduate of the University of Minnesota Medical School, Minneapolis, Minnesota. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the New York Academy of Medicine, the American Academy of Neurology, the American Psychiatric Association, the American Academy of Child Psychiatry, the Association for Research in Nervous and Mental Disease, the New York County Medical Society, and the Medical Society of the State of New York.

James Llewellyn Davis, MD, Newark. Died February 13, 1989; age 89. Dr Davis was a 1925 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a member of the Medical Society of the County of Wayne and the Medical Society of the State of New York.

Frans DeRook, MD, Albany. Died March 11, 1989; age 62. Dr DeRook was a 1952 graduate of the Medische Faculteit Rijksuniversiteit te Groningen, Groningen, Netherlands. He was a member of the Medical Society of the County of Albany and the Medical Society of the State of New York.

Edmund B. DiCapua, MD, Massapequa. Died March 6, 1989; age 72. Dr DiCapua was a 1951 graduate of Howard University College of Medicine, Washington, DC. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Walter James Gannon, MD, Buffalo. Died February 22, 1989; age 70. Dr Gannon was a 1944 graduate of the State University of New York at Buffalo School of Medicine. He was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Alan Herfort, MD, White Plains. Died March 13, 1989; age 67. Dr Herfort was a 1946 graduate of the New York University School of Medicine. He was a Diplomate of the American Board of Internal Medicine. His memberships included the New York Academy of Medicine, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

James Jacob Hillenbrand, MD, Oneida. Died March 2, 1989; age 59. Dr Hillenbrand was a 1956 graduate of Georgetown University School of Medicine, Washington, DC. He was a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the New York State Surgical Society, the Medical Society of the County of Madison, and the Medical Society of the State of New York.

Nathan Hirsch, MD, Utica. Died October 13, 1988; age 79. Dr Hirsch was a 1936 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the American Psychiatric Association, the Medical Society of the County of Oneida and the Medical Society of the State of New York.

Abraham Charles Hoffman, MD, Floral Park. Died February 27, 1989; age 75. Dr Hoffman was a 1943 graduate of Middlesex University School of Medicine, Waltham, Massachusetts. He was a Diplomate of the American Board of Pediatrics and a Fellow of the American Academy of Pediatrics. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Frank H. Jaklitsch, MD, Dix Hills. Died March 8, 1989; age 85. Dr Jaklitsch was a 1931 graduate of the University of Maryland School of Medicine, Baltimore. He was a Fellow of the American Academy of Family Physicians. His memberships included the New York State Society of Occupational Medicine, the American Occupational Medical Association, the Nassau County Medical Society, and the Medical Society of the State of New York.

Donald E. Janelli, MD, Lakeville, Connecticut. Died March 22, 1989; age 69. Dr Janelli was a 1945 graduate of the New York Medical College. He was a Diplomate of the American Board of Surgery, a Fellow of the American College of Surgeons, and a Qualified Fellow of the International College of Surgeons. He was a member of the New York Academy of Medicine, the Nassau County Medical Society, and the Medical Society of the State of New York.

David Howard Klasson, MD, Brooklyn. Died January 2, 1989; age 81. Dr Klasson was a 1936 graduate of the Medizinische Fakultaet der Universitaet Bern, Bern, Switzerland. He was a Fellow of the American College of Angiology and a Qualified Fellow of the International College of Surgeons. He was a member of the American Geriatrics Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Steven E. Koweity, MD, Albertson. Died March 15, 1989; age 75. Dr Koweity was a 1940 graduate of the Faculty of Medicine, Baghdad University, Baghdad, Iraq. He was a Diplomate of the American Board of Pediatrics and a Fellow of the American Academy of Pediatrics. He was a member of the American Public Health Association, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Jacob Kravitz, MD, Brookville. Died January 11, 1989; age 93. Dr Kravitz was a 1922 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the American Academy of Family Physicians, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Wolf Laitner, MD, Woodridge. Died February 16, 1989; age 75. Dr Laitner was a 1946 graduate of the Facolta di Medicina e Chirurgia dell'Universita di Genova, Genova, Italy. He was a Fellow of the American Academy of Family Physicians. He was a member of the Sullivan County Medical Society and the Medical Society of the State of New York.

Harold Sello Leopold, MD, Mamaroneck. Died February 29, 1989; age 89. Dr Leopold was a 1924 graduate of the Medizinische Fakultaet der Julius Maximiliams Universitaet, Wurzburg, Bayern, Germany. His memberships included the American Psychiatric Association, the Association for the Advancement of Psychotherapy, the New York County Medical Society, and the Medical Society of the State of New York.

Gerard C. Maglio, MD, Hobe Sound, Florida. Died February 5, 1989; age 80. Dr Maglio was a 1934 graduate of Georgetown University School of Medicine, Washington, DC. He was a Fellow of the American College of Surgeons. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Joseph Raymond Mascaro, MD, Utica. Died January 3, 1989; age 85. Dr Mascaro was a 1931 graduate of Georgetown University School of Medicine, Washington, DC. He was a Fellow of the American College of Anesthesiologists. His memberships included the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the Medical Society of the County of Oneida, and the Medical Society of the State of New York.

Peter Andian Perillo, MD, Jackson Heights. Died February 16, 1989; age 77. Dr Perillo was a 1937 graduate of the State University of New York Health Science Center at Brooklyn. He was a Diplomate of the American Board of Pediatrics and a Fellow of the American Academy of Pediatrics. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Gizella Perl, MD, Herzlia Pita, Israel. Died November 24, 1988; age 89. Dr Perl was a 1924 graduate of the Institutul de Medicina si Farmacie, Cluj, Romania. She was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Herman O. Pineas, MD, New York. Died November 22, 1988; age 96. Dr Pineas was a 1917 graduate of the Friedrich-Wilhelms Universitaet Medizinische Fakultaet, Berlin, Germany. He was a member of the American Psychiatric Association, the American Academy of Neurology, the New York County Medical Society, and the Medical Society of the State of New York.

Anthony R. Romanelli, MD, New York. Died February 24, 1989; age 65. Dr Romanelli was a 1949 graduate of the Facolta di Medicina e Chirurgia dell'Universita di Bologna, Bologna, Italy. He was a Diplomate of the American Board of Internal Medicine, a Fellow of the American College of Cardiology, and a Fellow of the American College of Chest Physicians. He was a member of the American Thoracic Society, the New York County Medical Society, and the Medical Society of the State of New York.

Irving Rosenstein, MD, Englewood, New Jersey. Died January 1989; age 83. Dr Rosenstein was a 1932 graduate of the Creighton University School of Medicine, Omaha, Nebraska. He was a member of the American Academy of Family Physicians, the Bronx County Medical Society, and the Medical Society of the State of New York.

Richard D. Schindler, MD, New York. Died January 23, 1989; age 98. Dr Schindler was a 1917 graduate of the Friedrich-Wilhelms Universitaet Medizinische Fakultaet, Berlin, Germany. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Stephen B. Scorza, MD, Bohemia. Died March 4, 1989; age 38. Dr Scorza was a 1977 graduate of Facultad de Medicina de la Universidad Autonoma de Guadalajara, Guadalajara, Jalisco, Mexico. He was a Diplomate of the American Board of Family Practice. He was a member of the American Academy of Family Physicians, the Suffolk County Medical Society, and the Medical Society of the State of New York.

Mohinder Kumar Sharma, MD, Harrison. Died December 20, 1988; age 55. Dr Sharma was a 1957 graduate of the Sawai Man Singh Medical College, Rajasthan University, Jaipur, Rajasthan, India. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Thomas Joseph Syracuse, MD, Williamsville. Died February 26, 1989; age 80. Dr Syracuse was a 1933 graduate of the State University of New York at Buffalo School of Medicine. He was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

James Edwin Thompson, MD, Sarasota, Florida. Died January 3, 1989; age 86. Dr Thompson was a 1927 graduate of the University of Texas Medical Branch, Galveston, Texas. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Charles E. Tilley, MD, Westbury. Died January 1, 1989; age 84. Dr Tilley was a 1930 graduate of the New York Medical College. He was a member of the American Psychiatric Association, the American Geriatrics Society, the Suffolk County Medical Society, and the Medical Society of the State of New York.

John D. Tocco, MD, Endicott. Died March 4, 1989; age 80. Dr Tocco was a 1936 graduate of the Facolta di Medicina e Chirurgia dell'Universita di Roma, Rome, Italy. He was a member of the Broome County Medical Society and the Medical Society of the State of New York.

George C. Trombetta, MD, Rochester. Died March 8, 1989; age 67. Dr Trombetta was a 1950 graduate of the University of Rochester School of Medicine-Dentistry. He was a Diplomate of the American Board of Obstetrics and Gynecologists and a Fellow of the American College of Obstetricians and Gynecologists. He was a member of the New York Academy of Medicine, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Elmer H. Tuttle, MD, Rochester. Died December 7, 1988; age 74. Dr Tuttle was a 1943 graduate of Cornell University Medical College, New York. He was a Diplomate of the American Board of Dermatology, Inc. He was a member of the American Academy of Dermatology, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Joseph R. Valinoti, MD, Manhasset. Died February 3, 1989; age 96. Dr Valinoti was a 1915 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Frances Elizabeth Vosburgh, MD, Albany. Died February 13, 1989; age 92. Dr Vosburgh was a 1925 graduate of Cornell University Medical College, New York. She was a Fellow of the American Academy of Family Practice. Her memberships included the New York Academy of Medicine, the American Medical Women's Association, the Medical Society of the County of Albany, and the Medical Society of the State of New York.

Max Waltuch, MD, New York. Died February 9, 1989; age 89. Dr Waltuch was a 1923 graduate of the Medizinische Fakultaet der Julius Maximiliams Universitaet, Wurzburg, Bayern, Germany. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

William D. Wheat, MD, New York. Died February 11, 1989; age 62. Dr Wheat was a 1950 graduate of the Louisiana State University School of Medicine, New Orleans, Louisiana. He was a Diplomate of the American Board of Psychiatry and Neurology and a Fellow of the American College of Physicians. He was a member of the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Robert Ralph Yanover, MD, Kings Point. Died February 11, 1989; age 93. Dr Yanover was a 1920 graduate of Wayne State University School of Medicine, Detroit, Michigan. He was a Qualified Fellow of the International College of Surgeons. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Savas J. Zavoyianis, MD, Bergenfield, New Jersey. Died February 18, 1989; age 92. Dr Zavoyianis was a 1924 graduate of the Faculty of Medicine, Aristotelian National University of Athens, Athens, Greece. He was a member of the American Academy of Dermatology, the New York County Medical Society, and the Medical Society of the State of New York.

FOR MY PATIENTS

NUMBER 9 / 1989

PICNICS, PARTIES, AND POISONINGS

Dear Patient,

Picnics, parties, and barbecues are usually fun times, but sometimes they result in food poisonings or foodborne illnesses. The symptoms of vomiting, diarrhea, and abdominal cramps within hours or days after eating certain foods can affect many persons about the same time after the party. The most common cause of these poisonings is the presence of specific bacteria or viruses or their by-products (toxins) which contaminated the food before it was eaten. It would take several pages to name the many types of bacteria or viruses which can grow in food and cause illness, but there are several preventive measures you can take before your next party.

BASIC PROBLEM AREAS

There are four general sources of food contaminations:

1. Improper food preparation or processing
2. Improper food handling
3. Improper food storage
4. Improper personal hygiene or sanitation

PREPARATION AND PROCESSING

Poultry, meats, seafood, and eggs must be thoroughly cooked (internal temperature over 160° F) to be free of harmful bacteria. For example, raw or incompletely cooked clams or oysters may contain live hepatitis virus, and raw or partially cooked eggs may carry salmonella bacteria.

In home canning, the exact time, pressure, and temperature required to destroy the botulism bacteria must be strictly followed according to accepted methods. For example, home-canned vegetables should be boiled, with stirring, for at least 3 minutes. Bulging cans of food — whether homemade or commercially made — should ***not*** be opened. Foods with "off odors" should ***not*** be eaten or "taste-tested."

HANDLING AND STORAGE

Try to serve meat dishes hot as soon as they are cooked. The time from initial preparation of food to service to the guests should be reduced to a bare minimum unless the temperature can be properly controlled. If perishable foods must be kept for more than 2 hours, place them in shallow containers and cover them. They should immediately be stored ***hot*** (above 140°F) on a stove, grill, or chafing dish; or immediately stored ***cold*** (under 39°F) in a refrigerator or ice chest. Pastries, custards, and salads should be refrigerated as soon as possible after they are made.

PERSONAL HYGIENE AND SANITATION

All persons involved in the preparation and handling of any foods must carefully follow all rules of personal hygiene. Especially important is thorough handwashing (with soap and dry towel) after use of the toilet. Fingernails must be clean too.

No one should prepare or handle food if he or she has any of these conditions:

- infection (boils or open sores) on the face or hands
- any nose or eye discharge including nose colds
- diarrhea

The cleanliness of the kitchen, utensils, tableware, plates, and glasses is very important in preventing food contamination with many types of bacteria and viruses.

Now that you know the basic food safety rules, go ahead and have that party, and enjoy it.

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Office Handouts for Your Patients

This issue of the *New York State Journal of Medicine* contains another "For My Patients" letter, which appears on the preceding page.

Use this letter series to keep your patients informed about timely issues concerning diseases, treatments and health maintenance. Simply make photocopies of the page and distribute them to your patients with your newsletter or other correspondence, or place them in your reception area for your patients to read and take home.

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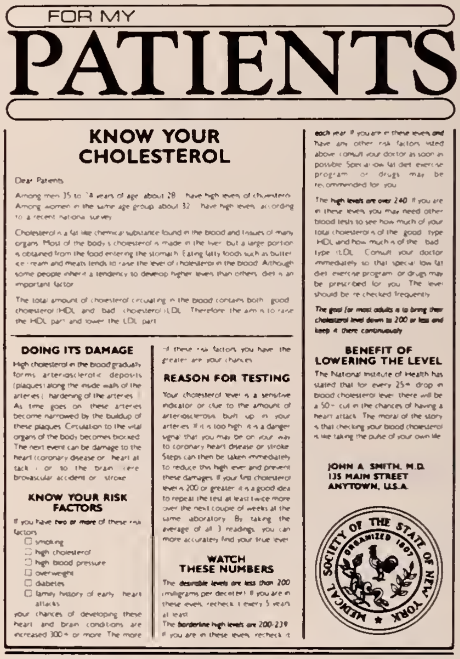

FOR MY

PATIENTS

KNOW YOUR CHOLESTEROL

Dear Patients

Among men 35 to 74 years of age, about 28 have high levels of cholesterol. Among women in the same age group about 32 have high levels, according to a recent national survey.

Cholesterol is a fat-like chemical substance found in the blood and tissues of many organs. Most of the body's cholesterol is made in the liver, but a large portion is obtained from the food entering the stomach. Eating fatty foods such as butter, ice cream and meats tends to raise the level of cholesterol in the blood. Although some people inherit a tendency to develop higher levels than others, diet is an important factor.

The total amount of cholesterol circulating in the blood contains both "good" cholesterol (HDL) and "bad" cholesterol (LDL). Therefore the aim is to raise the HDL part and lower the LDL part.

DOING ITS DAMAGE

High cholesterol in the blood gradually forms arteriosclerotic deposits (plaques) along the inside walls of the arteries (hardening of the arteries). As time goes on, these arteries become narrowed by the buildup of these plaques. Circulation to the vital organs of the body becomes blocked. The next event can be damage to the heart (coronary disease or "heart attack") or to the brain (cerebrovascular accident or "stroke").

KNOW YOUR RISK FACTORS

If you have **two or more** of these risk factors

☐ smoking
☐ high cholesterol
☐ high blood pressure
☐ overweight
☐ diabetes
☐ family history of early heart attacks

your chances of developing these heart and brain conditions are increased 300% or more. The more of these risk factors you have, the greater are your chances.

REASON FOR TESTING

Your cholesterol level is a sensitive indicator or clue to the amount of arteriosclerosis built up in your arteries. If it is too high, it is a danger signal that you may be on your way to coronary heart disease or stroke. Steps can then be taken immediately to reduce this high level and prevent these damages. If your first cholesterol level is 200 or greater, it is a good idea to repeat the test at least twice more over the next couple of weeks at the same laboratory. By taking the average of all 3 readings, you can more accurately find your true level.

WATCH THESE NUMBERS

The **desirable levels are less than 200** (milligrams per deciliter). If you are in these levels, recheck it every 5 years at least.

The **borderline high levels are 200-239**. If you are in these levels, recheck it **each year**. If you are in these levels **and** have any other risk factors listed above, consult your doctor as soon as possible. Special low fat diet, exercise program, or drugs may be recommended for you.

The **high levels are over 240**. If you are in these levels, you may need other blood tests to see how much of your total cholesterol is of the "good" type (HDL) and how much is of the "bad" type (LDL). Consult your doctor immediately so that special low fat diet, exercise program, or drugs may be prescribed for you. The level should be rechecked frequently.

The goal for most adults is to bring their cholesterol level down to 200 or less and keep it there continuously.

BENEFIT OF LOWERING THE LEVEL

The National Institute of Health has stated that for every 25% drop in blood cholesterol level there will be a 50% cut in the chances of having a heart attack. The moral of the story is that checking your blood cholesterol is like taking the pulse of your own life.

JOHN A. SMITH, M.D.
135 MAIN STREET
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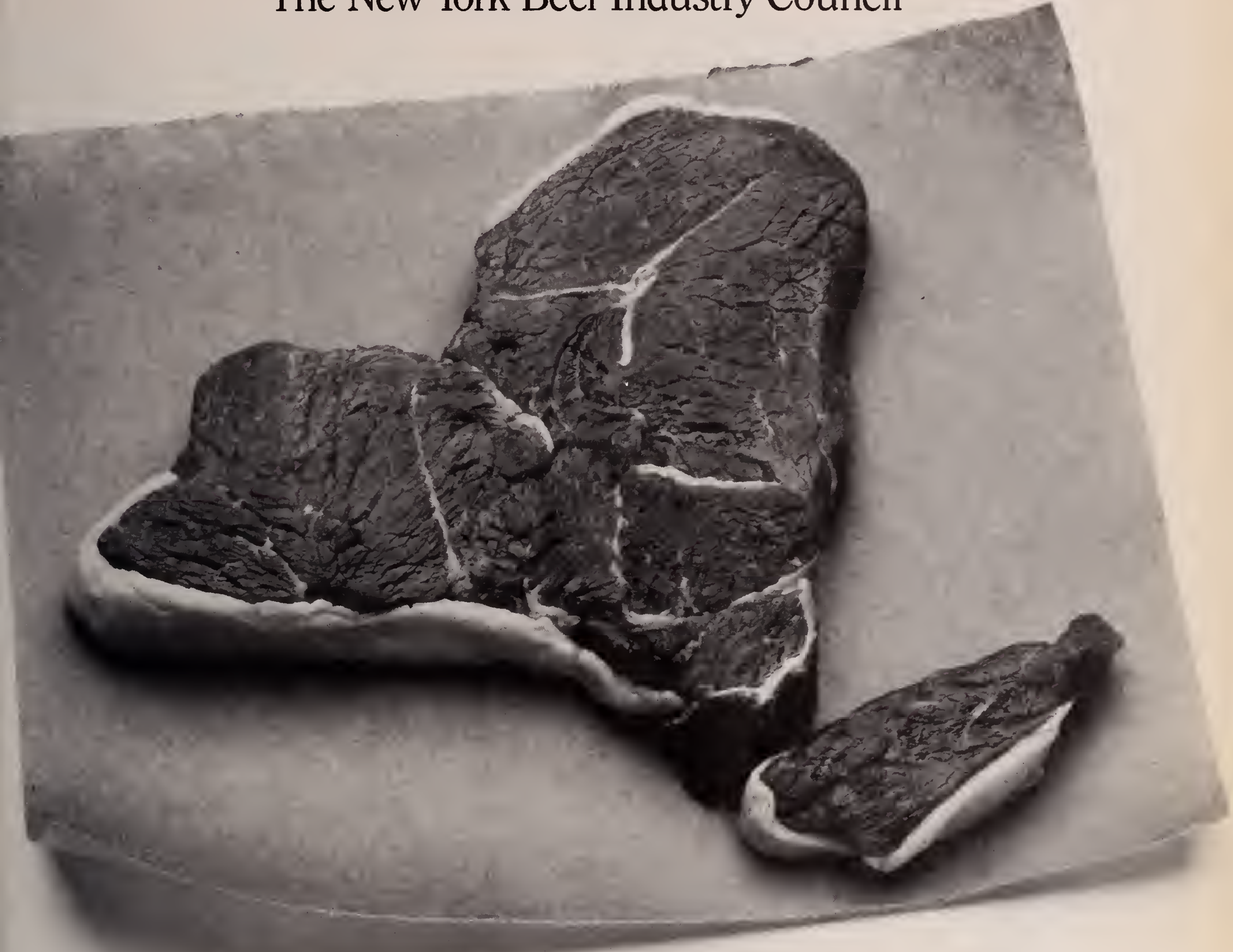

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Please see adjacent page for brief summary of prescribing information.

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vicodin ES ® CIII

(hydrocodone bitartrate 7.5mg [Warning: May be habit forming] and acetaminophen 750 mg)

INDICATIONS AND USAGE: For the relief of moderate to moderately severe pain.
CONTRAINDICATIONS: Hypersensitivity to acetaminophen or hydrocodone
WARNINGS:
Allergic-Type Reactions: VICODIN/VICODIN ES Tablets contain sodium metabisulfite, a sulfite that may cause allergic-type reactions including anaphylactic symptoms and life-threatening or less severe asthmatic episodes in certain susceptible people.
Respiratory Depression: At high doses or in sensitive patients, hydrocodone may produce dose-related respiratory depression.
Head Injury and Increased Intracranial Pressure: The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a preexisting increase in intracranial pressure. Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries.
Acute Abdominal Conditions: The administration of narcotics may obscure the diagnosis or clinical course of patients with acute abdominal conditions
PRECAUTIONS:
Special Risk Patients: VICODIN/VICODIN ES Tablets should be used with caution in elderly or debilitated patients and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture.
Cough Reflex: Hydrocodone suppresses the cough reflex; as with all narcotics, caution should be exercised when VICODIN/VICODIN ES Tablets are used postoperatively and in patients with pulmonary disease.
Drug Interactions: Patients receiving other narcotic analgesics, antipsychotics, antianxiety agents, or other CNS depressants (including alcohol) concomitantly with VICODIN/VICODIN ES Tablets may exhibit an additive CNS depression. The use of MAO inhibitors or tricyclic antidepressants with hydrocodone preparations may increase the effect of either the antidepressant or hydrocodone. The concurrent use of anticholinergics with hydrocodone may produce paralytic ileus.
Usage in Pregnancy:
Teratogenic Effects: Pregnancy Category C. Hydrocodone has been shown to be teratogenic in hamsters when given in doses 700 times the human dose. There are no adequate and well-controlled studies in pregnant women. VICODIN/ VICODIN ES Tablets should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.
Nonteratogenic effects: Babies born to mothers who have been taking opioids regularly prior to delivery will be physically dependent. The withdrawal signs include irritability and excessive crying, tremors, hyperactive reflexes, increased respiratory rate, increased stools, sneezing, yawning, vomiting, and fever.
Labor and Delivery: Administration of VICODIN/VICODIN ES Tablets to the mother shortly before delivery may result in some degree of respiratory depression in the newborn, especially if higher doses are used.
Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk and because of the potential for serious adverse reactions in nursing infants from VICODIN/VICODIN ES Tablets, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.
Pediatric Use: Safety and effectiveness in children have not been established
ADVERSE REACTIONS:
The most frequently observed adverse reactions include light-headedness, dizziness, sedation, nausea and vomiting. These effects seem to be more prominent in ambulatory than in nonambulatory patients and some of these adverse reactions may be alleviated if the patient lies down. Other adverse reactions include:
Central Nervous System: Drowsiness, mental clouding, lethargy, impairment of mental and physical performance, anxiety, fear, dysphoria, psychic dependence and mood changes.
Gastrointestinal System: The antiemetic phenothiazines are useful in suppressing the nausea and vomiting which may occur (see above); however, some phenothiazine derivatives seem to be antianalgesic and to increase the amount of narcotic required to produce pain relief, while other phenothiazines reduce the amount of narcotic required to produce a given level of analgesia. Prolonged administration of VICODIN/VICODIN ES Tablets may produce constipation.
Genitourinary System: Ureteral spasm, spasm of vesical sphincters and urinary retention have been reported.
Respiratory Depression: Hydrocodone bitartrate may produce dose-related respiratory depression by acting directly on the brain stem respiratory center. Hydrocodone also affects the center that controls respiratory rhythm, and may produce irregular and periodic breathing. If significant respiratory depression occurs, it may be antagonized by the use of naloxone hydrochloride. Apply other supportive measures when indicated.
DRUG ABUSE AND DEPENDENCE:
VICODIN/VICODIN ES Tablets are subject to the Federal Controlled Substance Act (Schedule III). Psychic dependence, physical dependence, and tolerance may develop upon repeated administration of narcotics; therefore, VICODIN/ VICODIN ES Tablets should be prescribed and administered with caution.
OVERDOSAGE:
Acetaminophen Signs and Symptoms: In acute acetaminophen overdosage, dose-dependent, potentially fatal hepatic necrosis is the most serious adverse effect. Renal tubular necrosis, hypoglycemic coma, and thrombocytopenia may also occur. Early symptoms following a potentially hepatotoxic overdose may include: nausea, vomiting, diaphoresis and general malaise. Clinical and laboratory evidence of hepatic toxicity may not be apparent until 48 to 72 hours post-ingestion.
Hydrocodone Signs and Symptoms: Serious overdose with hydrocodone is characterized by respiratory depression (a decrease in respiratory rate and/or tidal volume, Cheyne-Stokes respiration, (cyanosis), extreme somnolence progressing to stupor or coma, skeletal muscle flaccidity, cold and clammy skin, and sometimes bradycardia and hypotension. In severe overdosage, apnea, circulatory collapse, cardiac arrest and death may occur.

Revised June 1989

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(*continued from p 6A*)

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AROUND THE WORLD

CANADA

July 13–15. **Prenatal Diagnosis and Reproductive Genetics for the Obstetrician-Gynecologist.** 16 Cat I Credits. Four Seasons Hotel, Vancouver, British Columbia. Contact: Registrar, American College of Obstetricians and Gynecologists, 409 12th St, SW, Washington, DC 20024-2188. Tel: (202) 863-2541.

July 21–23. **12th Annual National Conference on Pediatric/Adult Allergy and Clinical Immunology.** The Four Seasons Hotel, Toronto, Ontario. Contact: Dr Elliott Middleton, Jr, 100 High St, Buffalo, NY 14203. Tel: (716) 845-2985.

EAST AFRICA

July 19–Aug 5. **Third World Medicine—Tropical Radiology and the Problem of AIDS.** 27 Cat I Credits. East Africa (Nairobi, Maasa-Mara, Lake Baringo, Lake Turkana, Mombasa, Lamu). Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077-1925. Tel: (404) 641-9773. □

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Index to Advertisers

Classified Advertising 5A, 8A, 19A, 20A

Knoll Pharmaceuticals 16A, 17A, 18A

Eli Lilly and Company . . . . 2nd Cover

Marion Laboratories 7A, 8A

Merck, Sharp and Dohme 3A, 4A

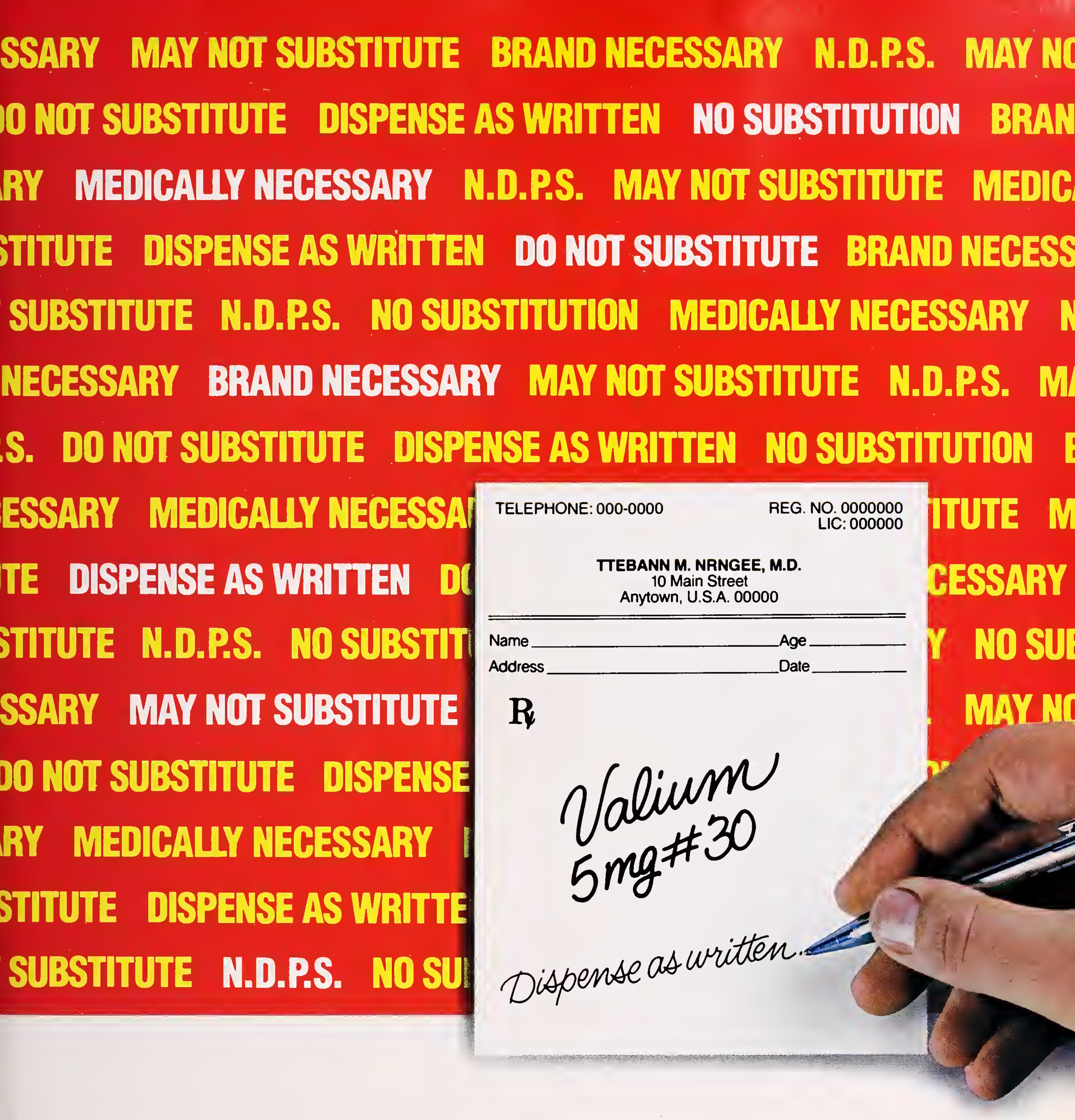
New York Beef Industry Council 15A

Palisades Pharmaceuticals 6A

Peck Packaging Corp. 5A

Roche Laboratories . . . . 3rd & 4th Cover

U.S. Air Force 2A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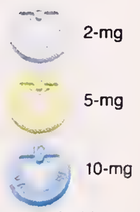

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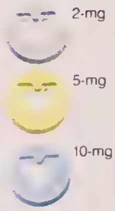

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Contents

COMMENTARIES

Strategies for preventing thromboembolism in patients with acute myocardial infarction 381
THOM W. ROOKE, MD

Modes of administration of heparin in clinical medicine 382
MICHAEL E. MENDELSOHN, MD

Cocaine 384
GEORGE R. GAY, MD

RESEARCH PAPER

Full dose subcutaneous heparin therapy 387
GERALD HOLLANDER, MD; ROBERT ROTHENBERG, RPh; ALVIN GREENGART, MD; JACOB SHANI, MD; EDGAR LICHSTEIN, MD

REVIEW ARTICLE

Cocaine: General characteristics, abuse, and addiction 390
NORMAN S. MILLER, MD; MARK S. GOLD, MD; ROBERT B. MILLMAN, MD

SPECIAL ARTICLES

A proposal concerning products liability 396
NEIL J. NUSBAUM, JD, MD

The prima facie tort rule: A possible medical-legal answer to the medical malpractice crisis 399
THEODORE SCHUSSLER, MD, JD

HISTORY

A history of the *New York Medical Journal* 403
PASCAL JAMES IMPERATO, MD

CASE REPORTS

Nonfatal pulmonary edema following cocaine smoking 415
LINDA EFFEREN, MD; DAVID PALAT, MD; JAY MEISNER, MD

Leiomyomatous tumors: Presentation of a giant gastric leiomyoma and a review of the literature 416
STEVEN P. COHEN, MD; CHERYL FRYDMAN, MD; MAURICE J. ZIMMERMAN, MD; FAZLOLLAH MOQTADERI, MD

Chest wall deformity following expansion of irradiated soft tissue for breast reconstruction 419
PETER BELA FODOR, MD, ALEXANDER J. SWISTEL, MD

Psoas abscess in the neonate 421
LUTGARDA ABAD VASQUEZ, MD; SANJIVAN PATEL, MD; JUDY G. SASLOW, MD; FRANCISCA T. VELCEK, MD

Pulmonic valve replacement for pulmonic insufficiency one year after mitral valve replacement 422
AARON J. GINDEA, MD; ROBIN S. FREEDBERG, MD; ALFRED CULLIFORD, MD; PAUL A. TUNICK, MD

LETTERS TO THE EDITOR

Omphalocele at 15 weeks 424
MICHAEL H. RATNER, MD

New York State's do-not-resuscitate law 424
JACK P. FREER, MD

The do-not-resuscitate law 425
TRACY E. MILLER, JD

Typhlitis 426
IRA S. SCHWARTZ, MD

ESSAY REVIEW

Judging medicine 427
ROBERT N. SWIDLER, JD

LEADS FROM EPIDEMIOLOGY NOTES 430
BOOK REVIEWS 434
NEWS BRIEFS 438
OBITUARIES 440
GUIDELINES FOR AUTHORS 442
MEDICAL MEETINGS AND LECTURES 7A

1989 CME Assembly Preliminary Program
(See Page 9A)

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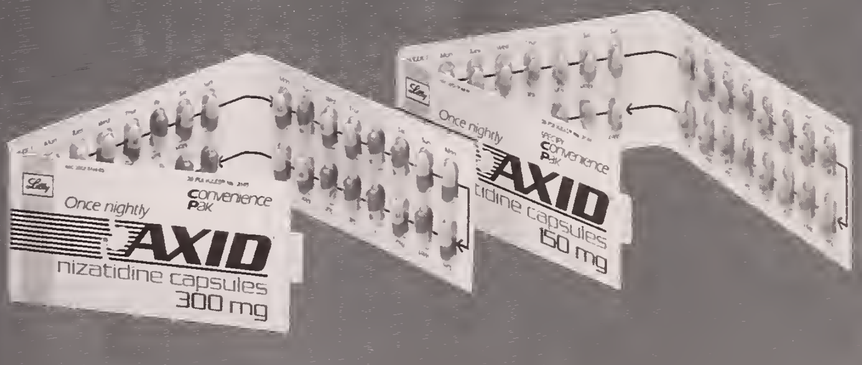

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Brief Summary

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Precautions: *General* – 1. Symptomatic response to nizatidine therapy does not preclude the presence of gastric malignancy.

2. Because nizatidine is excreted primarily by the kidney, dosage should be reduced in patients with moderate to severe renal insufficiency.

3. Pharmacokinetic studies in patients with hepatorenal syndrome have not been done. Part of the dose of nizatidine is metabolized in the liver. In patients with normal renal function and uncomplicated hepatic dysfunction, the disposition of nizatidine is similar to that in normal subjects.

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Drug Interactions – No interactions have been observed between Axid and theophylline, chlordiazepoxide, lorazepam, lidocaine, phenytoin, and warfarin. Axid does not inhibit the cytochrome P-450-linked drug-metabolizing enzyme system; therefore, drug interactions mediated by inhibition of hepatic metabolism are not expected to occur. In patients given very high doses (3,900 mg) of aspirin daily, increases in serum salicylate levels were seen when nizatidine, 150 mg b.i.d., was administered concurrently.

Carcinogenesis, Mutagenesis, Impairment of Fertility – A two-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a two-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups. The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid.

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, mouse lymphoma assay, chromosome aberration tests, and a micronucleus test.

In a two-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy – Teratogenic Effects – Pregnancy Category C – Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in one fetus and at 50 mg/kg it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in one fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers – Studies conducted in lactating women have shown that <0.1% of the administered oral dose of nizatidine is secreted in human milk in proportion to plasma concentrations. Caution should be exercised when administering nizatidine to a nursing mother.

Pediatric Use – Safety and effectiveness in children have not been established.

Use in Elderly Patients – Ulcer healing rates in elderly patients are similar to those in younger age groups. The incidence rates of adverse events and laboratory test abnormalities are also similar to those seen in other age groups. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Clinical trials of nizatidine included almost 5,000 patients given nizatidine in studies of varying durations. Domestic placebo-controlled trials included over 1,900 patients given nizatidine and over 1,300 given placebo. Among reported adverse events in the domestic placebo-controlled trials, sweating (1% vs 0.2%), urticaria (0.5% vs < 0.01%), and somnolence (2.4% vs 1.3%) were significantly more common in the nizatidine group. A variety of less common events was also reported; it was not possible to determine whether these were caused by nizatidine.

Hepatic – Hepatocellular injury, evidenced by elevated liver enzyme tests (SGOT [AST], SGPT [ALT], or alkaline phosphatase), occurred in some patients and was possibly or probably related to nizatidine. In some cases, there was marked elevation of SGOT, SGPT enzymes (greater than 500 IU/L) and, in a single instance, SGPT was greater than 2,000 IU/L. The overall rate of occurrences of elevated liver enzymes and elevations to three times the upper limit of normal, however, did not significantly differ from the rate of liver enzyme abnormalities in placebo-treated patients. All abnormalities were reversible after discontinuation of Axid.

Cardiovascular – In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in two individuals administered Axid and in three untreated subjects.

CNS – Rare cases of reversible mental confusion have been reported.

Endocrine – Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to Axid. Impotence and decreased libido were reported with equal frequency by patients who received Axid and by those given placebo. Rare reports of gynecomastia occurred.

Hematologic – Fatal thrombocytopenia was reported in a patient who was treated with Axid and another H_2-receptor antagonist. On previous occasions, this patient had experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported.

Integumental – Sweating and urticaria were reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported.

Hypersensitivity – As with other H_2-receptor antagonists, rare cases of anaphylaxis following administration of nizatidine have been reported. Because cross-sensitivity in this class of compounds has been observed, H_2-receptor antagonists should not be administered to individuals with a history of previous hypersensitivity to these agents. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

Other – Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine administration have been reported.

Overdosage: Overdoses of Axid have been reported rarely. The following is provided to serve as a guide should such an overdose be encountered.

Signs and Symptoms – There is little clinical experience with overdosage of Axid in humans. Test animals that received large doses of nizatidine have exhibited cholinergic-type effects, including lacrimation, salivation, emesis, miosis, and diarrhea. Single oral doses of 800 mg/kg in dogs and of 1,200 mg/kg in monkeys were not lethal. Intravenous median lethal doses in the rat and mouse were 301 mg/kg and 232 mg/kg respectively.

Treatment – To obtain up-to-date information about the treatment of overdose, a good resource is your certified regional Poison Control Center. Telephone numbers of certified poison control centers are listed in the *Physicians' Desk Reference (PDR)*. In managing overdosage, consider the possibility of multiple drug overdoses, interaction among drugs, and unusual drug kinetics in your patient.

If overdosage occurs, use of activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. Renal dialysis for four to six hours increased plasma clearance.

PV 2096 AMP [013089]

Additional information available to the profession on request.

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[1] Data on file, Lilly Research Laboratories.

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Calcium[1] and exercise are not enough to prevent postmenopausal osteoporosis, since estrogen deficiency is the primary cause.[2] PREMARIN is the only brand of estrogen indicated to prevent further bone loss and in a recently published study has been shown to reduce the risk of hip fractures by as much as 66%.[3]

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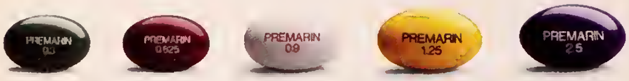
Proven benefits for menopause and beyond

See important information on adjacent page.

For moderate-to-severe vasomotor symptoms and for osteoporosis

PREMARIN®
(conjugated estrogens tablets)



0.3 mg 0.625 mg 0.9 mg 1.25 mg 2.5 mg

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For atrophic vaginitis

PREMARIN®
(conjugated estrogens)

Vaginal Cream

0.625 mg/g

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION AND PATIENT INFORMATION. SEE PACKAGE CIRCULARS.)*

PREMARIN® Brand of conjugated estrogens tablets, USP
PREMARIN® Brand of conjugated estrogens Vaginal Cream, In a nonliquefying base

> 1 ESTROGENS HAVE BEEN REPORTED TO INCREASE THE RISK OF ENDOMETRIAL CARCINOMA
> Three independent, case-controlled studies have reported an increased risk of endometrial cancer in postmenopausal women exposed to exogenous estrogens for more than one year This risk was independent of the other known risk factors for endometrial cancer These studies are further supported by the finding that incidence rates of endometrial cancer have increased sharply since 1969 in eight different areas of the United States with population-based cancer reporting systems, an increase which may be related to the rapidly expanding use of estrogens during the last decade The three case-controlled studies reported that the risk of endometrial cancer in estrogen users was about 4 5 to 13 9 times greater than in nonusers The risk appears to depend on both duration of treatment and on estrogen dose In view of these findings, when estrogens are used for the treatment of menopausal symptoms, the lowest dose that will control symptoms should be utilized and medication should be discontinued as soon as possible When prolonged treatment is medically indicated, the patient should be reassessed on at least a semi-annual basis to determine the need for continued therapy Although the evidence must be considered preliminary, one study suggests that cyclic administration of low doses of estrogen may carry less risk than continuous administration, it therefore appears prudent to utilize such a regimen Close clinical surveillance of all women taking estrogens is important In all cases of undiagnosed persistent or recurring abnormal vaginal bleeding, adequate diagnostic measures should be undertaken to rule out malignancy There is no evidence at present that "natural" estrogens are more or less hazardous than "synthetic" estrogens at equi-estrogenic doses
> 2 ESTROGENS SHOULD NOT BE USED DURING PREGNANCY
> The use of female sex hormones, both estrogens and progestogens, during early pregnancy may seriously damage the offspring It has been shown that females exposed in utero to diethylstilbestrol, a nonsteroidal estrogen, have an increased risk of developing, in later life, a form of vaginal or cervical cancer that is ordinarily extremely rare This risk has been estimated as not greater than 4 per 1,000 exposures Furthermore, a high percentage of such exposed women (from 30% to 90%) have been found to have vaginal adenosis, epithelial changes of the vagina and cervix Although these changes are histologically benign, it is not known whether they are precursors of malignancy Although similar data are not available with the use of other estrogens, it cannot be presumed they would not induce similar changes Several reports suggest an association between intrauterine exposure to female sex hormones and congenital anomalies, including congenital heart defects and limb-reduction defects One case-controlled study estimated a 4 7-fold increased risk of limb-reduction defects in infants exposed in utero to sex hormones (oral contraceptives, hormone withdrawal tests for pregnancy, or attempted treatment for threatened abortion). Some of these exposures were very short and involved only a few days of treatment The data suggest that the risk of limb-reduction defects in exposed fetuses is somewhat less than 1 per 1,000 In the past, female sex hormones have been used during pregnancy in an attempt to treat threatened or habitual abortion. There is considerable evidence that estrogens are ineffective for these indications, and there is no evidence from well-controlled studies that progestogens are effective for these uses If PREMARIN is used during pregnancy, or if the patient becomes pregnant while taking this drug, she should be apprised of the potential risks to the fetus, and the advisability of pregnancy continuation

DESCRIPTION: PREMARIN (conjugated estrogens, USP) contains a mixture of estrogens, obtained exclusively from natural sources, blended to represent the average composition of material derived from pregnant mares' urine It contains estrone, equilin, and 17α-dihydroequilin, together with smaller amounts of 17α-estradiol, equilenin, and 17α-dihydroequilenin as salts of their sulfate esters Tablets are available in 0 3 mg, 0 625 mg, 0 9 mg, 1.25 mg, and 2 5 mg strengths of conjugated estrogens Cream is available as 0 625 mg conjugated estrogens per gram

INDICATIONS AND USAGE: PREMARIN (conjugated estrogens tablets, USP). Moderate-to-severe *vasomotor* symptoms associated with the menopause (There is no evidence that estrogens are effective for nervous symptoms or depression without associated vasomotor symptoms and they should not be used to treat such conditions.) Osteoporosis (abnormally low bone mass) Atrophic vaginitis Kraurosis vulvae Female castration

PREMARIN (conjugated estrogens) Vaginal Cream is indicated in the treatment of atrophic vaginitis and kraurosis vulvae

PREMARIN HAS NOT BEEN SHOWN TO BE EFFECTIVE FOR ANY PURPOSE DURING PREGNANCY AND ITS USE MAY CAUSE SEVERE HARM TO THE FETUS (SEE BOXED WARNING)

CONTRAINDICATIONS: Estrogens should not be used in women (or men) with any of the following conditions 1 Known or suspected cancer of the breast except in appropriately selected patients being treated for metastatic disease 2 Known or suspected estrogen-dependent neoplasia 3 Known or suspected pregnancy (see Boxed Warning) 4 Undiagnosed abnormal genital bleeding 5 Active thrombophlebitis or thromboembolic disorders 6 A past history of thrombophlebitis, thrombosis, or thromboembolic disorders associated with previous estrogen use (except when used in treatment of breast or prostatic malignancy)

WARNINGS: Estrogens have been reported to increase the risk of endometrial carcinoma (see Boxed Warning) However, a recent large, case-controlled study indicated no increase in risk of breast cancer in postmenopausal women. A recent study has reported a 2- to 3-fold increase in the risk of surgically confirmed gallbladder disease in women receiving postmenopausal estrogens

Adverse effects of oral contraceptives may be expected at the larger doses of estrogen used to treat prostatic or breast cancer or postpartum breast engorgement, it has been shown that there is an increased risk of thrombosis in men receiving estrogens for prostatic cancer and women for postpartum breast engorgement Users of oral contraceptives have an increased risk of diseases, such as thrombophlebitis, pulmonary embolism, stroke, and myocardial infarction Cases of retinal thrombosis, mesenteric thrombosis, and optic neuritis have been reported in oral contraceptive users. An increased risk of postsurgery thromboembolic complications has also been reported in users of oral contraceptives If feasible, estrogen should be discontinued at least 4 weeks before surgery of the type associated with an increased risk of thromboembolism, or during periods of prolonged immobilization Estrogens should not be used in persons with active thrombophlebitis, thromboembolic disorders, or in persons with a history of such disorders in association with estrogen use They should be used with caution in patients with cerebral vascular or coronary artery disease Large doses (5 mg conjugated estrogens per day), comparable to those used to treat cancer of the prostate and breast, have been shown to increase the risk of nonfatal myocardial infarction, pulmonary embolism, and thrombophlebitis When doses of this size are used, any of the thromboembolic and thrombotic adverse effects should be considered a clear risk

Benign hepatic adenomas should be considered in estrogen users having abdominal pain and tenderness, abdominal mass, or hypovolemic shock Hepatocellular carcinoma has been reported in women taking estrogen-containing oral contraceptives Increased blood pressure may occur with use of estrogens in the menopause and blood pressure should be monitored with estrogen use A worsening of glucose tolerance has been observed in patients on estrogen-containing oral contraceptives For this reason, diabetic patients should be carefully observed Estrogens may lead to severe hypercalcemia in patients with breast cancer and bone metastases

PRECAUTIONS: Physical examination and a complete medical and family history should be taken prior to the initiation of any estrogen therapy with special reference to blood pressure, breasts, abdomen, and pelvic organs, and should include a Papanicolaou smear As a general rule, estrogen should not be prescribed for longer than one year without another physical examination being performed Conditions influenced by fluid retention, such as asthma, epilepsy, migraine, and cardiac or renal dysfunction, require careful observation Certain patients may develop manifestations of excessive estrogenic stimulation, such as abnormal or excessive uterine bleeding, mastodynia, etc Prolonged administration of unopposed estrogen therapy has been reported to increase the risk of endometrial hyperplasia in some patients Oral contraceptives appear to be associated with an increased incidence of mental depression Patients with a history of depression should be carefully observed Pre-existing uterine leiomyomata may increase in size during estrogen use The pathologist should be advised of estrogen therapy when relevant specimens are submitted If jaundice develops in any patient receiving estrogen, the medication should be discontinued while the cause is investigated Estrogens should be used with care in patients with impaired liver function, renal insufficiency, metabolic bone diseases associated with hypercalcemia, or in young patients in whom bone growth is not yet complete If concomitant progestin therapy is used, potential risks may include adverse effects on carbohydrate and lipid metabolism

Concomitant Progestin Use: The lowest effective dose appropriate for the specific indication should be utilized Studies of the addition of a progestin for 7 or more days of a cycle of estrogen administration have reported a lowered incidence of endometrial hyperplasia Morphological and biochemical studies of the endometrium suggest that 10 to 13 days of progestin are needed to provide maximal maturation of the endometrium and to eliminate any hyperplastic changes Whether this will provide protection from endometrial carcinoma has not been clearly established There are possible additional risks which may be associated with the inclusion of progestin in estrogen replacement regimens The choice of progestin and dosage may be important in minimizing possible adverse effects

The following changes may be expected with larger doses of estrogen:

a Increased sulfobromophthalein retention

b Increased prothrombin and factors VII, VIII, IX, and X, decreased antithrombin 3, increased norepinephrine-induced platelet aggregability

c Increased thyroid binding globulin (TBG) leading to increased circulating total thyroid hormone, as measured by PBI, T_4 by column, or T_4 by radioimmunoassay Free T_3 resin uptake is decreased, reflecting the elevated TBG, free T_4 concentration is unaltered

d Impaired glucose tolerance

e Decreased pregnanediol excretion

f Reduced response to metyrapone test

g Reduced serum folate concentration

h Increased serum triglyceride and phospholipid concentration

As a general principle, the administration of any drug to nursing mothers should be done only when clearly necessary since many drugs are excreted in human milk

Long-term, continuous administration of natural and synthetic estrogens in certain animal species increases the frequency of carcinomas of the breast, cervix, vagina, and liver However, in a recent, large case-controlled study of postmenopausal women there was no increase in risk of breast cancer with use of conjugated estrogens

ADVERSE REACTIONS: The following have been reported with estrogenic therapy including oral contraceptives. breakthrough bleeding, spotting, change in menstrual flow, dysmenorrhea, premenstrual-like syndrome, amenorrhea during and after treatment, increase in size of uterine fibromyomata, vaginal candidiasis, change in cervical erosion and in degree of cervical secretion, cystitis-like syndrome, tenderness, enlargement, secretion (of breasts), nausea, vomiting, abdominal cramps, bloating, cholestatic jaundice, chloasma or melasma which may persist when drug is discontinued, erythema multiforme, erythema nodosum, hemorrhagic eruption, loss of scalp hair, hirsutism, steepening of corneal curvature, intolerance to contact lenses, headache, migraine, dizziness, mental depression, chorea, increase or decrease in weight, reduced carbohydrate tolerance, aggravation of porphyria, edema, changes in libido

ACUTE OVERDOSAGE: May cause nausea, and withdrawal bleeding may occur in females

DOSAGE AND ADMINISTRATION:

PREMARIN® Brand of conjugated estrogens tablets, USP

1. *Given cyclically for short-term use only* For treatment of moderate-to-severe *vasomotor* symptoms, atrophic vaginitis, or kraurosis vulvae associated with the menopause (0 3 mg to 1 25 mg or more daily). The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible Administration should be cyclic (eg, three weeks on and one week off) Attempts to discontinue or taper medication should be made at three- to six-month intervals

2 *Given cyclically* Osteoporosis Female castration. Osteoporosis —0 625 mg daily Administration should be cyclic (eg, three weeks on and one week off). Female castration—1 25 mg daily, cyclically Adjust upward or downward according to response of the patient For maintenance, adjust dosage to lowest level that will provide effective control

Patients with an intact uterus should be monitored for signs of endometrial cancer and appropriate measures taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding

PREMARIN® Brand of conjugated estrogens Vaginal Cream

Given cyclically for short-term use only For treatment of atrophic vaginitis or kraurosis vulvae

The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible

Administration should be cyclic (eg, three weeks on and one week off)

Attempts to discontinue or taper medication should be made at three- to six-month intervals

Usual dosage range 2 g to 4 g daily, intravaginally, depending on the severity of the condition

Treated patients with an intact uterus should be monitored closely for signs of endometrial cancer and appropriate diagnostic measures should be taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding

Revised October 1987

References:

1. Nilas L, Christiansen C, Rødbro P Calcium supplementation and postmenopausal bone loss *Br Med J* 1984, 289 1103-1106 **2.** Riggs BL Research Directions in Osteoporosis Scientific Workshop, National Institutes of Health, February 9-11, 1987 **3.** Kiel DP, Felson DT, Anderson JJ, et al: Hip fracture and the use of estrogens in postmenopausal women *N Engl J Med* 1987,317 1169-1174 **4.** Christiansen C, Christensen MS, Transbøl I: Bone mass in postmenopausal women after withdrawal of oestrogen/gestagen replacement therapy *Lancet* 1981,1 459-461

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MANHATTAN

Aug 28–Sept 1. **Radiation, Physics, and Biology.** 32 Cat I Credits. NYU Medical Center. Contact: NYU Medical Center, Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

SOUTHAMPTON

Aug 14. **Non-Steroidal Anti-Inflammatory Drug Induced Gastric Mucosa.** 1 Cat I Credit. Southampton Hospital, Southampton. Contact: Charlotte Henry-Sullivan, Southampton Hospital, 240 Meeting House Lane, Southampton, NY 11968. Tel: (516) 283-2600.

AROUND THE NATION

CALIFORNIA

Aug 24–27. **Fifth Annual Physics and Biology of Radiology.** 25 Cat I Credits. Town & Country Hotel, San Diego. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077-1925. Tel: (404) 641-9773.

(*continued on p 30A*)

YOCON®
YOHIMBINE HCl

Description: Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubaceae and related trees. Also in Rauwolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless. Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.

Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:
1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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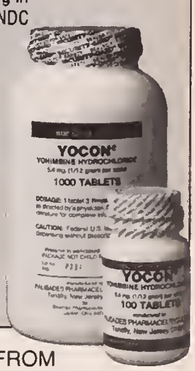

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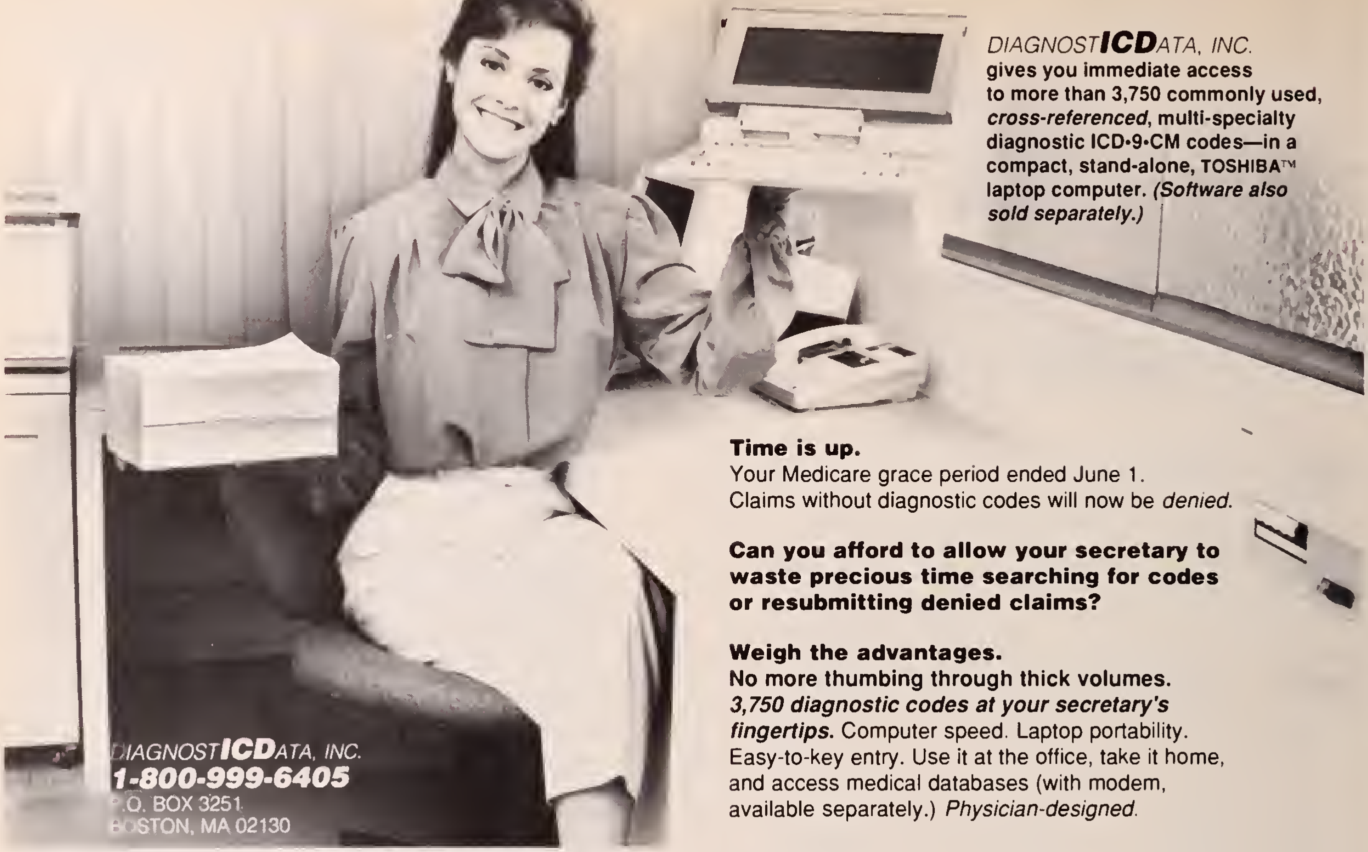

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COMMENTARIES

Strategies for preventing thromboembolism in patients with acute myocardial infarction

Thromboembolic venous disease is a major cause of morbidity and mortality in the United States. It is estimated that more than 300,000 hospitalizations and 200,000 deaths result annually from acute deep venous thrombosis (DVT) and pulmonary embolism.[1,2] When DVT occurs it is frequently followed by the so-called "post-phlebitic syndrome" (ie, leg edema, skin hyperpigmentation, indurated cellulitis, skin breakdown, ulcerations, pain, and other features) which results from persistent venous obstruction and/or chronic venous valvular incompetence. The post-phlebitic syndrome afflicts approximately 0.5% to 5% of the world's population,[3,6] and its treatment (which is usually lifelong in duration) is both uncomfortable and expensive. Prevention is therefore an attractive alternative to the treatment of DVT and its sequellae. Several high-risk situations in which prophylaxis is indicated have been identified and include congenital or acquired primary clotting abnormalities, congestive heart failure, malignancy, inflammatory bowel disease, orthopedic or gynecological surgery, obesity, advanced age, or prolonged immobilization from any cause.[2] Considerable effort has gone into the evaluation of different methods for preventing DVT in these situations. Strategies include full-dose continuous or intermittent intravenous heparin, fixed- or adjusted-dose subcutaneous heparin, heparin plus dihydroergotamine, coumadin, dextran, aspirin and other antiplatelet agents, leg elevation, elastic compression stockings, intermittent or sequential pneumatic compression, and others.[2] Unfortunately, no single method of prophylaxis has proven to be optimal in all situations; indeed, it appears that methods which are excellent at preventing DVT in certain situations may be ineffective in others. Another important consideration is the expense of prophylaxis, since methods that have similar effectiveness (in a particular situation) can differ markedly in cost. The science (and art) of DVT prophylaxis therefore involves matching a given high-risk situation with the appropriate prophylaxis strategy. When done correctly, the chance of developing a DVT and the risk of bleeding (or other side effects) are minimized in the most cost-effective manner possible.

In this issue of the *Journal*, Hollander et al[7] report their findings on the use of intermittent adjusted-dose subcutaneous heparin for the prophylaxis of thromboembolic events in patients hospitalized for acute myocardial infarction.[7] Prophylaxis in this group of patients represents a unique challenge because the potential for thromboembolism is two-fold: 1) prolonged immobilization (perhaps accompanied by a hypercoaguable state?) predisposes to venous stasis and subsequent DVT, and 2) the infarcted myocardium is thrombogenic and can precipitate intracardiac thrombus formation, especially over areas of the ventricle with impaired contractility. The magnitude of these problems is considerable. When sensitive methods of screening for DVT are used (such as radiolabeled fibrinogen scanning) the incidence of DVT in non-anticoagulated patients with acute myocardial infarction is 22–37%, and the associated rate of clinically detectable pulmonary embolism appears to be 4–5%.[7] Ventricular mural thrombus occurs in 42–58% of non-anticoagulated patients who die secondary to acute myocardial infarction, and in 4% of patients this thrombus is the presumed source of clinically evident cerebral emboli.[8] The incidence of ventricular thrombus formation is highest in patients with large or anterior infarcts. Fortunately, the in-hospital occurrence of both DVT and ventricular mural thrombus (along with their associated complications) can be markedly reduced by anticoagulation with intravenous heparin. However, the use of continuous intravenous heparin is awkward (requiring the patient to be attached to an infusion pump), expensive, and associated with a significant incidence of bleeding side effects. In this issue of the *Journal*, Hollander et al[7] examined a selected group of patients who were treated with an alternative strategy. These patients received continuous intravenous heparin while in the coronary intensive care unit, but were switched to adjusted dose intermittent (bid or tid) subcutaneous heparin after transfer to a regular hospital bed. The subcutaneous heparin was maintained until their discharge 7–10 days later. The results demonstrated that partial thromboplastin time could be maintained in the desired range throughout the treatment period, bleeding complications were minimal, and no clinically evident thromboembolic events occurred. There was also the suggestion that appreciable cost savings might be possible when compared to therapy with continuous intravenous heparin. These findings raise the possibility that intermittent subcutaneous heparin could be used in patients with acute myocardial infarction as an efficacious, safe, and cost-effective alternative to continuous intravenous heparin.

However, this study must not be viewed as an answer to the problem of thromboembolism prophylaxis in acute myocardial infarction, but rather as a starting point for investigation. Some of the questions immediately raised by the findings include:

- How does the incidence of thromboembolism, the bleeding rates, and the cost of treatment with subcutaneous heparin compare to those obtained when full dose continuous intravenous heparin is maintained throughout hospitalization? What if the patient is switched to oral coumadin instead of subcutaneous heparin? What about low-molecular weight heparin?
- Which patients benefit most from prophylactic anticoagulation? Do all patients need the same program?
- How long should prophylaxis be maintained in patients with recent myocardial infarction?
- How much heparin is the right amount? Would lesser degrees of anticoagulation protect as well with less bleeding? Would more anticoagulation protect better without raising the rate of bleeding complications? Could subcutaneous heparin be used immediately upon hospitalization for myocardial infarction, or is an initial period of intravenous administration important?

The information contained in the present study provides a starting point from which the important questions outlined above can be explored. Only by incorporating findings from small, provocative studies such as this into large clinical trials will the slow process of finding a "perfect" match between disease state, effective prophylaxis, and cost be made.

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Modes of administration of heparin in clinical medicine

Antithrombin is a 58,000 kilodalton protein that circulates in plasma and is capable of neutralizing the activity of thrombin and other serine proteases of the intrinsic coagulation cascade. Heparin is a polysaccharide related to the naturally occurring sulfated polysaccharides found in tissues throughout the body that catalyze the binding of antithrombin to these proteases of the clotting cascade. When heparin is present, the mucopolysaccharide portion of the molecule binds to lysyl residues on antithrombin and dramatically accelerates the rate of complex formation between antithrombin and the serine proteases it inhibits. In the early 1970s, investigators first suggested that the normal blood vessel might protect itself from thrombus formation by the presence of heparin or heparin-like species on the vascular endothelium. Endothelial-bound heparin-like molecules (heparans) have since been identified and characterized as capable of accelerating the action of antithrombin in the manner described above.[1]

Commercially available heparin has been used to produce anticoagulation for several decades. Heparin has found an expanding variety of clinical applications in recent years, and has been shown to have important properties in addition to its anticoagulant function. As examples, continuous intravenous heparin has recently been shown to lead to a reduced incidence of myocardial infarction in patients with unstable angina[2]; and certain fractions of commercial heparin have been shown to have antimitogenic properties, with the capability to inhibit or regulate smooth muscle cell growth.[3]

Heparin has been commonly used clinically both for the prevention and for the treatment of venous thromboembolism. In 1960, the only randomized, placebo-controlled trial of heparin ever performed in patients with pulmonary embolism showed a significantly lower mortality among treated patients (and was therefore discontinued prior to completion).[4] Heparin has found widespread clinical application since that time.

In order to achieve an effective anticoagulated state, a minimum level of heparin appears necessary. This effective level of anticoagulation is usually based clinically on prolongation of the activated partial thromboplastin time (PTT). A PTT that is at least one and a half times greater than the control value has been considered therapeutic in most studies. In general, bleeding rates during heparin therapy have been studied in randomized trials that have compared continuous intravenous heparin with intermittent intravenous heparin. Patients who receive intermittent infusions of heparin have, in general, had a higher frequency of hemorrhagic complications than those receiving continuous intravenous therapy.[5] However, in many of these comparison studies, the patients on continuous infusion heparin received a lower total dose of the drug, complicating subsequent interpretations.

A number of studies have demonstrated that subcutaneous therapeutic heparinization is safe and effective when compared to intravenous heparin therapy.[6-9] In addition, one recent study carried out in Britain has suggested that subcutaneous heparin (administered as the calcium salt) is more effective in helping lyse existing deep venous thrombus and preventing propagation of clot than is intravenous heparin.[10] In that study, equivalent total doses of heparin were used, and no significant difference between the two treated patient populations was noted with respect to serious complications. Half of the patients receiving intravenous heparin had some minor problem with the constant infusion pump, while half of those receiving subcutaneous heparin had bruising at the injection site.

If one accepts the idea that intermittent subcutaneous dosing of heparin is as safe as is continuous intravenous heparin therapy with respect to serious hemorrhagic complications, then who should receive subcutaneous heparin therapy to achieve therapeutic anticoagulation? While a definitive answer cannot be provided, some guidelines may be suggested based on the growing number of studies utilizing subcutaneous intermittent heparin, such as that by Hollander and colleagues in this issue of the *Journal*.[9] These investigators have demonstrated in a small series that subcutaneous heparin is as effective, safe, and equivalent in its ability to achieve anticoagulation based on in vitro clotting parameters as systemic continuous heparin therapy. In addition, they point out that the cost of intravenous administration is substantially greater than subcutaneous therapy at their institution.

Three factors might be considered when the possibility of intermittent subcutaneous heparinization is being entertained. First, what is the clinical indication for systemic anticoagulation using heparin? This is by far the most important question to be addressed before any form of systemic heparin therapy is initiated. For example, at this point in time it is probably preferable to rely on a continuous, intravenous infusion of heparin in high risk clinical situations that mandate heparin therapy, such as cases that involve patients with proven pulmonary embolism. This recommendation is based on the cumulative experiences with intravenous therapy in such patients and the absence of prospective, randomized trials that compare subcutaneous and intravenous heparin therapy in such populations.

If the clinical setting for intermittent subcutaneous heparin therapy is deemed reasonable or appropriate, one must next consider the relative advantages and disadvantages to inpatient or outpatient administration of subcutaneous heparin in an individual patient. A final consideration is the role of cost factors in selecting the appropriate therapy. The safety and efficacy of subcutaneous heparin in the two randomized studies of therapy in patients with deep venous thrombosis mentioned above[5,6] shed some light on these last two issues. Adequate heparin levels can be successfully maintained over 24-hour periods with twice-daily subcutaneous injections, at a reduction in both cost and complexity of drug administration. The ease of using subcutaneous therapy in such patients depends on whether one has learned to administer the medication in this fashion. Studies such as that of Hollander and colleagues[9] will help to familiarize the medical community with this technique.

Another population of patients in whom subcutaneous heparin therapy has proven useful is the pregnant patient who is in need of systemic anticoagulation, either because of thromboembolic complications or because of an existing prosthetic heart valve. In the ambulatory pregnant patient, in whom warfarin therapy is contraindicated, intermittent subcutaneous heparin may be the most appropriate anticoagulant therapy.[11]

Whatever mode of drug delivery is chosen, the most difficult questions regarding heparin therapy still remain. Should all patients with venous thrombosis be treated with heparin? While general agreement exists that patients with proximal vein thrombosis should be treated with systemic anticoagulation, the treatment of calf vein thrombosis remains a more controversial subject. What is the optimal way to monitor heparin therapy? How long should heparin therapy be continued? What is the relationship, if any, between the mode of systemic heparinization (subcutaneous versus continuous) and complications such as osteoporosis and thrombocytopenia? Many questions remain unanswered or only partially answered in studies to date. Large, randomized clinical trials of intravenous versus subcutaneous therapeutic heparinization in several common clinical populations will help answer some of these questions. In the interim, it is incumbent on the clinician to select the most appropriate form of therapy for individual patients. Intermittent, subcutaneous heparin therapy is clearly efficacious and cost effective in some settings. In the short term, it appears to be relatively safe as well. As clinical trials comparing subcutaneous to intravenous therapy emerge and as our therapeutic costs become more carefully regulated, full dose subcutaneous heparin therapy will begin to find its niche.

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Cocaine

The advantages offered by cocaine to medicine are legend. As little as 20 to 50 mg produces excellent topical anesthesia with concomitant vasoconstriction. A short latency period (two minutes to onset of surgical anesthesia) and a prolonged duration of action (at least 30 minutes) contribute to its classic popularity. Historically, cocaine opened the portals of local anesthesia and thus enormously broadened the vistas of surgical possibility. The medical community accepted cocaine with excitement and optimism. Its greatest virtue, however, was simultaneously its most glaring fault. Its very potency, combined with a narrow margin of safety (maximal dose 150-200 mg) contributed to the systemic toxicity that has markedly limited its clinical application. Cocaine is now legally classified in the United States as a "Schedule II"[1] drug ("high abuse potential with small recognized medical use").[1,2-5]

Cocaine has largely received its notoriety from the second of its dual pharmacologic actions. It is a powerful central nervous system stimulant of relatively short duration and low therapeutic index,[3,6] which has triggered enormous cross-cultural popularity in its illicit use. The "coqueros," or coca leaf chewers, of Peru aptly describe the cocaine-energized work state as "armado"—"armed." Cocaine heightens the sensory and emotional brightness and distinctiveness of the self against its environmental background. Focal awareness may degenerate into a paranoia where everything seems to be threatening the self, the object of the greatest interest in the universe.[1] Cocaine also produces an excited and occasionally rapturous euphoria, and thus creates a dependency unlike that of any drug we have yet encountered.[7] For some individuals for whom the world may seem cruel, capricious, and overwhelming, cocaine appears, however fleetingly, to allay depression and to make the unbearable seem bearable. Tragically, dependency, followed by systemic toxicity, all too rapidly supervene.

Whereas in the 1960s and 1970s cocaine was a glamor drug, now it has been remarketed for the younger, less affluent, and minority populations. The appearance of "crack" has been mirrored by a chaotic incidence of violent crimes as well. One need look no further than the morning paper for current updated references.

The general characteristics of cocaine, as well as the details of its abuse and addiction, are described in this issue of the *Journal* in a timely and comprehensive article by Miller, Gold, and Millman.[8] The informed health professional is directed particularly to this valuable current reference.

The alkaloid cocaine is methylbenzoylecgonine, or, structurally, 2-betacarbomethoxy-3-beta-benzocytropane, with the empirical formula $C_{17}H_{21}NO_4$. Cocaine is the only naturally occurring local anesthetic. It is a tropane—an ester of benzoic acid and the nitrogen-containing base ecgonine.[9]

In the form of its hydrochloride salt, it is a white crystalline powder with a melting point of 195°C. It is the water soluble hydrochloride salt which is available for medical purposes. Pure cocaine alkaloid (base or freebase) is a colorless, odorless, transparent crystalline substance that is almost totally insoluble in water, but freely soluble in diethyl ether. Further, the melting point of freebase is much lower than that of its salt (98°C). Herein lies the ability to volatilize and thus to "smoke" the base.[9-11]

"Crack" cocaine is a form of unrefined or unpurified freebase. Crack is made by combining cocaine hydrochloride with an agent such as baking soda and cooking it in water, creating an oil-like substance.[11] The oil separates from the water and solidifies into small chunks, which can be from 25% to 90% pure. The resulting substance resembles rock salt and can be smoked in pipes and cigarettes. Smoked "crack" cocaine moves rapidly into the bloodstream and reaches target organs (eg, the brain) in one circulation time. This creates an instantaneously high blood saturation; hence, intense euphoria. (If 15 mg of cocaine HCl "snorted" provided a recognized "high," imagine the "rush" of 50 to 100 mg smoked as the base!) Smoking has renewed popularity for another reason: It avoids needle use and thus some of the fear of AIDS transmission.[12,13]

Cocaine undergoes rapid biotransformation in the human body. Plasma pseudocholinesterases and liver enzymes produce hydrolysis, and N-demethylation takes place in the liver. Depending on urine acidity (greater excretion with lower pH), up to 20% of cocaine is excreted unchanged in the urine. Cocaine undergoes metabolism in blood with a half-life of approximately 1.5 hours.[1]

In the normal adrenergic (fight or flight) system, nerve cells transmit by way of the neurotransmitters norepinephrine or dopamine. Cocaine blocks the reabsorption of these neurotransmitters, effecting premature retriggering and hence synaptic hyperexcitability.[1,2,5]

Inhaled cocaine (in freebase or crack form) enters the circulating blood mass almost instantaneously, creating an adrenergic storm. The central nervous system is aroused, stimulating the large mobilizing muscle mass controlled by the reticular activating system. Central areas are stimulated involving fear, anger, hunger, thirst, body temperature, sexual arousal, and the "pleasure center."

Cocaine is truly sympathomimetic. It exaggerates an individual's response to a potentially hostile external environment. In acute overdose, confusion, dry throat, and dizziness are noted. Hyperreflexia and clonic-tonic convulsions may appear. Repeated cocaine usage may lead to depletion of normal cellular neurotransmitters, progressing to exhaustion and finally to a collapse of all life sustaining mechanisms.[7,11,14]

The vasomotor and vomiting centers also share in an early stimulation. Sweating and vomiting are commonly present.[3,7] The action of cocaine on the medulla results in a rapid and shallow respiratory pattern. Depression follows stimulation; irregular or Cheyne-Stokes respiration may appear. Hypoxia ensues, and death may then occur due to a central respiratory depression. A central effect on the heat-regulating center in the diencephalon may contribute to a dangerous elevation in body temperature. Hypertension and hypermetabolism may contribute directly to convulsion and/or central nervous system hemorrhage.[7]

Sinus tachycardia may quickly appear. The sensitive myocardium responds with ventricular ectopic beats. Cardiac standstill may occur due to direct cardiotoxicity; in all likelihood, however, cardiac death is due to ventricular fibrillation.[15,16]

The human body may be able to detoxify one minimum lethal dose of cocaine per hour, or more than 10 g per day. This stated dosage can be misleading, however, due to enormous individual variation. Thus, systemic toxicity to cocaine is thought to be due to an overwhelming adrenergic (or sympathetic) stimulation of the central nervous system and the cardiovascular and respiratory systems.[7,14,15]

In cases of fatal cocaine poisoning, an accelerated onset of symptoms and an accelerated progression through the sequential pattern of the cocaine reaction are seen. Convulsion and terminus may occur within two to three minutes, although death may be delayed for 30 minutes or more.[6] Immediate response is essential in carrying out resuscitative efforts in the course of the dramatically rapid and potentially lethal complications of cocaine overdose.[7]

As the immensity and the immediacy of the problem of illicit high-dose cocaine overdoses and poisonings have presented themselves in physicians' offices and emergency rooms, theories and regimens of treatment have been derived. Physicians must simultaneously work to maintain and stabilize the body's homeostatic mechanisms, while at all times being aware of just what is occurring at cellular receptor sites as well. Physicians must be empiric, in the most expeditious ways; then they must anticipate, theorize, and adjust.

The fulminating course of the cocaine reaction must be immediately and properly blunted. The airway must be secured, adequate oxygenation insured, and vital signs continuously taken. One or more large bore intravenous lines should be placed and kept open with isotonic or hypotonic solution. Acute hypertensive crisis with accompanying malignant encephalopathy is possible; hyperosmolar and hypertonic solutions should be avoided. A dangerous elevation in the body temperature must always be anticipated. Dantrolene should be stocked in the event of malignant hyperthermia.[17]

Core temperature must be continuously monitored. Cooling blankets and ice water for sponging and fans for cooling should be at hand. Hyperreflexia must be anticipated, and tonic-clonic seizures may occur. Seizure activity should be treated with diazepam, 0.25 mg/kg intravenous bolus, repeated as necessary. Metabolic acidosis may significantly affect the predisposition for both seizures and cardiac arrhythmias and should be treated with bicarbonate as indicated.[17]

Uncontrolled hypertension may result in pulmonary and cerebral edema, hemorrhage, or infarct.[14,18,19] The accumulation of catecholamines disposes the myocardium to arrhythmias, which may compromise cardiac output.[14] Further, coronary artery spasm may occur.[19] Propranolol has been widely employed to control the hypertensive crises of cocaine toxicity.[7,12,21] Should tachycardia, hypertension, and/or ventricular ectopy appear, propranolol, 1 mg by intravenous bolus, may be given.[13,21] This may be repeated at one-minute intervals, as with labetalol titrate, to a diastolic pressure of around 90 mm Hg and an apical/radial pulse of less than 100/min.[17] Labetalol offers the advantage of both alpha and beta blockage in attenuating a hyperdynamic cardiovascular state. Other combinations of alpha and beta blockers of short duration, eg, esmolol, should likewise be considered.[17]

All drugs should be titrated to clinical necessity minute-to-minute as monitored by cardiovascular and pulmonary response.[7] Central nervous system and cardiovascular events must be scrupulously monitored in timed sequence. Lidocaine may be employed either as a bolus or as an intravenous drip to suppress ventricular ectopy.[7]

In cases of advanced stimulative stage of cocaine overdose (patient unresponsive and undergoing convulsions), all rules of immediate intervention with life sustaining cardiopulmonary resuscitation measures apply. The mechanics of airway maintenance and external assistance to cardiac output are paramount.

Acidification of urine with NH_4Cl insures a more rapid excretion of cocaine.[22,23] Lithium, dopamine agonists, dopa, bromocriptine, imipramine, and amitriptyline, as well as the amino acids L-tryptophan and L-tyrosine have been discussed as methods to help rebuild depleted dopamine stores, hence helping to reverse the profound depression and listlessness (anhedonia) of withdrawal. (These modalities are to be carried out over at least a 30-day period).[24–26] The action of the benzodiazepines diazepam and midazolam appears to be especially beneficial in relieving anxiety, muscle spasm, and tension.[7]

These life saving efforts are but technical exercises which physicians are able to perform in the acute situation. The rehabilitation of the cocaine user is far more difficult and subtle. It employs every individual and group support system that can be mobilized and utilized for the remainder of the cocaine user's life.

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RESEARCH PAPER

Full dose subcutaneous heparin therapy

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ABSTRACT. Anticoagulation is used extensively for the treatment and prevention of thromboembolic disease. Full dose anticoagulation with heparin is usually administered intravenously via continuous 24-hour infusion to avoid complications with intermittent intravenous administration. Continuous intravenous therapy is costly and cumbersome for nurses and patients. Subcutaneous administration of heparin in full dosage offers an attractive therapeutic alternative. This study examines the safety, efficacy, and cost considerations of this form of therapy.

Fifteen patients admitted to the hospital for myocardial infarction were evaluated. After the patients were transferred from the cardiac intensive care unit, intravenous heparin was discontinued, while therapeutic anticoagulation was maintained via the subcutaneous route. Complications were limited to local hematoma formation. A cost comparison revealed substantial savings with subcutaneous administration.

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Anticoagulation is used extensively for the treatment and prevention of thromboembolic disease. Continuous 24-hour intravenous heparin infusion is generally preferred to avoid excessive complications with intermittent intravenous administration.[1,2] This route of therapy requires careful attention to the administration set-up and the use of intravenous infusion pumps. Subcutaneous administration of heparin in full therapeutic dosage offers an attractive alternative to intravenous therapy. This study examines the safety, efficacy, and cost considerations of full dose subcutaneous heparin therapy in postmyocardial infarction patients.

PATIENTS AND METHODS

The subjects were consecutive patients admitted with acute transmural myocardial infarction, as diagnosed by typical chest pain, electrocardiographic findings, and cardiac enzyme patterns; and without contraindication for anticoagulation. They were initially treated with intravenous heparin therapy to achieve partial thromboplastin times (PTT) one and a half to two times control values. On transfer from the cardiac intensive care unit, the intravenous line was removed, and patients were converted to subcutaneous, full dose heparin therapy, which was continued for the duration of the hospital stay (seven to ten days). The initial heparin dose was calculated by dividing the 24-hour intravenous dose into two or three aliquots and administering this dose every 12 hours or every 8 hours, respectively. Patients who had a greater than four-hour delay between cessation of the intravenous heparin and initiation of subcutaneous therapy received an initial bolus of 20,000 units subcutaneously. Further dosage adjustment was made, based on partial thromboplastin time, to maintain therapeutic anticoagulation.

The patients were followed for efficacy and complications of subcutaneous heparin therapy. Partial thromboplastin time was monitored at four and eight hours after a dose in patients who were on an every-8-hour regimen; and at four, eight, and 12 hours for patients on an every-12-hour regimen. Initial PTT determinations were obtained on the first to second day of therapy, adjustments were made, and PTT determinations were repeated on the third to fourth day of therapy. The determinations obtained after dose adjustment were used to calculate the "average" values reported. Heparin therapy was continued throughout the hospital stay. Patients were followed prospectively for clinical evidence of systemic embolization, deep vein thrombosis, pulmonary embolus, or significant bleeding complications. The patients' hematocrits and platelet counts were followed sequentially.

A cost comparison was made by estimating the cost of continuous intravenous heparin administration versus intermittent subcutaneous administration.

RESULTS

Fifteen patients with myocardial infarction were evaluated. The mean age was 68.3 years (± 7.1 years). The mean 24-hour intravenous dose of heparin was 26,227 units (± 4,820 units) and resulted in a mean PTT of 73 seconds (± 45)/31.5 (± 0.83) seconds. The mean 24-hour heparin dose on subcutaneous therapy was 32,033 units (± 8,041 units). Eight patients were maintained on an every-8-hour schedule, and seven patients were maintained on an every-12-hour schedule. The PTTs after a subcutaneous dose at four, eight, and 12 hours were 64 seconds (± 11)/31.6 (± 1.24), 55 seconds (± 14)/31.9 (± 1.6), and 60 seconds (± 7.7)/31.4 (± 1.13), respectively (Fig 1).

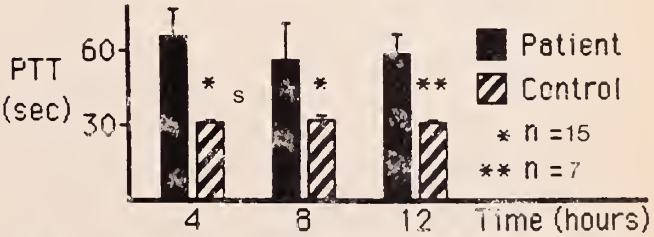

FIGURE 1. Partial thromboplastin time (PTT) measured four, eight, and 12 hours after subcutaneous heparin dose.

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Complications were limited to local hematoma formation seen in two patients. In one patient, who received 20,000 units subcutaneously every 12 hours, a hematoma developed involving his rectus sheath, perhaps secondary to inadvertent intramuscular injection; his partial thromboplastin times were in therapeutic range at the four-, eight-, and 12-hour determinations. In another patient, who received 7,000 units every eight hours subcutaneously, a 4.5-cm hematoma developed at the site of injection. No life-threatening bleeding complications occurred. No patients experienced clinically evident deep vein thrombosis, pulmonary emboli, or systemic emboli. No patients required discontinuation of subcutaneous heparin because of discomfort. Sequential determinations of hematocrit and platelet counts revealed no significant change during subcutaneous heparin therapy.

Cost comparisons are presented in Table I. The cost of continuous intravenous therapy was estimated to be $6.15 per day compared with $2.66 per day for intermittent subcutaneous therapy.

DISCUSSION

In 1952, Artz and Amspacher[3] demonstrated that subcutaneous injection of a concentrated aqueous solution of heparin could provide therapeutic anticoagulation for eight to 12 hours. These findings have subsequently been confirmed in a number of reports.[4-6] More recently, investigators have found that subcutaneous heparin was effective and safe in the treatment of deep vein thrombosis.[7-11] In addition, measured clotting parameters and plasma heparin levels were equivalent to determinations in patients receiving intravenous therapy.

The selective use of anticoagulation in myocardial infarction is an attempt at prophylaxis against deep vein thrombosis, pulmonary embolus, and left ventricular mural thrombi formation, with their ensuing complications. These issues are controversial and widespread. Thus, systemic anticoagulation is not recommended for all patients with acute myocardial infarction. In 1974, Pitney et al[7] reported on the use of full dose subcutaneous heparinization in the prevention of venous thrombosis after acute myocardial infarction. Twenty-one patients treated with subcutaneous therapy were compared to 17 patients treated with intravenous therapy. Deep vein thrombosis occurred in one of the 21 patients treated with subcutaneous heparin and two of the 17 patients treated with intravenous heparin. No serious complications of therapy were noted. Our study of 15 patients who sustained myocardial infarction confirms these previous findings. Subcutaneous heparin therapy was effective in achieving therapeutic partial thromboplastin time with no serious thromboembolic events noted.

TABLE I. Comparison of Cost of Subcutaneous Heparin and Intravenous Heparin for Therapeutic Anticoagulation

Intravenous Heparin (via constant infusion)	
D5W—500 mL/day to maintain IV line	$0.75
D5W—250 mL/day	$0.70
Regular IV tubing 1/day	$1.25
Controller tubing 1/day	$2.70
Syringe/needle 1/1/day	$0.16
Heparin Na+ (5,000 units/mL) – ($1.17/10mL) . . 25,000 units	$0.59
Total cost	$6.15/day
Subcutaneous Heparin (every eight hours)	
Syringes/needles (@$0.16/dose)	$0.48
Heparin Na+ (20,000 units/mL) – ($1.09/mL) . . 30,000 units	$2.18
Total cost	$2.66/day

The estimated cost of intravenous therapy is nearly three times that of subcutaneous therapy at our institution. Rooke and Osmundson[12] recently reviewed the relative cost of various protocols for the use of heparin in patients being treated for deep vein thrombosis. They found the cost of intermittent subcutaneous therapy to be less than one third that of continuous intravenous administration. The cost of intravenous administration in the patients described here is likely underestimated, as it does not take into consideration the additional nursing care required to maintain indwelling intravenous lines, as well as the cost of intravenous infusion pumps.

Complications in these patients were limited to local hematoma, including a rectus sheath hematoma which developed in one patient. This may have been secondary to inadvertent intramuscular injection, although rectus muscle hematoma has been previously recognized during anticoagulation.[13] It has occurred during intravenous anticoagulation as well, and may be related to local trauma. A forceful cough, for example, may induce relatively trivial trauma to the microvasculature in the rectus abdominis muscle, which would ordinarily be unimportant. In a patient undergoing anticoagulation therapy, it may provoke clinically important bleeding and hematoma.

Intravenous heparin therapy is often the only reason to maintain an indwelling catheter. This foreign body often serves as a nidus for local and systemic infection. In addition, patient mobility is greatly restricted, and the patient is often disturbed by annoying infusion pump alarms. The subcutaneous route allows for the discontinuation of the intravenous catheter, thus allowing greater patient mobility, facilitating progressive ambulation, and reducing the risk of infectious complications.

Intravenous administration does not allow for chronic outpatient therapy. Full dose subcutaneous heparin has been used during pregnancy to allow discontinuation of warfarin sodium and to avoid prolonged hospitalization.[14] Changing heparin requirements during pregnancy necessitate careful monitoring in this population; however, therapeutic anticoagulation can be achieved without major complications. If outpatient self-administration proves feasible, heparin may be an attractive alternative to warfarin sodium for long term anticoagulation in many clinical settings.

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FROM THE LIBRARY

PRACTICAL APPLICATION OF HEPARIN FOR LONG-TERM ANTICOAGULANT THERAPY

The superiority of heparin as an anticoagulant over that of the coumarin derivatives has been demonstrated repeatedly in both in vivo and in vitro experiments, and its safety in use as compared to the oral anticoagulants is beyond dispute. Nevertheless, in spite of these obvious advantages heparin has not received widespread use as the anticoagulant of choice in conditions requiring prolonged or long-term anticoagulant therapy because of the disadvantage of repeated intravenous injections.

It is true that with increased experience in the use of oral anticoagulants the incidence of bleeding has decreased markedly, and reports of fatal hemorrhage are rare, not because they do not still occur, but because it is no longer of medical interest to report them. Also, the ready effectiveness of emulsion of vitamin K_1 oxide in combating hypoprothrombinemia has somewhat quieted the qualms of many in the use of these anticoagulants.

The intermittent intravenous injection method of the Scandinavian workers still has the disadvantage of repeated intravenous injections, and the erratic action and pain of the depository heparin has not popularized this method. The introduction of a concentrated aqueous heparin led Stats and Neuhof to the use of this as a deep intramuscular injection, and they demonstrated a good anticoagulant effect for an eight- to twelve-hour period after a single injection. However, the intramuscular injections are extremely painful, and it has been the experience of one of us (M.S.) that patients refuse further injections after several weeks of therapy by this route. Besides, painful hematomas occur occasionally.

The undisputed superiority of heparin as both an effective and a safe anticoagulant makes one wish for a simple method of administering this drug for long-term anticoagulant therapy.

Artz and Amspacher, in 1952, demonstrated that a subcutaneous injection of a concentrated aqueous solution of heparin produced good anticoagulation for an eight- to twelve-hour period, and these findings were confirmed by Engelberg in a number of clinical studies. Engelberg repeatedly demonstrated prolonged anticoagulation with subcutaneous injections the extent and duration depending on the dose of heparin given. He stated that a twenty-four-hour anticoagulation was possible with a single subcutaneous injection if the dose were large enough. . . .

Therapeutic anticoagulation with subcutaneous heparin can be maintained for an indefinite period in the hospitalized patient because of the painlessness, simplicity, and safety of this method of heparin administration. It is not impractical, since an outpatient or office procedure for administration is possible by some member of the family. Subcutaneous injections of many substances—insulin, mercurial diuretics, vitamins, to mention a few—are being administered by lay people today. The parallel and constant effect obtained by injections into the anterior surface of the thigh as compared to the posterior iliac area makes self-administration even more practical. . . .

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(*NY State J Med* 1959; 59:1054–1059)

Cocaine: General characteristics, abuse, and addiction

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The widespread use of cocaine resumed in the 1960s when many illicit drugs gained popularity among young people in the United States. As the use of cocaine increased, reassurance from the medical profession that cocaine use was benign appeared in spite of earlier warnings. *The Comprehensive Textbook of Psychiatry* by Friedman and Saddock[1] contained this quotation: "If used no more than two or three times per week, cocaine creates no problems." Another article in *Scientific American*[2] claimed that cocaine was "no more habit forming than potato chips." Cocaine now ranks as the third most common drug in the United States, after alcohol and marijuana, to which individuals become addicted. The morbidity and mortality from cocaine abuse and addiction are lessons that apparently were forgotten. In the mid- and late 19th century, accounts of cocaine's adverse consequences were frequently presented in the medical literature. The epidemic that occurred in the United States waned in the early 1900s.

Users in the United States currently consume billions of dollars worth of cocaine annually. The cost in lives, in income lost to industry and individuals, and in danger and disruption to society is staggering. The debate over whether cocaine is addictive is left to academicians, as practitioners treat millions of new cases of cocaine addiction yearly.[3,4] Addiction to cocaine is rapid and overwhelming, rendering the user powerless over the choice to abstain or moderate the use of cocaine. The addiction is identified by the intense preoccupation with acquiring cocaine, compulsive use in spite of adverse consequences, and repeated relapse after resolving to reduce or eliminate its use.

PREPARATIONS AND ROUTES OF ADMINISTRATION

Cocaine has many names that reflect its appearance and effects. Coca paste is known as "base," "pasta," "pitillo," and "buscuso." The powdered form is known as "snow," "coke," and the rock form as "crack" because of the sound it makes burning in the pipe. Cocaine is an alkaloid present in the leaves of the *Erythroxylum coca*, a tree or shrub indigenous to western South America. Abundant amounts are found in the wild state in Peru, Colombia, Ecuador, and Bolivia. The coca leaf bears no pharmacologic relationship to the cocoa (cocoa or chocolate) bean or to the kola nut, although confusion between them is reflected in the medical literature.[5]

Coca paste is derived by dissolving dried coca leaves in a solution of kerosene or gasoline, alkaline bases, potassium permanganate, and sulfuric acid. The coca paste contains cocaine, sulfate, other coca alkaloids, diluents, and adulterants. The salt form of cocaine hydrochloride is prepared from coca paste that is mixed with hydrochloric acid.[6] "Freebase" cocaine is the free cocaine molecule that may be prepared from cocaine hydrochloride by extraction with ether or sodium hydroxide. The freebase is the cocaine base itself that has thus been freed from its salt form.[7] The freebase is vaporized easily with flame heat, unlike the salt form, to allow for ready inhalation.

Crack is not a new drug but represents a new strategy in the sale and marketing of street cocaine. Crack is a "rock" form that is sold in tiny pellets of ready-made or prepackaged freebase cocaine without further chemical processing required, as in the case of the powder form or the salt form of cocaine hydrochloride. Crack is 25–100% pure freebase cocaine, depending on the amount of adulterants added. After vaporization, crack is immediately and completely absorbed when smoked in a water pipe. The introduction of crack in rocks in small vials that sell for about $20 has increased accessibility to lower income and adolescent users by virtue of its lower initial cost.

Many of the adulterants and diluents that are added to the coca paste and cocaine hydrochloride are not removed by ordinary extraction procedures for freebase preparation. Many preparations contain manganese carbonate that can produce a Parkinson syndrome; lidocaine, which looks and numbs like cocaine; and ephedrine, which has weak stimulant properties to enhance potency.[10,11]

The major routes of administration for cocaine are intranasal, intravenous, and smoke inhalation. The intranasal route remains the most popular method of use. The majority of frequent and infrequent consumers "snort" cocaine hydrochloride powder through the capillary-rich mucosa of the nose. The intravenous route is preferred by those who often inject other drugs such as opiates. The greatest concern over increased cocaine use has been due to smoking of freebase preparations.[12,13] Smoking takes 5–10 seconds, intravenous injection 30–120 seconds, and intranasal administration 1–3 minutes to produce effects in the brain. The addiction potential may correlate with the faster and higher peak blood levels obtained with

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smoking. Clinical histories of cocaine addicts suggest that the incidence is highest and the course fastest for those who use the inhalation route (days), whereas the intranasal route requires a more protracted course for addiction to occur.[14]

Cocaine freebase is inhaled through the bronchial tree to the capillary-rich alveoli in the lungs which provide a vast surface area (72m^2 of membranes) for instantaneous absorption with the gaseous exchanges. The inhalation route also bypasses the venous system because the pulmonary venous (arterial) blood flow from the lungs to the heart is pumped directly through the aorta into the cerebral circulation and brain. The intravenous circulation is a longer route for cocaine because it follows the systemic return of venous blood through the vena cavas into the heart and eventually to the lungs before returning to the heart for distribution to the brain. The intranasal route is an even slower route that requires passage of cocaine through the tiny capillaries and venules before entry into the systemic venous circulation. Vasoconstriction of capillaries limits intranasal and to a lesser extent intravenous absorption. All routes of administration of cocaine are addicting, but the relative rates and potency differ, with inhalation appearing to be the most potent, followed by intravenous use, then intranasal or oral use.

NEUROCHEMISTRY

Several neurotransmitter systems in the brain may contribute to the many effects of cocaine. There is direct evidence for the involvement of various neurotransmitters in the brain, which include dopamine, norepinephrine, and serotonin. This evidence has been obtained from in vitro and in vivo animal studies and from studies of subjective and objective cocaine effects in humans.

Many cocaine effects may be attributable to the dopamine system. Specifically, cocaine rapidly blocks the re-uptake of the dopamine neurotransmitter into its presynaptic nerve terminal for storage by the vesicles. The re-uptake mechanism is the principal means of termination of action of the neurotransmitter.

Greater concentration and a longer duration of dopamine result from the blockade of the re-uptake mechanism.[15,16] The acute effects of cocaine on dopamine neurons in vitro include enhanced dopamine activity by dopamine re-uptake blockade and by increased dopamine synthesis. Dopamine appears to be intimately involved in the reward system in the limbic system. Dopamine neurons in the central nervous system originate in the ventral tegmental area, and project to the limbic structures that include the nucleus accumbens (mesolimbic pathways) and the limbic cortex that includes the frontal cortex (mesocortical pathways). The neurons of the reward system originate in the lateral hypothalamus. These neurons descend as part of the median forebrain bundle to terminate in the ventral tegmental area to synapse on dopamine neurons. Opiates appear to stimulate reward sensations and behavior by acting on opiate receptors in the postsynaptic neurons in the ventral tegmentum. Cocaine and other stimulants appear to enhance dopamine transmission by acting on the other end of the dopamine neurons at the presynaptic site of the terminal dopamine fibers in the mesolimbic and mesocortical areas.[17,18]

The subjective and objective effects of cocaine in humans can be correlated with the demonstrated actions of cocaine on neurons in various locations in the brain. The limbic system is the substrate for mood, drive states such as hunger and sex, memory, and reward behavior. The basal ganglia is the substrate for motor behavior. The substantia nigra neurons that contain dopamine project to the caudate and putamen. The amygdala and septal areas, which control mood, contain dopamine neurons. Alterations in these nuclei produce observable and reportable mood changes in animals and humans. Electrodes implanted in the septal area in humans produce intense euphoria if stimulated. Similar electrodes placed in the "orgasmic" center near the septal area in humans and animals result in sexual pleasure and behavior (mounting, erection, grooming). Stimulation of the amygdala produces aggressive, angry behavior and destruction of it produces placid, tranquil behavior in humans and animals.[15,16]

EFFECTS

Acute Intoxification: Psychological and Physiological Effects. Cocaine is a central nervous system stimulant similar to amphetamine, methylphenidate, etc. Acute cocaine intoxification produces a variety of physiological effects and virtually any psychiatric symptom. The intensity and duration of the acute effects correlate with the rate of rise and height of the peak of the level of cocaine in the blood. The faster, higher levels of cocaine reaching the brain are reflected by the rapid and sharp increases in cocaine levels obtained in the blood. The route of administration determines the blood level achieved. Inhalation results in the quickest and highest peak cocaine blood levels, followed by the intravenous, then intranasal, then oral routes.

The results of both human and animal studies indicate that the psychological and physiological effects of cocaine depend on the distribution and delivery of the drug to target organs. The brain is a major organ affected by cocaine in the genesis of abuse and addiction. Neurochemical events are initiated by the interaction between cocaine and the brain to produce predictable and stereotypic changes in psychological responses and behavior.

The acute psychological effects begin when cocaine reaches the brain, which is within seconds by any route of administration. The major areas affected are mood, cognition, drive states, and level of awareness. An immediate and intense euphoria, analogous to a sexual orgasm, occurs and may last seconds to minutes depending on the route of administration. Other alterations arising from elevation in mood include giddiness, enhanced self-confidence, and a forceful boisterousness. The subsequent level of mood is milder euphoria mixed with anxiety, which lasts for 30–90 minutes, followed by a more protracted anxious state that exists over a span of hours. The thoughts may race and the person may become talkative, even garrulous, with tangential and incoherent speech. Appetite is suppressed, as is the case with most central nervous system (CNS) stimulants during the intoxication, followed by an increased appetite on the rebound. In low doses, the libido is stimulated, and sexual performance in men is enhanced by a prolonged erection and a heightened orgasm. In high doses, spontaneous ejaculations and orgasm may occur. The level of consciousness is elevated, so that hypervigilant arousal attends the other mental alterations and may lead to insomnia. A loss of self-awareness often occurs with or without a decrease in recent memory, which may lead to acts and decisions of poor judgment. Motor activity is often increased, with agitated and fidgety behavior accompanying subjective sensations of restlessness and perpetual motion.[5,19,20]

The acute physiological effects are manifestations of sympathetic nervous system discharge; these include hypertension, sweating, hyperpyrexia, urinary and bowel retention, muscular contractions, and cutaneous flushing.[14,15] During acute intoxication, the physiological effects are transient and short-lived with low to moderate doses, and are usually benign. No adverse sequelae result unless underlying medical conditions exist that are worsened by a sustained increase in tone of the sympathetic nervous system. The adrenal glands and sympathetic nerve endings contain epinephrine and norepinephrine, the effects of which are enhanced by their re-uptake blockade by cocaine.[5,6,19,20]

At higher doses, marked anxiety, fear, apprehension, loss of control of thoughts and of judgment, frank paranoid delusions, and auditory and visual hallucinations frequently occur. Amnesia, delirium, hyperthermia, stupor, and coma may paradoxically result from this stimulant in large enough doses by any route of administration. These very high dose effects are sometimes seen in "body packers," smugglers who swallow plastic bags of cocaine powder that dissolve in their gastrointestinal tracts. Sometimes the cocaine is absorbed in lethal amounts into the blood stream in such individuals. Seizures, usually of the generalized toxic, chronic convulsive type, occasionally occur, again by any route, and may lead to status epilepticus with sustained convulsions that arrest respiration and induce fatal cardiac arrhythmias. Fatal cardiac arrhythmias may develop with or without seizures. They are most often ventricular and rapid in onset. Atrial tachycardias are not likely to be fatal unless an underlying, predisposing heart problem already exists.[11] Blood pressure may be dangerously elevated, so that in a susceptible individual, stroke with cerebral infarction and/or hemorrhage may be induced. The idiosyncratic reaction or variability of psychological and physiological factors in the individual may be important, in addition to dosage, in determining the severity and type of any toxicity from cocaine.[11,23]

The craving or drive to use cocaine is strongest and most compelling after recent use and diminishes with continued abstinence, precipitously in the first few days and more gradually over weeks. The craving or drive to use cocaine may reappear even after prolonged abstinence, especially if addictive use has occurred.[6,21] Cocaine addicts and users report that impulsive urges spontaneously arise in response to environmental cues after years of abstinence from cocaine.

Chronic Intoxication: Psychological and Physiological Effects. Chronic, persistent and regular use of cocaine by any route of administration has characteristic consequences that are easily identified if the clinician is informed and alert. Chronic administration of cocaine produces virtually all of the psychiatric syndromes/disorders, ie, affective disorder, schizophrenic characteristics, and personality disturbances. Tolerance to and dependence on cocaine readily occur, and the withdrawal from chronic cocaine use also has a predictable and typical, protracted psychological and physiological course.[22,24,25]

The development of tolerance is reflected by the waning of some of the acute effects of intoxication, which become less intense and shorter in duration with continued, chronic use. The euphoria lessens with the onset of tolerance. An extremely interesting phenomenon is that most cocaine addicts recall that the very first or initial trial of cocaine yielded the greatest and most satisfying euphoria. Subsequent episodes of cocaine-induced euphoria are not as intense or as long, even with larger doses taken more frequently. The effects of euphoria increasingly diminish with further cocaine use over months and years in spite of increasingly higher doses—up to several grams of cocaine per day. The addict in a sense continues to chase the initial "high," with greater intensity but with lesser results. Furthermore, users experience less euphoria with successive use within a short time frame over hours, despite freebasing almost continuously within a binge.

Other adverse effects that accompany repeated cocaine use include increased anxiety and depression. The addict may experience profound depression with sensations of helplessness, hopelessness, and suicidal thinking while continuing the compulsive use. Paranoid delusions are common and quite distressing to the addict who mistakenly believes others are "spying on him" or "out to get him." A less intense but pervasive suspiciousness and paranoia are persistently experienced by the addict. Derogatory auditory and bizarre visual pseudohallucinations are less common in the chronic user. (The pseudohallucinations are experienced by the cocaine user as "not real.") Insomnia, depressed appetite, and intense anxiety combine to produce ferocious hunger, fatigue, and eventual physical and mental exhaustion from the effects of the drug. The cocaine addict is typically irritable, suspicious, anxious, dysphoric, and guarded in less severely intense intoxicated states that alternate with the sometimes high dose use during intoxication.[25,26]

The libido is depressed and sexual performance is impaired with consequent impotency in males and inorgasmia in females during chronic cocaine use. Men have difficulty in achieving and maintaining erections and experiencing ejaculations, whereas females are sexually unresponsive and inorgasmic following prolonged bouts of cocaine intake. Muscular twitches, tremors, and weakness are common with chronic use, especially with high dose inhalation. Nasal ulcers and bleeding and chronic sinusitis are almost inevitable consequences of chronic intranasal use. Weight loss, sometimes extreme, malnutrition, and poor self-hygiene and care result from heavy, prolonged cocaine use.

The acute and prolonged withdrawal from cocaine in the abstinent state following chronic and regular use does occur and is dramatic, but is different from that seen with opiates. Opiate or narcotic withdrawal is characterized by piloerection, nausea, muscle cramps, distress, anorexia, diaphoresis, and intense craving or drive to use opiates. It has a peak period between 24 and 72 hours and subsides gradually over days. Cocaine withdrawal has an initial, acute phase that lasts from hours to days and is termed the "crash." During the crash, an intense depression, fatigue, hypersomnolence, hyperphagia, and drive for repeated cocaine use are present. These signs and symptoms are opposite in direction from the state of intoxication, and are bona fide expressions of withdrawal and a physiological exhaustion. The later phase (weeks to months) is characterized by mood lability with alternating anxiety and depression, anhedonia, low energy, sleep disturbances, suspiciousness, and recurring "urges" for cocaine that arise spontaneously and in response to cues in the environment. These symptoms subside gradually over months and perhaps years in some heavy chronic users, although the urge or drive to use cocaine may be stimulated voluntarily or involuntarily at any time, and for an indefinite time into the future.[26]

ADDICTION, TOLERANCE, AND DEPENDENCE

Addiction to cocaine is defined as the preoccupation with the acquisition and compulsive use of cocaine, with relapse. Loss of control is pervasive and is the sine qua non of addictive behavior. The cocaine addict continues to seek and use cocaine in spite of sometimes overwhelming psychiatric, medical, legal, and financial consequences.

Tolerance and dependence regularly occur but may be difficult to ascertain in the clinical interview because of denial of the extent and amount of use of cocaine by the addict. Cocaine addicts frequently increase the amount of use dramatically in an attempt to maintain certain cocaine effects. The dependence syndrome is characterized by the withdrawal or "crash" that occurs in the abstinent state.[24,26] Tolerance to and dependence on opiates readily occur, although they are greater and more clearly definable in the case of narcotics. The addiction to either cocaine or opiates is similar in the strength and type of preoccupation with acquisition, compulsive use, and relapse to either drug.

The strength of the addiction is illustrated by experiments performed on Rhesus monkeys. These monkeys were allowed to self-administer cocaine by pressing a bar. The cocaine was available in unlimited amounts over indefinite periods of time. All the monkeys pressed the bar to receive cocaine until inanition and death. The loss of control for self-administration of the cocaine was sufficiently potent to produce disregard for the instincts of self-preservation. The behavior in the Rhesus monkeys is readily seen in humans who pursue cocaine without regard for health and welfare, as manifested by loss of large sums of money, liberty, and life.[15,17]

Tolerance and dependence are expected reactions of the body to the presence of a foreign drug. Tolerance and dependence are not specific for addiction, however, and occur in the absence of addictive use. Tolerance is an adaptation of a cell membrane or receptor to the drug. As with cocaine, the dopamine receptor is altered by decreasing in number in response to excessive amounts of dopamine by cocaine re-uptake blockade. Dependence may be viewed as the return of the receptor number to normal and expressed in the corresponding functions for dopamine, manifested by the symptoms of withdrawal.[16,17]

METABOLISM

The metabolism of cocaine in humans involves both blood and liver enzymes. Cocaine is rapidly metabolized into a number of water-soluble metabolites that are excreted in the urine. The major metabolites in the urine are benzoylecgonine and ecgonine methyl ester that result from hydrolysis of cocaine by the action of esterases in the liver and in the serum. Some benzoylecgonine may be formed nonenzymatically in vivo. These metabolites are water soluble.[27,28] Ecgonine methyl ester may be present in large quantities during the first 12 hours. Cocaine plasma concentrations fall rapidly after ingestion to amounts detected only by highly sophisticated analytical methods. Benzoylecgonine concentrations have a slower rate of excretion, so that it is detectable in the urine longer than cocaine and ecgonine methyl. Consequently, most analytic tests measure benzoylecgonine.[27]

As previously noted, following the administration of cocaine by the intranasal, intravenous, or inhalation route, peak blood levels are reached in seconds to minutes depending on the route and purity of the drug. The half-life of cocaine is approximately one hour (ie, it takes the body one hour to excrete half the drug). The short half-life contributes to the rapid administration of cocaine during binges, in which an inhalation may be repeated as often as every five minutes.[28]

NATURAL HISTORY OF DRUG USE IN COCAINE ADDICTION

Alcoholism and other drug abuse diagnoses are highly prevalent among cocaine addicts. Ninety-eight percent of cocaine addicts have used marijuana before initiating cocaine use. More than 80% have used alcohol before using cocaine. Furthermore, alcohol is used before marijuana in the sequence of drug use in the natural history of the progression to cocaine addiction.[29]

In a study of 263 inpatients who were given a structured psychiatric interview, 83% qualified for DSM-III-R diagnoses of cocaine and alcohol dependence. Alcohol was the first drug used, at an average age of 14 years, followed by marijuana at an average age of 15 years, and eventually cocaine at 22 years. These findings also correlate with others that confirm 80% of alcoholics below the age of 30 use at least one other drug addictively, often marijuana and then cocaine.[30]

INTERVENTIONS

Acute detoxification or "crashing" from cocaine frequently does not require more than supervised supportive psychological treatment by professionally trained nurses and physicians. The depression, somnolence, hyperphagia, anxiety, paranoia, and craving subside gradually over days. No significant alterations in vital signs, withdrawal seizures, or delirium usually occur. Relapse to cocaine usually results, however, unless some outside, experienced intervention is provided.

Acute hospitalization may be indicated for special consequences of chronic cocaine use that include suicide risk, inability to abstain from cocaine and other associated drug use such as alcohol, which may require supervised detoxification. Acute detoxification is not sufficient to prevent relapse. Professional treatment and rehabilitation aimed at the addictive process are required in order for the addict to achieve and maintain the desired goal of abstinence. Abstinence is necessary, as the cocaine addict, by virtue of the addiction, is unable to moderate the amount and frequency of cocaine intake so as to avoid adverse consequences.

Pharmacologic interventions may include benzodiazepines (chlordiazepoxide) for the anxiety and agitation that is especially intense the first few days. Chlordiazepoxide given on an as-needed basis in 25-mg to 50-mg doses every four to six hours for the first one to three days is usually sufficient to control anxiety and agitation. Some centers employ the tricyclic antidepressants desipramine and imipramine to neutralize the signs and symptoms of the crash. The usual doses are desipramine, 100–200 mg orally or imipramine, 150–250 mg orally in one or more divided doses. The duration may be two weeks to two months depending on the severity of the dependence syndrome. The greater the depression and craving, the longer the need may be for the antidepressants. Also, the longer and more intense the use as measured by chronicity and dose, the longer and higher the dose may be for the antidepressants. More often, the need for antidepressant medication is short term, in low to moderate doses or none at all. Bromocriptine, a dopamine agonist, is used by some, although its efficacy has not been proven. The theory is that the dopamine depletion is overcome by the dopamine agonist effect of bromocriptine. The usual doses are listed in Table I. Beta blockers, such as propranolol, may be effective in neutralizing the peripheral effects of the catecholamine by blocking the sympathetic effects at the target organs. However, as the sympathetic effects occur in intoxication, beta-blockers are not required in detoxification but may be used during intoxication.[31,32]

The tricyclic antidepressants, bromocriptine, and propranol alone will not significantly promote abstinence from cocaine. These agents may help reduce the relapse to cocaine in the ensuing weeks only if combined with other group and individual therapies, and self-help groups such as Alcoholics Anonymous, Narcotics Anonymous, and Cocaine Anonymous. Effective inpatient and outpatient treatment exists. Abstinence-based programs for other drugs such as alcohol also work for the cocaine addict. The long term management rests with the group and individual therapies and the self-help groups.

CONCLUSIONS

Cocaine addiction reminds us of the devastation that accompanies any activity that consumes the mind, body, and spirit. Cocaine addiction is identifiable by the intense preoccupation with acquisition as evidenced by huge sums of money, in the billions of dollars, exchanging hands in the United States. The compulsive use of cocaine in spite of adverse consequences continues to invade towns and cities, overtake children, and rob individuals of dignity and health. The relapse to cocaine is frequent in spite of costly and life threatening consequences, because of its potent addictive nature.

Effective treatment is available. The morbidity and mortality can be significantly reduced by prevention. Prevention is initiated by education and a willingness to eliminate cocaine as an alternative. An awareness of the catastrophic power of cocaine hopefully will lead to its return to a chapter in history in which it will only be remembered.

TABLE I. Bromocriptine Regimen for Cocaine Withdrawal

Day	Bromocriptine Dose* (mg)	Frequency
1–2	1.25	twice a day
3–4	1.25	three times/day
5–6	2.5	twice a day
7–11	2.5	three times/day
12–14	5	twice a day
15–20	†	†

* Titration of the dose is necessary with anticraving and antiwithdrawal effects weighed against possible side effects. Lower doses than those listed above are often sufficient. Maintenance bromocriptine may be necessary if symptoms recur after discontinuation.

† Decrease by approximately 50% every two days.

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COCAINE AND THE SO-CALLED COCAINE HABIT

New York Neurological Society Meeting of November 2, 1886

Dr. W. A. Hammond [see the article in this issue on the history of the *New York Medical Journal—Ed*] made some remarks upon his personal experience with some of the preparations of cocaine. He had used only the fluid extract and various wines of coca and hydrochloride of cocaine. The fluid extract had been discarded by him for the last two or three years, mainly because it had been badly borne by the stomach; it excited nausea and was disagreeable to the taste. He then began the use of the wines, but, finding that they differed so much in their effects, he gave them up until he suggested to Thurber & Co. to try to make a wine of coca free from tannin and extractive matters, and they had, he believed, entirely succeeded in doing so . . . .With this preparation he had experimented extensively, not only upon others, but upon himself. He had used it in spinal irritation with excellent results—results which could not be attributed alone to the wine, but in part to the cocaine. He had used it also as a general tonic and for fatigue. For some time past he had been in the habit of taking a wineglassful at the close of his day's duties, and with benefit. It certainly had a decidedly restorative effect without being followed by a feeling of depression. He had also used it in some cases of dyspepsia with a very irritable state of the stomach . . . .It was remarkable to what an extent the irritability of the stomach was overcome by doses of two or three teaspoonfuls of the wine of coca repeated at intervals of fifteen or twenty minutes, until half a dozen doses had been taken . . . .

He then spoke briefly of the physiological effects of coca, and said that the first writer who had described its effects upon the native Indians of South America had given an exaggerated account of its baneful influence . . . There had recently been some very striking stories in the newspapers regarding the injurious effects of the drug upon persons who had become addicted to its use. In order to determine whether or not there was any truth in these statements, he made some experiments upon himself. He first injected hypodermically a grain of the hydrochloride of cocaine, which caused an exhilaration of spirits and a happier state of mind than he had enjoyed during that day. He was unable to sleep that night until four or five o'clock in the morning, and when he got up he had a severe headache. He also had a large evacuation of urine. The effect of the drug was to produce an exhilaration such as would be produced by two or three glasses of champagne. The next night he injected two grains, which produced the same pleasant feeling, and in addition he felt an inordinate desire to write. He wrote eight or ten pages of foolscap, and thought it was the best that he had ever written, but the next morning he found that it was the most extreme nonsense . . . .The next night he injected three grains, and, although he again felt the disposition to write, he did not indulge it, but he talked a great deal, and made speeches. He knew what he was about, and was able to restrain himself, but it was pleasant to speak. He went to sleep late, and again awoke with a severe headache . . . .Three nights later he injected eight grains, with about the same effects. The next night he injected eighteen grains, making six different punctures, all within the space of twenty minutes. He became intensely exhilarated, and was unable some hours afterward to recall what he had done. He was in his office, but in some way got to bed, and the next day he found things in more or less disorder in his office. His headache remained for two days, and there was over-action of the heart, palpitation; he could hear it beating on raising his arm to his head. Exaggerated action of the heart had also attended the smaller doses. But he experienced none of the horrible effects which were said to attend the use of the drug in large or continued doses; no disposition to murder or to acts of violence. He acquired no habit; he was able to quit its use at once . . . .

As to the cocaine habit, he regarded it as similar to the tea or coffee habit, and unlike the opium habit. He did not believe there was a single instance of a well-pronounced cocaine habit, the patient being unable to stop it at any time if he chose to do so . . . .

Dr. J. B. Mattison, of Brooklyn, could not agree with Dr. Hammond that there was not a cocaine habit. Within a few months he had had seven cases of the cocaine habit under his care—five in physicians, two in druggists. He certainly believed there was such a thing as cocaine addiction . . . .

(*New York Medical Journal* 1886; 44:637-639)

A proposal concerning products liability

NEIL J. NUSBAUM, JD, MD

Personal injury litigation has had a major direct impact on physicians in the form of medical malpractice lawsuits. There has been debate regarding the ways in which malpractice law can affect medical practice, including the prospect that physicians will shun those areas of specialization in which the threat of malpractice litigation is highest.[1] Recent trends in another field of the law, that of products liability, likewise can influence medical practice, and in ways that may be detrimental to the public health.

THE IMPACT OF LIABILITY LAW ON MEDICAL PRODUCTS

The issue of products liability has become a complex one in our society, the full ramifications of which are beginning to become clear to the wider society, not just the legal community. The range of "products" which have proven fatal in recent years range in complexity from the space shuttle *Challenger* to tobacco products. The injured class accordingly varies in level of sophistication from a small group of astronauts who had spent months studying the shuttle before the accident, to a broad class of cigarette smokers (including many who began smoking as children) who vary widely in the extent of their knowledge of the health hazards of smoking. Of most immediate concern to the health professions, medicines and medical devices have also been affected by the products liability law, and it is submitted that this application of the law sometimes runs counter to the interests of society as a whole.

American legal theory has previously recognized in the abstract that there are reasons why drugs should be treated gingerly by the products liability laws. As stated in a prominent academic compendium on the law of torts, using a somewhat archaic example, there are some

> ... unavoidably unsafe products. There are some products which, in the present state of human knowledge, are quite incapable of being made safe for their intended and ordinary use. These are especially common in the field of drugs. An outstanding example is the vaccine for the Pasteur treatment of rabies, which not uncommonly leads to very serious and damaging consequences when it is injected. Since the disease itself invariably leads to a dreadful death, both the marketing and the use of the vaccine are fully justified, notwithstanding the unavoidable high degree of risk which they involve. Such a product, properly prepared, and accompanied by proper directions and warning, is not defective, nor is it unreasonably dangerous. The same is true of many other drugs, vaccines, and the like, many of which for this very reason cannot legally be sold except to physicians, or under the prescription of a physician. It is also true in particular of many new or experimental drugs as to which, because of lack of time and opportunity for sufficient medical experience, there can be no assurance of safety, or perhaps even of purity of ingredients, but such experience as there is justifies the marketing and use of the drug notwithstanding a medically recognizable risk. The seller of such products, again with the qualification that they are properly prepared and marketed, and proper warning is given, where the situation calls for it, is not to be held to strict liability for unfortunate consequences attending their use, merely because he has undertaken to supply the public with an apparently useful and desirable product, attended with a known but apparently reasonable risk.[2]

These broad principles, however, may not be enough to shield the manufacturer of a product from litigation. Witnesses may proffer testimony that a particular product was a teratogen[3] or produced immunologic defects leading to cancer,[4] even if most experts disagree with those hypotheses. In many cases, the court will decide that a sufficient factual dispute has been raised that the issue should be decided by the jury. Courts have varied considerably in their willingness to allow jurors to consider hypotheses which are not fully accepted by the scientific community[5,6,7] and/or regulatory authorities such as the Food and Drug Administration.[8]

Some of these same issues are now being addressed by the European nations as well. In Britain, whose products liability law is less strict than that of some of the other Common Market countries, a drug manufacturer may avoid liability if "the state of scientific and technical knowledge at the relevant time was not such that a producer of products of the same description as the product in question might be expected to have discovered the defect" in the drug he produced.[9] The British likewise have had to confront the issue of the quality of information provided by drug companies to prescribing physicians,[10] and in one case a pharmaceutical company and its medical director were convicted of making misleading claims as to the benefits of an anti-inflammatory drug in an advertisement in a medical journal.[11] European regulatory authorities have also had to confront the issue of weighing risks and benefits of a drug; in the case of isotretinoin, the European nations have adopted a variety of approaches, many of them being much more restrictive than that of the FDA.[12,13]

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Most of the states have confronted the issue of liability in the case of blood products. They have classified blood transfusion as a "service" rather than as a "product." The legal effect is to shield the blood bank from suit if it follows accepted transfusion testing procedures, even if the unit of blood transmits hepatitis to the recipient.[14] One could argue the point whether blood transfusion is more like a service (transfusion) or a product (blood). The more fundamental issue is that society has recognized that blood transfusion is a procedure with known morbidity and mortality even when done optimally, and that society is not served by allowing litigation when injury occurs in the absence of negligence. This still leaves some room for suits against blood banks on a negligence basis.

The application of these rules in the setting of blood transfusion is of particular relevance in light of the accumulating number of cases of transfusion-associated AIDS arising from blood products administered before HIV serologic testing of blood products was instituted. In this setting, plaintiffs have charged defendants with negligence, with varying legal results.[15,16] One hopes that the courts will not permit broad allegations of negligence to be used as a back door to impose liability on blood banks for transfusions given according to then-accepted practice.

The issue of products liability is one that impacts most directly on the manufacturers of medical products. In at least one instance, however, a drug company has tried to turn physicians' fears of their own potential legal involvement in products liability suits into a marketing tool. One manufacturer has instituted a "physician's product liability protection policy" protecting the prescribing physician from products liability suits brought against the physician for prescribing that company's product; but only if the patient receives that company's version of the drug rather than a generic or other substitution.[17] This advertising approach focuses on fears of physician-prescribers; another drug company, by contrast, has advertised to physicians a limited program of patient refunds if the physician believes the drug "fails to achieve the desired therapeutic benefit."[18,19]

A Proposal for Limited Protection from Litigation

The primary issue concerning medical items, whether they are drugs, blood products, vaccines or medical devices, should be whether the risk/benefit ratio is such that they have a place in medical practice. If that test is passed, they should be regarded as reasonably safe, and liability should not attach for their manufacture and sale. If at some future time additional evidence (ie, post-marketing reports of adverse drug reactions) makes the risk/benefit ratio unfavorable, then the drugs should be withdrawn. The emphasis here is on a federal regulatory body making the decision based on the scientific evidence before a product is allowed to be sold, rather than on suits brought after use by parties alleging that they are injured by a product that should not have been sold.

This approach would offer manufacturers more uniform and predictable rules to follow. It also has the potential to protect consumers from injury before the fact, rather than just compensating them afterward. In this regard, the scheme suggested here should be distinguished from recent proposals for federal legislation to limit the scope of state product liability law.[20]

If the FDA approval process for a drug is to be not only a legal shield for a manufacturer, but also an effective protection for the consumer, vigorous funding of the FDA operations including post-marketing surveillance will be important. One potential route to raise funds for such enforcement activities would be to levy a fee on drug sales. These funds could come in part from any savings of the drug companies on product liability insurance under this scheme, and so might not produce a net rise in drug prices. Orphan drug products would pay little into the fund, because of their small sales volume, so this proposal would not present an economic disincentive to orphan drug development.[21]

The scheme advocated here looks at medicines and medical products as a group of goods in which there are important but competing public interests. They would be closely regulated, but their prompt release would be encouraged once there is reasonably strong evidence of safety and efficacy. The same rationale should apply to drugs, blood products, intrauterine devices,[22] or cardiac pacemakers.

Most manufactured goods are regulated much less heavily by the federal government, and in many cases there is no urgent societal need for the product. In such cases, the goal of compensating an injured party becomes relatively more important, even if the application of products liability law to the product might make a manufacturer less likely to market the product. One might thus be willing to apply a stricter products liability standard to a recreational vehicle that killed a child driver than to a pediatric medication that caused a fatal reaction.[23]

The rule proposed here is predicated on the idea that the regulatory process is relied upon to protect consumers, and that accordingly the federal regulators must have all relevant information at their disposal. In order to make such a system operate effectively to protect consumers, it is crucial that the FDA have full information regarding the product, including any post-marketing data.[24] One powerful weapon to encourage continuing full disclosure by the manufacturer to the FDA would be to provide that a material nondisclosure would void any special protection from a products liability suit. The rationale here is that, if the manufacturer has not allowed the FDA a fair opportunity to review the product in advance to protect consumers, then the consumer should have the full benefit of state products liability law to seek relief after he has suffered injury. This remedy would be in addition to possible criminal penalties against the manufacturer.[25]

One can also consider whether promotion, or over-promotion, of a medical product should cause the manufacturer to lose the benefit of a medical products liability rule. Such promotional efforts can even include patient testimonials by a sports celebrity,[26] may involve prescription or over-the-counter drugs, and at times have involved claims that exceed those approved by the FDA.[27,28] Such advertisements have in recent years increasingly been directed to the ultimate consumer as well as to physicians.

The easiest case to consider is that in which a drug company sells an over-the-counter product and makes advertising claims directly to the consuming public, on which the public may act directly without the intervention of a prescribing physician. If a patient uses aspirin for an indication not recommended by the FDA, but recommended nevertheless by the manufacturer's advertisements, it is reasonable that the manufacturer should be subject to the usual products liability rules.

The balancing of risks and benefits may differ from one drug to another. The FDA has already acted to limit tightly the use of isotretinoin because of its teratogenic effects.[29] On the other hand, the FDA has been moving to liberalize the testing procedures for new drugs for AIDS and other life-threatening illnesses.[30] It can be anticipated that drugs approved for marketing on a fast track are more likely to have their complications appear only in post-marketing surveillance, but this is one of the factors that the FDA could properly consider in deciding when to give marketing approval.

The argument presented here is based on the idea that the patient is voluntarily exposed to the risks of the drug but is also the one who stands to benefit from the drug. The situation may be different, however, when the benefits and risks do not all flow to the same person. Vaccinations cause their adverse effects primarily in those who receive them, but the herd immunity protects the larger society as well as the person vaccinated. Vaccination is often not a voluntary choice of the child or the parents, but a mandatory requirement for entering school. Congress recognized some of the special circumstances surrounding childhood vaccination and enacted the National Childhood Vaccine Injury Act to provide a scheme to compensate injured vaccinees.[31,32]

Government regulatory efforts in practice sometimes are less strict for health-related products than they are for medicines. The Food and Drug Administration has only recently moved to regulate absorbency ratings for tampons, despite their association with toxic shock syndrome.[33] In the case of a water purifier which reportedly could contaminate water with methylene chloride, several federal regulatory agencies were involved but none actually ordered a recall of the product.[24] In the case of another product, "smokeless" cigarettes, the manufacturer has disputed FDA jurisdiction.[35] It is not proposed that peripheral government regulation should protect a manufacturer from a products liability suit; the proposal discussed here is predicated on the assumption that FDA regulation will be as strict as physicians have come to expect for the approval of prescription drugs.

In summary, it is important that drug development and marketing not be excessively hindered by manufacturers' fears of products liability litigation. There is a profound public interest in insuring that promising new drugs reach the market promptly, once their safety and efficacy have been reasonably demonstrated. This goal is better accomplished by means of vigorous regulation and evaluation by the Food and Drug Administration than it is by the products liability law.

The most direct way to achieve this result would be federal legislation providing that use of any FDA-approved drug or device should be deemed a service rather than a product, and so put outside the scope of state products liability laws. Under the broad powers granted to the Congress by the federal constitution,[36] it could certainly preempt state tort law in this fashion if it chose explicitly to do so.

In the absence of express congressional action, the federal courts must try to interpret the laws to divine what Congress had intended. This was one of the issues raised in a recent lawsuit brought on behalf of Terry Abbot, a four-year-old with neurologic injuries that allegedly were produced by a diphtheria/tetanus/pertussis (DTP) vaccination. The trial judge ruled that federal law controlled the case, and on this basis granted judgment in favor of the vaccine manufacturer. This decision was reversed on appeal, when the US Court of Appeals held that even though the vaccine and its design were FDA-approved, a lawsuit could still be brought under Virginia state law arguing that the design of the vaccine was defective.[37] The United States Supreme Court declined a petition to hear the case,[38,39] without discussion of the merits, so it remains unclear how the Supreme Court might rule on this matter in some future case.

The individual states also have the ability to modify their own tort laws, as some indeed have done in the area of blood transfusion. The more practical and comprehensive way to approach the issue of product liability for medicines and medical devices, however, would be at the federal level. Drugs are brought to market in the United States at a national level, rather than state by state, and the severity or laxity of a single state's products liability laws would have only a modest impact on a manufacturer's decision to market a drug nationally.

In conclusion, what is needed is a federal statute to limit the application of state products liability laws to drugs and medical devices. Such legislation could protect a manufacturer from the threat that a jury might brand as dangerous and defective a product that the FDA had approved as safe and effective. It thus would reinforce the role of the FDA as the primary regulator of drug marketing. More important, it would help insure that new drugs are promptly made available to physicians and their patients. In order for such a broad grant of power to the FDA to be politically acceptable, however, it is crucial that the FDA be seen to have both the skill and the resources effectively to monitor drug safety.[40,41]

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The prima facie tort rule: A possible medical-legal answer to the medical malpractice crisis

THEODORE SCHUSSLER, MD, JD

James Sammons, MD,[1] executive vice-president of the American Medical Association, stated in 1985 that there were no "ten easy steps" to solve the medical malpractice crisis and suggested various areas for consideration and problem-solving, such as risk management, tort reform, and coalition building. Various other authors[2] have contributed their expertise to this problem. Robertson,[3] a physician risk management expert, identifies major high litigation risk areas, using case studies, and presents risk management topics. Danzon,[4] an economist, delves into the ramifications and emotional and financial costs that malpractice suits hold for society, and states that malpractice is increasing in spite of enacted reforms.

LEGISLATIVE EFFORTS

Various states have opted for legislative efforts to deal with the malpractice crisis, protect physicians, and limit their liability. In 1985, New York State approved a bill to reform the system of medical malpractice adjudication as well as to reduce contingent fees to lawyers and to grant potentially higher and significant costs against malpractice plaintiffs for frivolous claims and lawsuits.[5]

In 1986, New York State amended its Civil Practice Law and Rules to require a plaintiff suing for medical malpractice to attach a certificate of merit to his complaint.[6,7] The certificate of merit must allege that the plaintiff's attorney has reviewed the facts of plaintiff's case with a duly licensed physician who is knowledgeable in the particular area in contention and has concluded that there is a reasonable basis for bringing the action.

If a certificate of merit had not been served, a plaintiff must then present a reasonable excuse for not complying with the statute and submit proof as to the merits of his case or face the possibility of having his claim dismissed.[8]

The amended rules also provide the basis for awarding costs against a malpractice plaintiff for a frivolous action, up to $10,000.[9] However, in *Favelli v Adler*,[10] the court held that a suit was not frivolous as a matter of law if questions of fact existed regarding vicarious liability by virtue of a partnership, joint practice, or a partnership by estoppel.

The law was also amended to provide for periodic payments in malpractice cases, or "structured judgments,"[11] as well as providing the basis for determining the judgment to be entered,[12] the form of security for periodic payments,[13] the posting and maintaining of security for periodic payments,[14] what to do in case of failure to make payment,[15] and the effect of the death of a judgment creditor on payment.[16]

In addition, New York State's effort to hold down the rising costs of medical malpractice premiums has been upheld by the courts.[17] The Illinois State Medical Society has actively worked on liability tort reform legislation, limiting attorneys' contingency fees, eliminating a special damages provision, promoting physicians' countersuits for malicious prosecution, as well as calling for the establishment of pretrial screening panels and standards for expert witnesses.[1] As early as 1975, the California Medical Association, working with broad-based coalitions (ie, combining with other loosely related groups seeking tort liability reform in general), had led the nation in compre-

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hensive liability tort reform.[1]

However, these new laws, helpful as they might be, still do not give a physician full rights to a day in court. Malpractice lawsuits will still be brought and physicians will still be "put to the test."

Perhaps it is time that physicians take their own advice to heal themselves. There may be some legal penicillin available to the medical profession under the present case law that could be used advantageously. Physicians may have available a claim under the doctrine known as the prima facie tort rule.[18] The general rule of law holds that for every wrong, there should be a redress or remedy. What are the physician's redresses for malpractice claims that are wrongfully instituted? The judicial realities demand a reevaluation of the tort law available to the physician, the pitfalls and inadequacies of the traditional tort law, and the feasibilities of any new alternative approaches such as, for example, the prima facie tort rule. Although the tort law presented here is basically that of New York State, it tends to be representative of that of most other states and jurisdictions.

Before one can discuss the prima facie tort rule, one must review the usual defenses available to physicians, such as counter-suits for malicious prosecution, abuse of process, and defamation. In addition, one needs to consider whom physicians should sue. Obviously, they can sue the party who originally sued them, ie, the patient-plaintiff in the original malpractice suit. They could even counterclaim and combine both suits. What about third parties who helped institute the original action, eg, the patient-plaintiff's attorneys? These are interesting questions worth exploring. Medical malpractice defenses and responses deserve a second look and consequently should be reexamined and evaluated with an eye toward their feasibility, practicality, and legality.

MALICIOUS PROSECUTION

Malicious prosecution is the institution of judicial proceedings maliciously, spitefully, and without probable cause.[19(pp28-31)] The mere giving of false information, even maliciously and without probable cause, to investigating officials, does not by itself constitute the institution of judicial proceedings.[20]

Elements. Four elements are necessary to make out a case of malicious prosecution. First, the previous malpractice case must have ended in the physician's favor. It need not have been disposed of on the merits, so a plaintiff's failure to appear in court, resulting in a dismissal, could be sufficient. In *Fay v O'Neil*,[21] even a co-plaintiff's failure to appear was considered sufficient to satisfy this element. However, a settlement agreement before any court action is not considered a favorable termination, and a cause of action for malicious prosecution will fail.[22,23]

Second, there must be a lack of probable cause for instituting the former proceeding; that is, the plaintiff lacked a reasonable, good-faith basis for bringing the malpractice action.[24-26]

Third, there must be malice in fact. This can be inferred by a jury[27] and need not be proved by direct evidence, since "want of probable cause of malice is seldom established by direct evidence of an ulterior motive."[28]

Finally, the physician must have suffered damages in the prior civil malpractice action. The damages may relate to his reputation,[29] his person,[30] or his property.[31]

A determination against the physician in the former proceeding is a complete defense barring any present action for malicious prosecution. However, where the plaintiff in the prior malpractice suit lost his case against the physician, the fact that he was advised by his lawyer to sue the physician may mitigate an award of punitive damages (damages over and above actual damages intended as a penalty), but not compensatory damages.[32]

ABUSE OF PROCESS

Can the previously sued physician bring an action against the unsuccessful malpractice plaintiff for abuse of process? Abuse of process arises when the legal process is used for purposes for which it was not intended after the process is properly issued.[19(p32)] For example, if William Roe obtains a warrant for John Doe's arrest in order to force Doe's father to withdraw a claim he has against Roe's father, that is an abuse of process.[33] Likewise, the service of a subpoena on a witness to compel him to settle a claim in order to avoid the inconvenience and expense of going to court is also an abuse of process.[35]

Elements. The elements of a cause of action for abuse of process consist of malice in fact, want of probable cause, and legal damages. A termination favorable to the plaintiff, or any termination, is not necessary.[35] Unlike the tort of malicious prosecution, which is based on improper institution of litigation, the tort of abuse of process is based on the improper use of an incidental process. Since circumstances under which abuse of process can arise are rare in the ordinary scheme of things, this tort would not serve as an average "defense vehicle" to be placed in the physician's legal-medical bag. Lawsuits for abuse of process can be brought in New York State, but the chances of success are slim and they usually are dismissed within a short time on the grounds that they fail to state a cause of action, ie, there is no case.

DEFAMATION

Could an aggrieved physician sue the unsuccessful malpractice plaintiff for defamation? The gist of this tort is damage to the reputation of a living person who has a right to his good name.[19(pp68-81)] Defamation may be written (libel) or oral (slander). False statements that injure a person in his business or profession are defamatory. Any statement which on its face subjects a party to ridicule, scorn, etc, is defamatory per se. However, when a party must produce extrinsic evidence to show how he was harmed, then the defamation is not per se.[36]

Elements. The elements of the tort of defamation are publication (ie, communication to a third party) of a defamatory statement concerning the plaintiff, and malice. Intent to defame is not an element of this tort. Malice can be legal malice,[37] ie, implied from the publication itself, or actual malice, ie, knowledge of the falsity of the publication or a reckless disregard for the truth.[38] Proof of actual malice is required when the plaintiff sues for punitive damages.

There are five principal defenses to an action of defamation: truth,[39] privilege, fair comment, full and fair report, and the defense of reply. The fact that a statement is true, even if defamatory, is always an absolute defense to an action for defamation. Formerly, the burden was on defendants to prove the truth of their defamatory statements. Now, however, the plaintiff may have the burden of proving that the statement is false, a far more difficult task.

A defamatory communication may also be privileged. A judge in a judicial proceeding has an absolute privilege, but as to parties in a judicial proceeding there is only an absolute privilege where the subject matter of the defamatory statement is relevant to the charges.[40] There is a qualified privilege for statements made in the course of carrying out a public duty, such as reporting a crime to the police,[41] and certain private communications are qualifiedly privileged, for example, attorney-client, doctor-patient, husband-wife, priest-confessor, etc.[42]

Fair comment[43] refers to statements on matters of public interest such as the qualifications of a candidate for public office. However, the comment must be fair, truly stated, reasonable, and without malice. A full and fair report of a public and official proceeding is privileged.[44]

A defendant may also contend that his statements were made in reply to plaintiff's, which would be a good defense if the response did constitute a full and fair reply.

The jury has considerable flexibility in awarding damages, which may be nominal, compensatory, or punitive.[45] Damage awards can be mitigated in several ways, as where the defendant can show some parts of his statements to be true, gives his source, shows that the plaintiff has already been compensated elsewhere, retracts his statements and makes an honest endeavor to undo the wrong, shows plaintiff's bad reputation in general, or demonstrates that all his defenses taken together may constitute a fair comment.[46]

Between what may be "privileged," "fair comment," "fair report," etc, as well as "mitigated" damages, the seriously aggrieved physician may have little "equal time, response and justice" available to him. The scales of justice tend to weigh too heavily against the physician in all but the most outrageously blatant cases.

INADEQUACIES OF STANDARD TORTS

One can see how the standard tort causes of action available to physicians who believe they have been wronged by malpractice suits would be difficult to sustain. In addition, they can only be directed against the original plaintiffs in the malpractice suits. Is there another, easier way to fit the original plaintiffs' actions into a tortious category that would enable wronged physicians to sue more easily and have their day in court? What of those who helped the original plaintiffs and without whose help the malpractice suits, with all their problems and anguish, would not and could not have been brought? Is it possible to go against the lawyer or lawyers in the original malpractice suits? If malpractice is based on a doctor's failure to follow the standards of his medical community in the practice of his profession, why is it any less a tortious act when an attorney advises his client to bring an action that the attorney's skill, training, and intelligence, as well as the standards of his legal community, should have told him was not meritorious and would not lie? Indeed, if a lawyer's legal advice to bring a suit against a physician can be used as a defense to show lack of malice in an action for malicious prosecution, for example, [47] or can go towards mitigation of damages,[32] why can't this judicial recognition of the damages caused by a lawyer's advice also serve as a basis for bringing a suit against the lawyer himself on behalf of the wronged physician?

PRIMA FACIE TORT

The prima facie tort could be the answer for physicians who have been unjustly sued. Prima facie tort is a catch-all label for tortious actions that do not fit into other categories. Previously, if a cause of action did not fit into a previously defined "tort," it would be dismissed. The prima facie tort rule can save a cause of action if the necessary criteria are met. The prima facie tort rule is based on the theory that any intentional infliction of temporal (present and specific) damages can give rise to a cause of action that requires a justification as a matter of substantive law, if the defendant is not to escape liability for his tortious acts. In other words, for every wrong there must be a redress or remedy or compensation.[19(pp83-84)] Thus under the prima facie tort rule, if in substance a complaint sets forth a tortious or wrongful act, the defendant must reply. This rule was set forth in *Advance Music Corporation v American Tobacco Co.*[48] In that case, the plaintiff sued on two traditional grounds, fraud and slander of title (an attack on a person's property or trade and not on a person's personal reputation). The plaintiff contended that defendant, on a nationwide radio show, "The Hit Parade," presented ten songs as "the top 10 songs of the land." The court held that there was no fraud since the plaintiff was not defrauded, ie, he was not fooled into believing that his song was not "top 10" caliber. The court also held that there was no slander of title since plaintiff could not show damages. The court, however, held for the plaintiff on the basis that the intentional infliction of temporal damages is itself a cause of action which as a matter of substantive law demands a justification if the defendant is not to escape his wrongdoing and justice is to be served.

The prima facie tort rule is covered by a six-year statute of limitations, whereas the specific torts are usually covered by shorter statutes of limitations. If the facts show that there is a specific tort available, the courts will not allow a claim to be placed under the prima facie tort rule in general,[49] and especially not to take advantage of the longer prima facie tort rule statute of limitations.[50]

In *Widger v Central School District No. 1*,[51] the plaintiff was a teacher who had been suspended by a school board and who brought an action alleging four causes of action: breach of contract, abuse of process, tortious interference with contract rights, and prima facie tort. The lower court had dismissed all the plaintiff's causes of action. However, the appeals court reversed and allowed the cause of action based on the theory of prima facie tort, stating that the allegations of an intentional infliction of temporal or specific damages by the defendants were sufficient to state a cause of action. The addition of the prima facie cause of action avoided an earlier dismissal since the other three causes of action were properly dismissed, and allowed the plaintiff to have his full day in court.

In *Metromedia, Inc v Mandel*,[49] the court stated the rule that when specific, recognizable tortious acts are asserted, the plaintiff's remedy could only be under the appropriate classical tort and not under the prima facie tort rule. The court pointed out that the key to the prima facie tort rule is the infliction of intentional harm resulting in damages, without any excuse or justification, by an act or series of acts that would otherwise be lawful. The prima facie tort rule appears to be a legal fail-safe system for cases that do not fit into the pigeonhole of standard torts and thus would otherwise have slipped through the fragile net of justice. So, as stated previously, where another cause of action is available, then the prima facie tort rule is out. In *Sloan v Clark*,[52] the plaintiffs were limited partners who went into bankruptcy because of their inability to meet their obligations on loans made on the strength of warehouse receipts issued by the defendant. They sued, among other grounds, on the grounds of prima facie tort. The court held that there was no cause of action in prima facie tort because, while the limited partnership sought to sue the defendant under the prima facie tort rule for causing its bankruptcy, the same defendant was already being sued in another case by the trustee in bankruptcy on behalf of the limited partnership. Here, the prima facie tort rule was not the only available means for serving justice.

JUSTICE SERVED

The legal creation of the prima facie tort rule serves the full, proper, and more complete ends of justice. If a physician feels that there has been an abuse and misuse of justice and the law, he should have the right to seek redress within the very same judicial system, in the same court, and under the very same legal roof, so to speak, where the physician's original adversary had his day in court. A physician is not a lesser citizen with lesser rights. The courts should be permitted to decide in each case if there has been a temporal infliction of intentional harm resulting in damage to the physician which demands redress for justice to triumph.

The courts should also decide if an attorney who advises a layman to bring an improper action or who carries out such layman's orders is a proper subject against whom redress might, as a matter of substantive law, be sought.

It certainly would be worth the efforts of some aggrieved physician and some properly aggressive attorney to bring an action based on prima facie tort against a prior malpractice plaintiff where the facts, circumstances, and justice demand it. A successful outcome might make a potential plaintiff and his attorney think twice before bringing an action. The original patient-plaintiff has no "malpractice countersuit insurance," and his attorney may not be able to have the action summarily dismissed before the trial. The defendant-doctors' all-too-familiar feeling of having a "case hanging over their heads," along with its costly intrusions, both time and money, could now be shared with their original plaintiffs and their third-party helpers, a nice democratic equalism. The numbers of frivolous lawsuits will diminish while those truly meritorious cases can continue to be brought in the interest of justice. Perhaps then, with the help of the prima facie tort rule, the physician will have succeeded in legally healing himself.

In summary, the prima facie tort rule may be available when an aggrieved party can show temporal infliction of deliberate or intentional harm, resulting in injury or damage for which redress is necessary as an act of simple justice and for which no other tort remedies are available.

What constitutes intentional harm? What constitutes redress? What are the elements of the prima facie tort? How far will the courts go in their evaluation of prima facie tort cases? Future case law—or even legislative action, if warranted—will establish the ground rules and determine the extent to which the tort will be developed. The legal door has been opened, even if it is only slightly ajar at present. This door must be opened further so that the prima facie tort can develop to its full potential to better serve the ends of justice and equity to the advantage of both professions. The law would witness fewer frivolous lawsuits, and medicine would see its practitioners have their day in court.

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A history of the *New York Medical Journal*

Pascal James Imperato, MD

The *New York Medical Journal*, which was never affiliated with the Medical Society of the State of New York (MSSNY), had its origins in the waning days of the American Civil War. In an editorial published in the first issue in April 1865, an unnamed editorialist laid out the reasons for the establishment of the *Journal*.[1] Paramount among these was the impact of the war itself on medical journalism.

> The changes incident to a state of war have led to the discontinuance of some of the medical periodicals of our country, have diminished the importation of foreign books and journals, and have lessened the number of reprints of professional matter.
>
> From the same causes there has been an immense increase in the activity of the Medical profession; hospitals have become vastly more numerous, and are crowded with inmates; medical schools have larger classes in consequence of the greater demand for surgeons for the military and naval services; young men are called earlier to assume responsible duties; and experience, in novel forms, is accumulating, and the want of current medical literature, especially for that bearing upon the topics of the day, has augmented in a similar degree.
>
> Our professional brethren in military and naval hospitals, in the field, and on our national vessels are isolated from the centres of medical education, and cut off from their usual sources of professional reading; they also want means of communication with the profession in civil life, through which their rapidly accumulating experience may find expression.
>
> To meet these wants, in some degree, by judicious digests of American and foreign literature and the publication of good original matter, and to serve as an organ of expression for medical men—in the public service as well as in civil life—and as a channel of communication on subjects of professional interests, is the object we propose for the *New York Medical Journal*.[1]

The editorial went on to say that the *Journal* was "well supported, both financially and by adequate literary ability" and that it had a "well organized editorial corps that would ensure a regular supply of material."[1] It was also stated that the names of collaborators would serve "as a guarantee for the tone and character of the *Journal*."[1]

Origins

The *New York Medical Journal* was founded by a group of physicians headed by William Alexander Hammond, MD, who had arrived in New York City in 1864, somewhat of a piece of wrecked timber.[2] In 1864 he had been court martialed and dismissed from the US Army where he had served as surgeon general. He was an enterprising man, tall, burly, and full of enormous energy and new ideas. He was " . . . optimistic to the verge of the visionary . . . ."[2] However, his various schemes often had " . . . some defect that a more prudent man would have guarded against . . . ."[2] Hammond's " . . . methods were not always in conformity with the unwritten code of professional ethics."[3] In the view of his contemporaries he was " . . . a charming companion, faulty, but . . . guileless . . . ."[2]

Hammond, who was born in 1828 in Annapolis, Maryland, received his medical degree from the Medical Department of the University of the City of New York. He entered the medical corps of the US Army in 1849, but resigned in 1860 to become professor of anatomy and physiology at the University of Maryland. At the outbreak of the Civil War, he reenlisted in the army and in 1862 was commissioned surgeon general at the age of 34 years on the recommendation of the members of the US Sanitary Commission. He was young, brash, somewhat pompous, and arrogant, but a man of enormous vision and energy who breathed new and needed life into the army's medical department. His appointment rankled senior officers and his direct manner brought him into conflict with

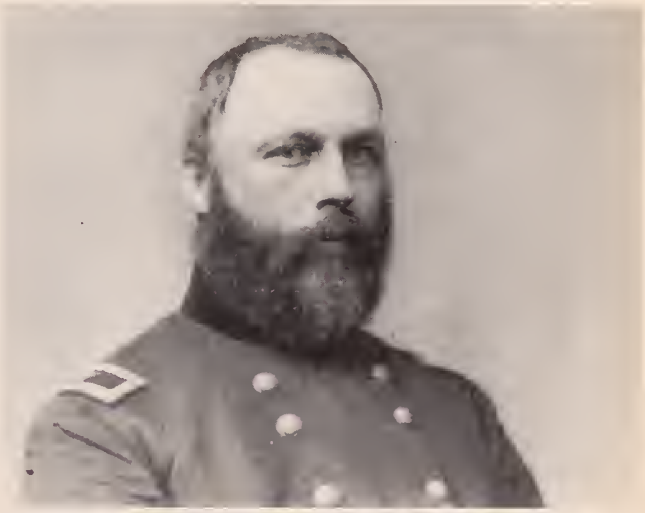

FIGURE 1. William Alexander Hammond, MD, principal founder of the *New York Medical Journal* (courtesy, Public Affairs Office, US Army Surgeon General's Office).

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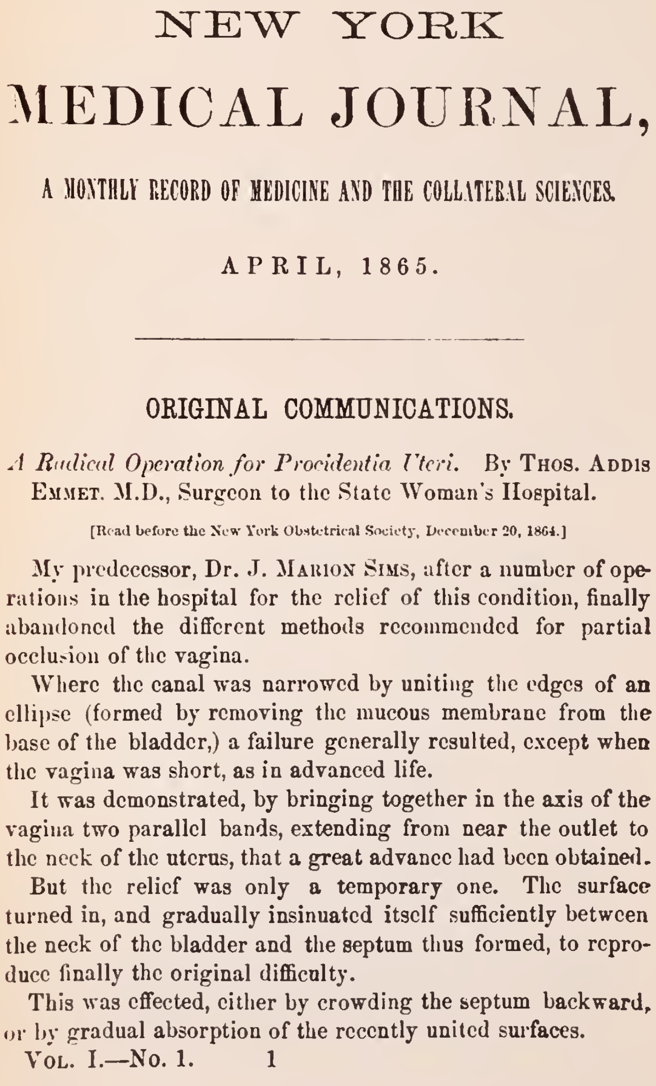
many, including Secretary of War Edward M. Stanton, his superior. Stanton was abrasive, irritable, hasty, headstrong, and at times unjust. Hammond and he clashed almost immediately. Hammond also swelled the ranks of his opponents by issuing a famous circular no. 6 removing calomel and tartar emetic from the army drug list. This naturally infuriated many physicians who were accustomed to using these drugs and incensed manufacturers and distributors.

Stanton resolved to rid himself of Hammond and resorted to well established bureaucratic maneuvers to induce him to resign. In July 1863 he appointed a commission headed by an old Hammond enemy to examine the affairs of the medical department, ordered Hammond into virtual exile to New Orleans on a vague inspection trip, and then shunted him to Nashville where he annulled his powers as surgeon general.[4] Stanton then appointed Colonel Joseph K. Barnes as acting surgeon general. Hammond was being vilified and would soon be victimized. Firmly believing himself innocent of any wrongdoing he requested either reinstatement or a court martial. At his own insistence he eventually got the latter.[4]



NEW YORK

MEDICAL JOURNAL,

A MONTHLY RECORD OF MEDICINE AND THE COLLATERAL SCIENCES.

APRIL, 1865.

ORIGINAL COMMUNICATIONS.

A Radical Operation for Procidentia Uteri. By THOS. ADDIS EMMET, M.D., Surgeon to the State Woman's Hospital.

[Read before the New York Obstetrical Society, December 20, 1864.]

My predecessor, Dr. J. MARION SIMS, after a number of operations in the hospital for the relief of this condition, finally abandoned the different methods recommended for partial occlusion of the vagina.

Where the canal was narrowed by uniting the edges of an ellipse (formed by removing the mucous membrane from the base of the bladder,) a failure generally resulted, except when the vagina was short, as in advanced life.

It was demonstrated, by bringing together in the axis of the vagina two parallel bands, extending from near the outlet to the neck of the uterus, that a great advance had been obtained.

But the relief was only a temporary one. The surface turned in, and gradually insinuated itself sufficiently between the neck of the bladder and the septum thus formed, to reproduce finally the original difficulty.

This was effected, either by crowding the septum backward, or by gradual absorption of the recently united surfaces.

VOL. I.—No. 1. 1

FIGURE 2. First page of the first issue of the *New York Medical Journal*, volume 1, April 1865.

The charges against Hammond were rather flimsy but Stanton probably believed him corrupt since the war office was, as it had been before Hammond's time, full of grafters.[4] Hammond was pleased to be court martialed, believing that he would be fully vindicated. Although there were several charges, the case hinged on the irregular purchase of medical supplies. Hammond defended his purchases on the grounds that his decisions were in the best interests of the ill and injured. No evidence was produced that Hammond profited from the purchasing arrangements. However, he had clearly left himself open to suspicion because of his methods of operation.

None could deny that he had accomplished in 15 months as surgeon general what predecessors had not in years. The star witness was a vindictive enemy of Hammond's. Yet Hammond was confident he would be vindicated. With the secretary of war and the star witness both his enemies, it was no surprise that he was found guilty of complicity in fraud and dismissed from the army.[4] As Duncan says, writing 56 years later, "when Hammond was ejected from his office in 1863, progress stopped."[4] Mrs Hammond's appeal to President Abraham Lincoln was politely turned down, Lincoln deferring to Stanton as far as the case was concerned.[4] Penniless, disgraced, and in debt (he had borrowed $6,000 to defend himself), Hammond went to New York City where he began a private practice, specializing in neurology. A visionary and somewhat of a risk-taker, he helped found the *New York Medical Journal* in 1865. Had he not been dismissed from the army, it is likely that the *Journal* would never have materialized. Hammond established the *Journal* at a time when it was common for individual physicians to launch medical journals on their own. The sole property of these physicians, these journals were printed by printers who at that time were frequently publishers as well. The line between printer and publisher was still an ill-defined one. Often shares in these journals were sold to other physicians or venture capitalists and in a number of instances publishers eventually acquired the publications.

EARLY HISTORY

The *New York Medical Journal* was first published in New York City by Miller and Mathews and was subtitled, *A Monthly Record of Medicine and the Collateral Sciences*.[2] It was published in a $5\frac{1}{2}'' \times 9''$ format which approximated that of many medical books of the time. This size was continued until 1881. At the time it was launched, the *Journal* was primarily intended as a publication for physicians in New York City. However, later on the publishers sought to appeal to a larger national audience. During its lifetime, the *Journal* was put out by several publishers. The acquisition of other existing medical journals resulted in several modifications of the *Journal's* name and subtitle.

Two volumes were initially published per year, each with six numbers, extending from April to September and from October to March. Volumes 2 and 3 (1866) were published by Balliere Brothers and volumes 4 and 5 (1867) by A. Simpson and Company. Moorhead, Simpson and Bond published volume 6 (1868). However, difficulties arose with this publishing house as explained under the heading "varia" in the September 1868 issue of the *Journal*.[5]

> The Medical Gazette, and American Journal of Obstetrics.—In consequence of changes in the house of Moorhead, Bond & Co., these Journals have been, like ourselves, for a time afloat, but have finally drifted into a harbor of safety, and we are happy to announce that their publication will be continued. The Editors of each have become the proprietors of these Journals. The Obstetrical Journal will be issued for Dr. Dawson by the House of W.A. Townsend & Adams, favorably known as publishers of the American reprint of Braithwaite's Retrospect, and the Gazette is published by Mr. John Sarell, of this city.
>
> Issues of both these Journals have made their appearance this week, doubtless much to the relief of the subscribers as well as the satisfaction of the proprietors.[5]

In the summer of 1868, Hammond and his partners sold the *Journal* to D. Appleton & Company along with the *Quarterly Journal of Psychological Medicine and Medical Jurisprudence* which he had also founded in 1865.[6] He served as co-editor of the latter journal until 1872 and later served from 1876 to 1883 as co-editor of the *Journal of Nervous and Mental Diseases*. D. Appleton & Company published the *Journal* until July 12, 1900, when it was purchased by the A. R. Elliott Publishing Company of New York City.

The *Journal* greatly increased its circulation in 1903 when it purchased the *Philadelphia Medical Journal* from a group of Philadelphia physicians.[7,8] The *Journal* then temporarily changed its name to *New York Medical Journal and Philadelphia Medical Journal, Consolidated*. In 1905 the *Medical News*, which had been published by Lea Brothers & Company since 1843, was purchased. The *Journal* thereafter appeared under the title, *New York Medical Journal, Incorporating the Philadelphia Medical Journal and the Medical News*.[8] It was subtitled: *A Semimonthly Review of Medicine and Surgery, Established 1843*. The 1843 date actually referred to the starting date of the *Medical News* which was folded into the *Journal* in 1905.[8] The acquisition by the A. R. Elliott Publishing Company of the two Philadelphia-based medical journals and their circulations made the *Journal* one of the most widely circulated independent medical journals in the United States.

LATER HISTORY

In April 1922 another medical journal, the *Medical Record*, was acquired from William Wood and Company of New York, then a leading medical book publisher in the country.[8] The *Medical Record* had been published for 56 years and was for many years a prominent journal. After this acquisition, the *Journal* was known as the *New York Medical Journal and Medical Record* and carried the subtitle, *Philadelphia Medical Journal and the Medical News*. Although the *Journal* was published in New York City, the table of contents page carried Philadelphia, New York, and Chicago as the places of publication, probably in an attempt to give it national character and appeal.

In 1924, the *Journal* underwent a major change in name from *New York Medical Journal and Record* to *Medical Journal and Record*. The subtitle was *A National Review of Medicine and Surgery*.[9] The name *New York* was dropped, as an unsigned editorial explained, because ". . . we have outgrown any purely local functions."[9] The publishers, the A. R. Elliott Publishing Company of New York, were clearly intent on putting out a general medical journal with broader national and international appeal.[9] It was not merely a matter of name change but also one of broadening the scope of the *Journal's* editorial content and increasing the size of the *Journal's* circulation.[9] A supplement, averaging 16 pages, was added to each semimonthly issue. Printed on yellow paper to distinguish them from the rest of the editorial content, these supplements were thematic in nature, covering such subjects as neuropsychiatry, genitourinary medicine, pediatrics, etc. Aiming to be progressive and in the vanguard of medical progress, the *Journal's* content now included articles reporting on new diagnostic and therapeutic advances.

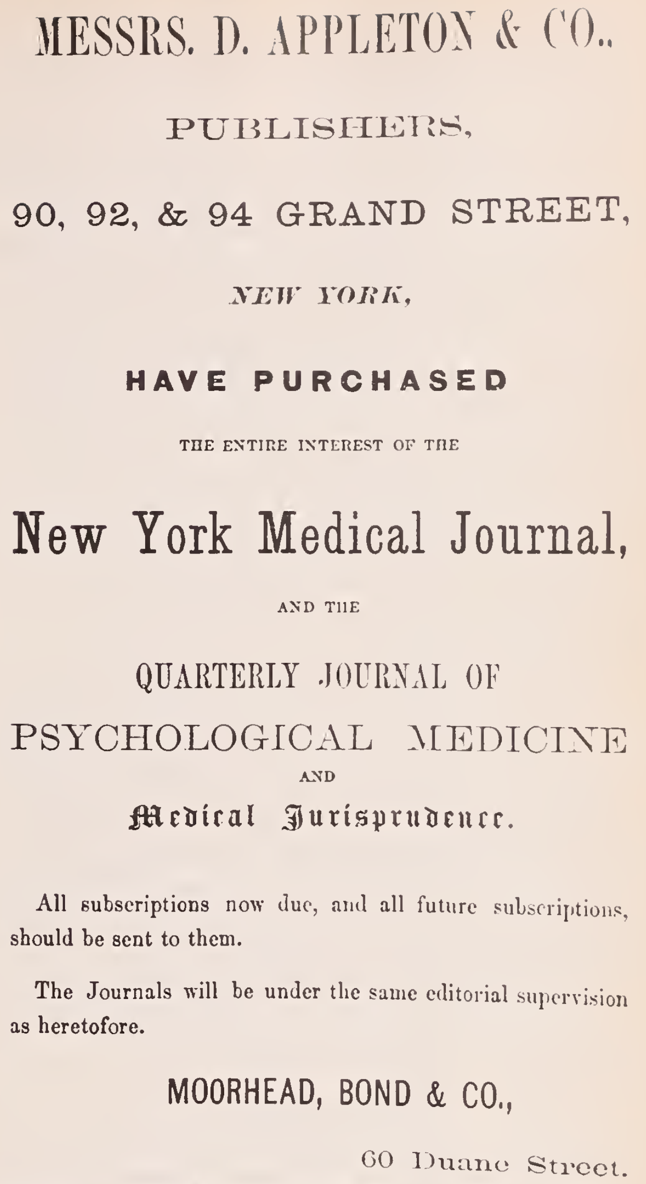


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FIGURE 3. Notice of acquisition of the *New York Medical Journal* by D. Appleton & Company.[6]

Immediately prior to the changes of 1924, the categories of articles in the *Journal* fell under the general headings of original communications, editorial articles, book reviews, practical therapeutics, proceedings of societies, and abstracts from the current literature. In 1924, several categories were added in addition to the supplements. These included history, news items, obituaries, foreign correspondence, medicolegal notes, and medicoliterary notes.

Volume 118 (July–December 1923), which appeared under the title *New York Medical Journal and Record*, had a total of 788 pages of editorial content exclusive of stacked advertising that regularly appeared at the beginning and end of each number. Volume 119 (January–June 1924), which appeared under the new name, had 632 pages of editorial content exclusive of advertising that continued to be stacked and 160 pages of supplements which were separately numbered. The total number of pages per volume in 1924 averaged 792, close to the 788 for 1923. This indicates that the publishers were successful in keeping printing costs roughly the same while repackaging the editorial content. The reduction in regular content primarily affected the original communications section. Printed page reductions here and elsewhere enabled the publishers to put out an appealing 16-page supplement with every semimonthly issue without increasing the total page count.

The *Medical Journal and Record* like its predecessor the *New York Medical Journal* was published in an 8½″ × 11″ format. This format was first adopted in 1882 when the smaller 5½″ × 9″ size, first used in 1865, was changed. Although the *Medical Journal and Record*, as the *Journal* was now known, embodied several medical journals, in a sense its publisher saw it as a continuation of the *New York Medical Journal* and thus continued to use that publication's volume numbers. It carried the subtitle *Incorporating the Philadelphia Medical Journal and the Medical News* in the first transition number of January 2, 1924, but thereafter dropped it.[10] Yet, drawing on its descent in part from the *Medical News* it continued to give 1843 as the date it was established.

EDITORS

William Alexander Hammond, MD. During the first three years of the *Journal's* existence, no editor was named on the masthead. However, an editorial signed "WAH" indicates that Hammond had a major role in editing the publication. A possible reason for not mentioning Hammond's editorship may have been concerns about the adverse affect of his much publicized army dismissal on circulation. Hammond was a prolific writer and prior to founding the *Journal* had authored numerous medical books and articles. He had also served as the co-editor of the *Maryland and Virginia Medical Journal* in 1861.[11]

Hammond's name first appeared on the masthead as a co-editor in the October 1868 (volume 6) issue of the *Journal*. His co-editor was Edward Swift Dunster, MD. Hammond and Dunster continued as co-editors until 1869 (volume 8). In the March 1869 issue (volume 8) a notice appeared under "Miscellaneous and Scientific Notes" stating:

> The connection of the undersigned with the *New York Medical Journal* has been but nominal for the last two years. With this number it ceases altogether.
>
> The *Journal* will continue under the editorial supervision of Dr. E. S. Dunster.
>
> William A. Hammond[12]

Hammond and his partners had sold the *Journal* the previous summer to D. Appleton & Company and a prominent notice to that effect had appeared in the *Journal*. It is of interest that the publishers requested a public statement of Hammond's disassociation with the *Journal*. The reasons for this are unclear. Hammond went on to have a distinguished career in medicine, as noted in the *New York Medical Journal* when he died in 1900.

> He founded two medical journals, the *Quarterly Journal of Psychological Medicine and Medical Jurisprudence* and the *New York Medical Journal*. The contents of the quarterly consisted largely of his own writing, and it was of a character to gain for him a great reputation. Before long he was made professor of diseases of the mind and nervous system in Bellevue Hospital Medical College and subsequently in the Medical Department of the University of the City of New York. A few years later he took part in founding the New York State Hospital for Diseases of the Nervous System, and afterward became a member of the faculty of the New York Post-graduate Medical School. In the course of his career in New York he wrote a treatise on diseases of the nervous system and one on insanity. Both books were received with favor by the profession and added largely to Dr. Hammond's reputation. Perhaps his most important service to neurology was his recognition and admirable description of a morbid condition known by the name he gave it, athetosis. He did not long keep up his connection with medical journals, medical schools, and hospitals, and after a time he was no longer a frequent contributor to medical literature. At length he took to amusing himself with writing fiction. The first of his stories, entitled *Lal*, met with considerable commendation; the four or five others were not so well received. A few years ago Dr. Hammond conceived a great liking for the city of Washington. He bought property in that city, built a handsome residence and a private hospital [Hammond Sanitarium], and changed the scene of his career. After he left New York he frequently came back for brief visits, and on such occasions he was almost sure to put in an appearance at the office of this journal, where, no matter what the perplexities of the "make-up," he was always welcomed.
>
> Since Dr. Hammond died the remark has more than once been made that he came very near being a great man. He committed errors, errors that a man of narrower intellect but greater worldly wisdom would be tolerably sure to avoid, and some of them led to cruel aspersions of his moral character. It has always seemed to us that he lacked inflexibility of purpose; scheme after scheme, all laudable and promising (as he pictured them), would arise in his mind, for he was optimistic almost to the verge of the visionary, but some defect that a more prudent man would have guarded against too often barred their fruition. His enthusiasm in these schemes was so manifestly the dominator of his thoughts that it was useless for his friends to argue against it. At the same time Dr. Hammond was an exceptionally fair man in argument, always ready to concede gracefully any point scored by an opponent. He was a charming companion, faulty, but, it is our conviction, guileless.[2]

Hammond bore no malice to those who did him so much harm in ousting him from the army. When President Abraham Lincoln died in 1865, Hammond ran an editorial in the *Journal* praising him, signing it WAH.[13] In the middle of this editorial he wrote the following, obviously alluding to the injustice done to himself:

> That he was honest in his opinions, sincere in his convictions, merciful in his impulses, acting in all things for the best, according to the light he had, no one can doubt who knew him and judged him fairly, as man should judge his fellow mortal. If, during his administration, acts were done in his name not in themselves right; if others were omitted which should have been per-

> formed; if wrongs were inflicted upon individuals for which full amendment can never be made, the responsibility cannot with justice be fastened upon him. It rests with those who were appointed to advise and assist him in the multitudinous duties of his great office, and in whom he trusted with the child-like confidence which formed so prominent a feature of his open and ingenuous disposition.[13]

Hammond set about to clear his name and did so with dignity and without maligning those who had been his adversaries. He stated the facts as he saw them and refrained from either asking for financial compensation or analyzing the motives of others. In 1864 he published his own account of his dismissal,[14] carrying the issue into the first volume of the *Journal* in the form of an editorial.

This unsigned editorial reported on the retribution received by " . . . one of the leaders of the conspiracy which resulted in the removal of Dr. William A. Hammond from the office of Surgeon General . . . ."[15] The editorial reported in detail on the court martial and sentence of Silas H. Swetland, whom Hammond had refused to appoint as Inspector of Liquors for the Medical Department when he was surgeon general.[15] Swetland, it was alleged, then became pivotal in trumping up charges against Hammond. Thus Swetland's conviction on several counts of fraud by the United States Army cast aspersions on his integrity and credibility.

In 1878 Hammond took his case before Congress. Both the Senate and the House heard the plea, the Senate Committee on Military Affairs taking the lead in hearing the case. The Senate Committee on Military Affairs was impressed by Hammond's arguments. He did not deny all blame or impugn the motives of those whom he thought had conspired to persecute him and admitted that he had not avoided certain antagonisms.[4]

In its report the committee said:

> It is reasonable therefore to infer that men of the positive natures possessed alike by Secretary Stanton and Dr. Hammond will decline to yield, or stand by for each other to pass, when they crossed and crowded upon what they conceived to be the path of mutual duty. When they collided it was the gage of battle hurled by both—a war of the Titans, a struggle for mastery. One or the other must have fallen in a conflict of such a nature; for there was no middle ground of accommodation between them. Secretary Stanton . . . no doubt became impressed that that displacement of Surgeon General Hammond would conduce to the benefit of the public service, and, possessing the greater power, accomplished, by means of indirection, the desired result.
>
> The situation in 1862 was one filled with difficulties; there were, of necessity, antagonisms and jealousies; chaos reigned supreme and the fate of a single person weighed not a featherweight in the balance. Men of elevated rank and station were cast down to give place to others, in some cases the experiment utterly failing. Into the whirlpool of events, Dr. Hammond was drawn and carried down.[4]

The Senate Committee also concluded that all of the charges against Hammond except one (falsehood) were either disproved by the defense, abandoned by the prosecution, or eliminated by the findings of the court. The charge of falsehood was in retrospect seen by the Senate Committee as trifling if not frivolous, and insufficient to arraign, try, and convict him.[4]

Both houses passed bills clearing Hammond's name after which President Rutherford B. Hayes annulled the proceedings and sentence of the court. Hammond's name was placed on the retired list of the army on August 17, 1879.[4] He had to some degree been vindicated. Writers such as Duncan believe him to have been the victim of a gross miscarriage of justice spawned by Secretary Stanton's dislike of him and clearly the US Congress saw the matter in that light.

Hammond's legacies to medicine were many. He founded the Army Medical Museum (later the Armed Forces Institute of Pathology) when he was surgeon general at the age of 34 years, founded two medical journals at the age of 37 years, was a co-founder of a hospital, and authored numerous authoritative medical articles and books in addition to being a busy clinical practitioner.

Edward Swift Dunster, MD. Edward S. Dunster, MD, co-edited the *Journal* with William A. Hammond, MD, at least as early as 1868 and probably before this. He and Hammond were listed as co-editors from 1868 through the spring of 1869 when Hammond left. It is probable, given Hammond's statement of only nominal involvement in 1867 and 1868, that Dunster carried most editorial responsibility during those years.[12] Dunster continued as the sole editor from April 1869 through June 1871. Volumes 10 through 12 carried only five numbers, perhaps due to printing transition problems after the takeover by D. Appleton & Company. In January 1871, the *Journal* reverted to six numbers per volume which spanned January–June and July–December.

Dunster, who was born in Maine, received his undergraduate education at Harvard and his medical degree in New York City. During the Civil War he served as a medical officer in the Army of the Potomac and then entered private practice in New York City. He taught at Dartmouth, the University of Vermont, and at the Long Island College Hospital. In 1873 he accepted the position of professor and chairman of obstetrics and diseases of women and children at the University of Michigan in Ann Arbor. He was 39 years old at the time. He died of pneumonia on May 3, 1888, in Ann Arbor at the age of 53 years. His obituary notice says of him that:

> In every position that he occupied he made himself felt as a man of the utmost force and worth, and he was widely recognized and admired for his insight into the requirements of medical education and the principles of ethics and for his skill and energy in expounding his views. He was also highly esteemed for his personal qualities.[16]

William T. Lusk, MD. William T. Lusk, MD, succeeded Dunster and served as editor from 1871 to 1873, in the latter year as co-editor with James B. Hunter.[17] As was typical of his time, Lusk studied medicine in Berlin and Heidelberg before graduating from Bellevue Hospital Medical College in 1864. A specialist in obstetrics and gynecology, he was also professor of physiology at Long Island College Hospital and professor of obstetrics and diseases of women at Bellevue Hospital Medical College in the 1870s. In 1889 he served as president of the New York State Medical Association (see below).[18] He died in New York City on June 12, 1897, at the age of 59.[18]

James Bradbridge Hunter, MD. Lusk was succeeded by James Bradbridge Hunter, MD, who had served as the co-editor in 1873. Hunter, an 1866 graduate of the Col-

FIGURE 4. James B. Hunter, MD, editor of the *New York Medical Journal*, 1874–1879.[19(p712)]

lege of Physicians in New York, served as editor of the *Journal* from 1874 to 1879. Like Lusk, Hunter was an eminent gynecologist. He was an attending surgeon at the Woman's Hospital, a founder of the New York Cancer Hospital, and professor and chairman of gynecology at the New York Polyclinic. He served for a time as president of the latter institution. Hunter devoted a great deal of time and effort to the *New York Medical Journal* and under his editorship it improved. He built upon the excellence started by his predecessor Lusk. Like Lusk he eventually gave up the editorship of the *Journal* because of the growing demands of his private practice.

Hunter died at 52 years of age, on February 10, 1889, ten years after he had left the editorship of the *Journal*. Lengthy obituaries about him appeared in the *American Journal of Obstetrics, Gynecology and Diseases of Women and Children*[19] and in the *Medical Record*.[20] The New York Obstetrical Society and the faculty of the New York Polyclinic both passed resolutions in his memory and honor.[21] In these accounts, Hunter was praised as a superb diagnostician and surgeon. However, he was a reserved man. One writer summed up these traits by saying:

> By nature reserved, reticent and undemonstrative, he was not one to have many intimates; but by those who were brought into close contact with him he was much beloved . . . In times of trial the tenderness which he would display was a marvel to those who had not previously seen this characteristic tested, and there were no limits to the gentle, loving, sympathy which the sufferings of others would call forth.[20]

Another writer said "The fragrance of his good deeds will outlast the reputation of the distinguished surgeon."[19]

Frank Pierce Foster, MD. Hunter was followed by Frank Pierce Foster, MD, who like him was a graduate of the College of Physicians and Surgeons (1862) and a gynecologist. Foster also had an interest in dermatology. His tenure was the longest of any editor of the *New York Medical Journal*. He served for 31 years, from 1880 to 1911. Foster was, in the words of the *Journal of the American Medical Association*, "dean of the medical journalistic profession . . . ."[22] Under his skill and energy, the editorial content of the *Journal* rapidly improved and the *Journal* moved from being a monthly publication to a weekly one.[22] It was under Foster that the *Journal* became one of the leading medical publications in the country. During the early years of Foster's editorship, the *Journal* was known as the *New York Medical Journal and Gynecologic Review*, a title perhaps reflecting the interests which both he and his predecessor shared.

Writing a quarter of a century after his father's death, Hugh Molleson Foster said:

> In 1880 he became editor of the *New York Medical Journal* . . . . When he assumed the editorship the journal was a rather anaemic monthly, not of the highest rank and with a limited circulation. When he died it was one of the most important medical weeklies of the world—certainly there is none in this country of higher order—and its circulation was second only to that of *The Journal of the American Medical Association*.[23]

In a letter to his son in 1901, Foster said that when James Bradbridge Hunter, MD, resigned the editorship, the Appletons left the choice of a successor to Dr Fordyce Barker, who chose him. Foster was eminently suited for the position. A linguist who knew several languages including Latin, Greek, French, German, Italian, and Spanish, he was a man of letters who had already published numerous medical papers and had translated a medical text from French, *The Mother's Work with Sick Children*, published by G. P. Putnam & Sons. He had also edited a new edition of *Adler's German Dictionary*.[23]

Foster, who was born in 1841 in Concord, New Hampshire, was a grandnephew of Daniel Webster on his mother's side. He had a deep love of music and books which he maintained throughout his life.[23] After receiving his medical degree from the College of Physicians and Surgeons, he served at the New York Hospital, sailed as a ship's surgeon around Cape Horn, served in the National Guard of New York State during the later years of the Civil War, and then entered private practice in New York City. During the 1860s he conducted research on calf lymph smallpox vaccine and presented his findings at the New York Academy of Medicine in November 1870. Prior to that date and for several years afterwards he produced vaccine in a laboratory in Connecticut. According to his son, his net monthly profits from this business were $2,500, an enormous sum in those times. However, when vaccination became compulsory, public health agencies began to produce vaccine and distribute it free to physicians. As a result Foster's vaccine business " . . . was destroyed."[23] Foster courageously fostered vaccination in New York City at a time when there was strong opposition to it from many physicians.

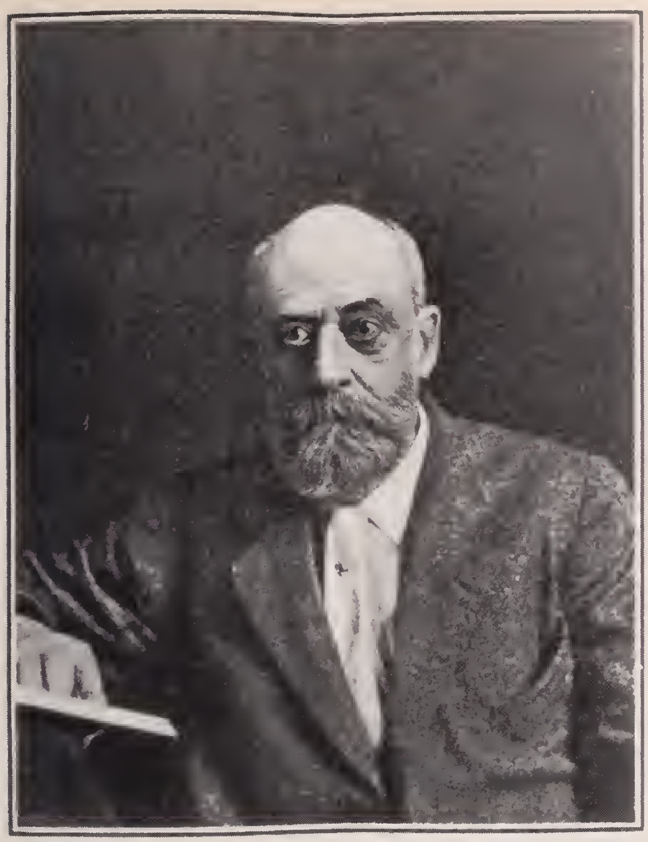


FIGURE 5. Frank Pierce Foster, MD, who served as editor of the *New York Medical Journal*, 1880–1911 (*NY Med J* 1911; 94:391).

During his early years as the *Journal's* editor, Foster maintained a large private practice specializing in gynecology and dermatology and served as librarian of the New York Hospital Library. Between 1888 and 1894 he published his four-volume *Foster's Illustrated Encyclopedic Medical Dictionary*. This was a classic reference of its time in which " . . . terms were given in Latin, Greek, Italian, Spanish, German and English."[23] In addition to all these efforts, he wrote for other medical journals, testified as an expert witness in criminal cases, and helped colleagues with their medical writing.

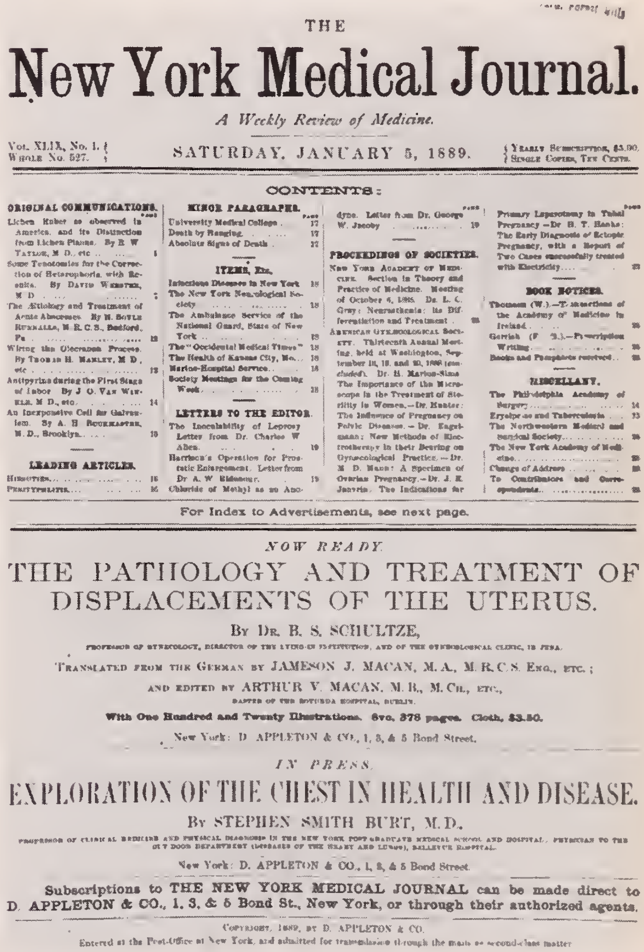
Foster, overworked and overextended, developed appendicitis which went on to rupture and abscess formation. He survived two surgical procedures, but after recovering gave up his private practice except for " . . . a few old and devoted patients."[23] In the 1890s Foster authored his two-volume *The Handbook of Practical Therapeutics*, and an abridged edition of his dictionary. He also served as the editor of the medical departments of Funk and Wagnall's Standard Dictionary and edited the textbooks of the Chatauqua School of Nursing.[23]

Although the *Journal* was thriving, D. Appleton & Company, its publisher, was not. According to Foster's son, the following occurred:

> In the spring of 1900 the firm of D. Appleton & Co., owners and publishers of the *New York Medical Journal* and publishers of all his other writings, failed. He had no warning whatever and his financial condition was by no means strong enough to withstand such a calamity. Saturday morning he went down to the office as usual to get his check and was told that there would be no payments, that the firm had failed. The shock was so great and so sudden that I think he never fully recovered."[23]

The *Journal* was purchased by the A. R. Elliott Publishing Company of New York City and Foster continued as editor until his death in 1911. In 1910, Foster was diagnosed as having throat cancer. After a year of suffering he died at the summer cottage of his son-in-law in Chadwick, New Jersey, on August 13, 1911, at the age of 69 years.[22,23]

Foster's death was a great blow to the *Journal* staff. His son later wrote, "Even stenographers in the office of the *New York Medical Journal* speak of him with a catch in the voice one does not often find in relatives."[23] He was a man who had a prodigious memory, sound judgment, and superb writing skills and who was a meticulous scholar. He was a Renaissance Man of his time who loved music, the arts, literature, and who had as his friends not only physicians but also poets, writers, actors, and clergymen. His own journal printed an effusive eulogy after his death (vol 94:388) and a long obituary (vol 94:391–392). His son has left a detailed portrait of his personal and professional lives which, even allowing for the expected prejudice of a devoted and loving son, portrays him as a truly exceptional man. This same view was put forth by his contemporaries. His son recounts a story that sums up the



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CONTENTS:

ORIGINAL COMMUNICATIONS.
Lichen Ruber as observed in America, and its Distinction from Lichen Planus. By R. W. Taylor, M. D., etc. . . . 1
Some Tenotomies for the Correction of Heterophoria, with Results. By David Webster, M. D. . . . 7
The Ætiology and Treatment of Acute Abscesses. By M. Boyle Ruxhalla, M. R. C. S., Bedford, Pa. . . . 10
Wiring the Olecranon Process. By Thomas H. Manley, M. D., etc. . . . 13
Antipyrine during the First Stage of Labor. By J. O. Van Winkle, M. D., etc. . . . 14
An Inexpensive Cell for Galvanism. By A. H. Buckmaster, M. D., Brooklyn . . . 15

LEADING ARTICLES.
Hirsuties . . . 16
Phenylurethan . . . 16

MINOR PARAGRAPHS.
University Medical College . . . 17
Death by Hanging . . . 17
Absolute Signs of Death . . . 17

ITEMS, Etc.
Infectious Diseases in New York . . . 18
The New York Neurological Society . . . 18
The Ambulance Service of the National Guard, State of New York . . . 18
The "Occidental Medical Times" . . . 18
The Health of Kansas City, Mo. . . . 18
Marine-Hospital Service . . . 18
Society Meetings for the Coming Week . . . 18

LETTERS TO THE EDITOR.
The Inoculability of Leprosy. Letter from Dr. Charles W. Allen . . . 19
Harrison's Operation for Prostatic Enlargement. Letter from Dr. A. W. Bidenoug . . . 19
Chloride of Methyl as an Anodyne. Letter from Dr. George W. Jacoby . . . 19

PROCEEDINGS OF SOCIETIES.
New York Academy of Medicine. Section in Theory and Practice of Medicine. Meeting of October 6, 1888. Dr. L. C. Gray: Neurasthenia; its Differentiation and Treatment . . . 20
American Gynæcological Society. Thirteenth Annual Meeting, held at Washington, September 18, 19, and 20, 1888 (concluded). Dr. H. Marion-Sims: The Importance of the Microscope in the Treatment of Sterility in Women.—Dr. Hunter: The Influence of Pregnancy on Pelvic Diseases.—Dr. Engelmann: New Methods of Electrotherapy in their Bearing on Gynæcological Practice.—Dr. M. D. Mann: A Specimen of Ovarian Pregnancy.—Dr. J. E. Janvrin: The Indications for Primary Laparotomy in Tubal Pregnancy.—Dr. H. T. Hanks: The Early Diagnosis of Ectopic Pregnancy, with a Report of Two Cases successfully treated with Electricity . . . 23

BOOK NOTICES.
Thomson (W.).—Transactions of the Academy of Medicine in Ireland . . . 23
Gerrish (F. H.).—Prescription Writing . . . 23
Books and Pamphlets received . . . 23

MISCELLANY.
The Philadelphia Academy of Surgery . . . 24
Erysipelas and Tuberculosis . . . 25
The Northwestern Medical and Surgical Society . . . 26
The New York Academy of Medicine . . . 26
Change of Address . . . 26
To Contributors and Correspondents . . . 26

For Index to Advertisements, see next page.

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EXPLORATION OF THE CHEST IN HEALTH AND DISEASE.

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FIGURE 6. Table of contents and cover of the *New York Medical Journal*, volume 49, January 5, 1889.

challenge posed to the *Journal's* publisher by his death.

> An illustrative comment was made upon a story circulated about him and a fellow medical editor. The other was editor of the New York Medical Journal's nearest rival, the Medical Record. Both men were on the same train on their way to a medical convention. Dr. George F. Shrady turned to my father and said, "Frank, I've got your obituary all ready in my drawer and I suppose you've got mine, but who are going to edit these two journals when we're gone?"[23]

Foster was clearly a hard act to follow. Although the *Journal* continued to flourish after his death, in a historical sense, the period of his editorship was unique both for its length and for the fact that it represented a golden era of excellence.

Charles Eucharist de Medicis Sajous, MD. Following Dr Foster's death in 1911, Charles Eucharist de Medicis Sajous, MD, a Philadelphia endocrinologist, became supervising editor, with a Brooklyn, NY, physician, Claude Lamont Wheeler, MD, serving as editor. Sajous and Wheeler served as co-editors from 1912 through 1915, after which Sajous served as editor alone for 1916. Wheeler, who had an AB degree from LaVal University in Quebec, was an 1889 graduate of McGill University. He was born in Canada but came to New York City in 1891 after practicing medicine in Burlington, Vermont, for a few years. He had served as associate editor of the *Journal* under Foster for several years and thus provided the *Journal* with editorial continuity. Wheeler died on December 23, 1917, at his home in Flatbush, Brooklyn, from pneumonia at the age of 52 years.[24,25] When Sajous was appointed supervising editor in December 1911, the publishers took pains to point out that although he had not been previously associated with the *Journal* in an editorial function, he had been credited in the November 11, 1911, issue of the *Journal* with classifying "thyroid secretion as an opsonin." The publishers also quoted from a 1905 letter from Foster to Sajous in which the former said, "I have not at any time had the slightest misgivings as to your work in the study of internal secretions."[26] Thus there was a conscious attempt to confer a sort of legitimacy of editorial inheritance on Sajous from Foster whose three decades' long imprint on the *Journal* was substantial.

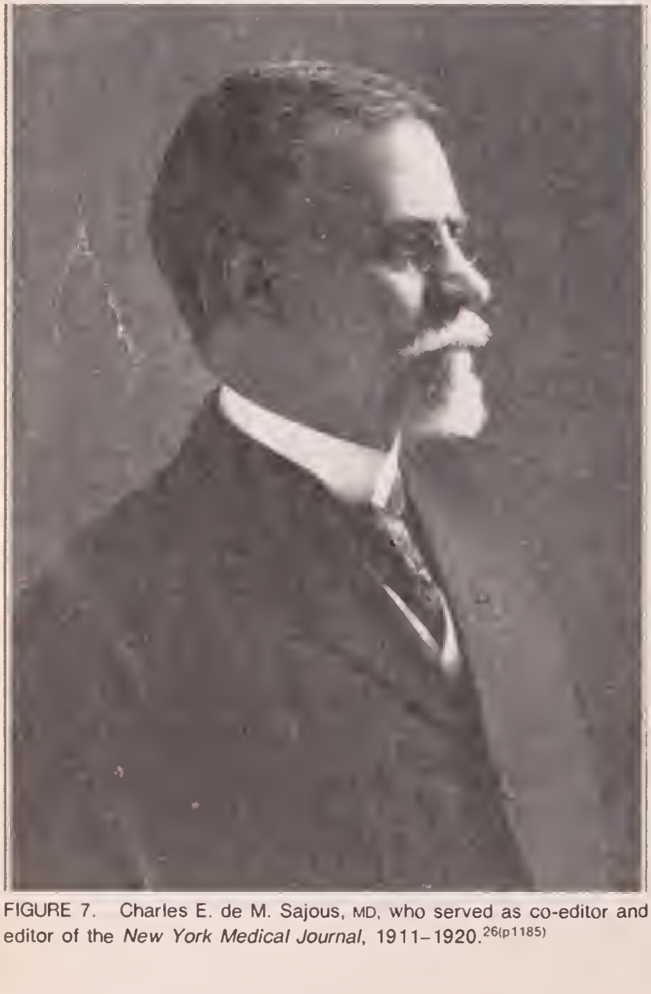


FIGURE 7. Charles E. de M. Sajous, MD, who served as co-editor and editor of the *New York Medical Journal*, 1911–1920.[26(p1185)]

Unlike Foster, Sajous was a busy clinician with a large private practice. However, he was a highly respected clinical investigator. Born at sea under the American flag in 1852 near France, he received his preliminary education in France and then attended Jefferson Medical College from which he graduated in 1878. In introducing him to readers, the *Journal* said the following:

> We have the honor to announce that with this issue, Dr Charles E. de M. Sajous, of Philadelphia, becomes Supervising Editor of the *New York Medical Journal*. While it is largely superfluous to do more than announce to our readers this important event in the history of medical journalism, yet we feel that a few details concerning the life and accomplishments of the distinguished physician will be of interest.
>
> Doctor Sajous, while born at sea under the American flag, received his preliminary education in France, along those classical lines toward which educators are again trying to bring back the early training of scientific men. In 1878 he graduated with honors from Jefferson Medical College, and for the next two years was resident physician of the Howard Hospital, of Philadelphia, where the custom of seeing and prescribing for every patient, 30,000 in the outdoor department alone, gave the young interne a firm clinical foundation in every branch of medicine, including the specialties.
>
> In 1881, Doctor Sajous was appointed professor of anatomy and physiology in the Wagner Institute of Science, lecturer in the Philadelphia School of Anatomy, and clinical assistant in the laryngological department of Jefferson Medical College; in 1883 he succeeded Dr. J. Solis-Cohen as clinical lecturer and chief of the latter department; in 1891 he departed for Paris, where he was destined to spend six years in the research work which resulted ultimately in the publication of the two volumes of *Internal Secretions and the Principles of Medicine*, a work which gave the author high rank as an original investigator. On his return from Paris, Doctor Sajous was for a time dean of Medico-Chirurgical College. On the reorganization of Temple University, he accepted the chair of pharmacology and therapeutics, which he still holds.
>
> In 1888, recognizing the scant facilities then afforded American physicians to keep abreast of modern medicine, Doctor Sajous founded the *Annual of the Universal Medical Sciences*, for which, with the collaboration of some of the best known men in America and Europe, his publishers obtained a circulation of over 500,00 volumes. Before this work suspended publication it had established a standard in American medical literature which has ever since commanded respect; and recognition was not lacking for the pioneer, who received from France membership in the Legion of Honor, from Belgium the Order of Leopold, while he was created a Knight Commander of the Liberator, and received other honors governmental and scientific. In America, Doctor Sajous has been president and vice-president of several societies, as well as Fellow of the College of Physicians of Philadelphia, and of the American Philosophical Society. The *Cyclopaedia of*

FIGURE 8. Smith Ely Jelliffe, MD, a renowned psychiatrist, who served as co-editor of the *New York Medical Journal*, 1917–1920 (courtesy of Robert Stragnell, MD).

> *Practical Medicine*, which succeeded the *Annual* in 1898, was intended more for the general practitioner and offered many of the advantages of a postgraduate course; it has already attained a sale of 240,000 volumes and the seventh edition is now in course of preparation.[26]

Thus, although Sajous was a busy practitioner, he was also a distinguished clinical scientist of the day and the author of widely read medical articles and texts. Under his editorship, the *Journal* continued to flourish. After serving alone as editor in 1916, Sajous was joined by a coeditor, Smith Ely Jelliffe, MD, in 1917. Jelliffe was a noted psychiatrist and like Foster and Hunter a graduate of Columbia University College of Physicians and Surgeons (1889). Jelliffe served as the editor of the *Medical News* from 1900 until it was taken over by the *New York Medical Journal* in 1905. He was also the editor of the *Journal of Mental and Nervous Disease* for close to 40 years. He also served as editor of the *Psychoanalytic Review* and authored numerous articles and textbooks. Although he maintained a large private practice, Jelliffe was active not only in medical writing and editing but also as a leader in organized medical groups. He served as president of the American Neurological Association, the New York Neurological Society, and the American Psycho-Analytical Society. In 1941 he gave his library of 15,000 volumes and more than 25,000 reprints on nervous and mental diseases to the Neuro-Psychiatric Institute of Hartford Retreat. He died on September 25, 1945, at his summer home near Huletts Landing, Lake George, NY, at the age of 79 years.[27]

In March 1921, the *Journal* changed from a weekly to a semimonthly.[28] Because of the demands of their private practices and other commitments, Sajous and Jelliffe left the editorship of the *Journal* in 1920. Sajous died on April 27, 1929, at his home in Philadelphia at the age of 76.[29]

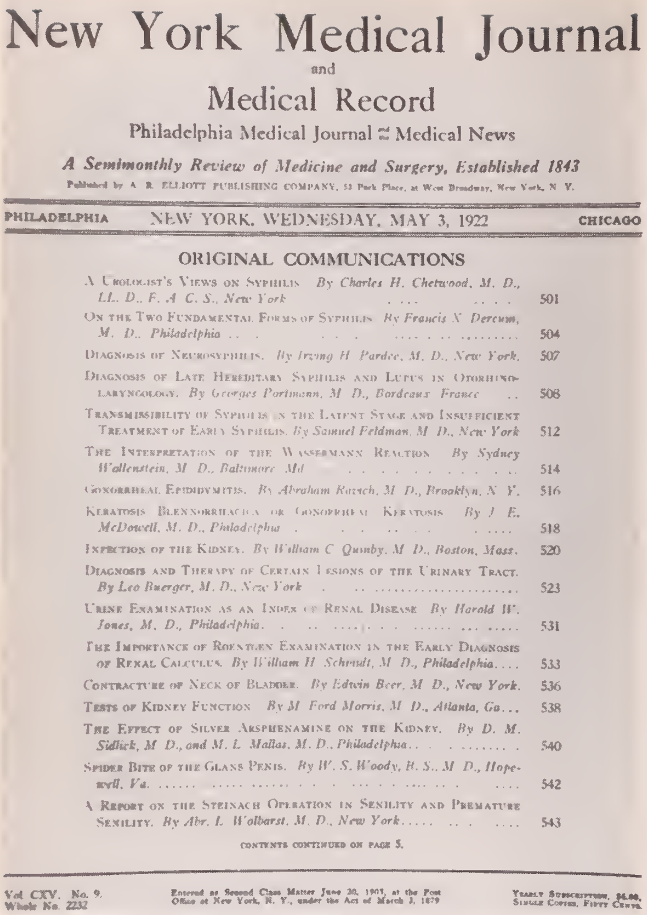
Gregory Stragnell, MD. The last editor of the *Journal* was Gregory Stragnell, MD, who like a number of his predecessors was a graduate of Columbia University College of Physicians and Surgeons (1914). Born in 1888, he assumed the editorship in May 1922[8] and continued as editor for 24 years. He began his editorship with the first issue under which the *Journal* was known as the *New York Medical Journal and Medical Record* after the acquisition of the *Medical Record*.[8] Stragnell was the son-in-law of Dr Smith Ely Jelliffe who had been previously associated with the *Journal* (personal communication, Robert Stragnell, MD, Prescott, Ariz, February 6, 1989). Stragnell edited the *Journal* under its new name until December 1923 when the name was once again changed.

FIGURE 9. Gregory Stragnell, MD, as he appeared in 1929. He served as editor of the *New York Medical Journal* and its successors, the *New York Medical Journal and Medical Record*, the *Medical Journal and Record*, and the *Medical Record* (courtesy of Robert Stragnell, MD).

NAME CHANGES AND A CHANGE IN OWNERSHIP

The *New York Medical Journal and Medical Record* became the *Medical Journal and Record* in January 1924. The basic content of the *Journal* was broadened as previously described and special supplements published with each semimonthly issue. In the early 1930s, the economic impact of the Great Depression is evident in the ever shrinking size of the semimonthly issues. Whereas volume 119 in 1924 had 632 pages plus 160 pages of supplements (separately paginated), the *Journal's* volume 138 in 1933 had 496 pages including 192 pages of supplements. Thus it was close to 300 pages slimmer than its predecessors in the mid-1920s. The supplements, instead of being separately paginated as before, were now paginated along with the regular editorial content in a probable attempt to give bulk to each issue.[30]

An editorial in the *Journal* entitled, "Doctor and Depression" provides insights into one of the reasons why the *Journal* was in difficulty. "Physicians have lost their



New York Medical Journal
and
Medical Record
Philadelphia Medical Journal and Medical News
A Semimonthly Review of Medicine and Surgery, Established 1843
Published by A. R. ELLIOTT PUBLISHING COMPANY, 53 Park Place, at West Broadway, New York, N. Y.

PHILADELPHIA NEW YORK, WEDNESDAY, MAY 3, 1922 CHICAGO

ORIGINAL COMMUNICATIONS

A Urologist's Views on Syphilis. *By Charles H. Chetwood, M. D., LL. D., F. A. C. S., New York* 501
On the Two Fundamental Forms of Syphilis. *By Francis X. Dercum, M. D., Philadelphia* 504
Diagnosis of Neurosyphilis. *By Irving H. Pardee, M. D., New York.* 507
Diagnosis of Late Hereditary Syphilis and Lupus in Otorhinolaryngology. *By Georges Portmann, M. D., Bordeaux, France* .. 508
Transmissibility of Syphilis in the Latent Stage and Insufficient Treatment of Early Syphilis. *By Samuel Feldman, M. D., New York* 512
The Interpretation of the Wassermann Reaction. *By Sydney Wallenstein, M. D., Baltimore, Md.* 514
Gonorrheal Epididymitis. *By Abraham Rozach, M. D., Brooklyn, N. Y.* 516
Keratosis Blennorrhagica or Gonorrheal Keratosis. *By J. E. McDowell, M. D., Philadelphia* 518
Infection of the Kidney. *By William C. Quinby, M. D., Boston, Mass.* 520
Diagnosis and Therapy of Certain Lesions of the Urinary Tract. *By Leo Buerger, M. D., New York* 523
Urine Examination as an Index of Renal Disease. *By Harold W. Jones, M. D., Philadelphia.* 531
The Importance of Roentgen Examination in the Early Diagnosis of Renal Calculus. *By William H. Schmidt, M. D., Philadelphia*.... 533
Contracture of Neck of Bladder. *By Edwin Beer, M. D., New York.* 536
Tests of Kidney Function. *By M. Ford Morris, M. D., Atlanta, Ga.*... 538
The Effect of Silver Arsphenamine on the Kidney. *By D. M. Sidlick, M. D., and M. L. Mallas, M. D., Philadelphia*.... 540
Spider Bite of the Glans Penis. *By W. S. Woody, B. S., M. D., Hopewell, Va.* 542
A Report on the Steinach Operation in Senility and Premature Senility. *By Abr. L. Wolbarst, M. D., New York*.... 543

CONTENTS CONTINUED ON PAGE 5.

Vol. CXV. No. 9.
Whole No. 2232

Entered as Second Class Matter June 30, 1903, at the Post Office at New York, N. Y., under the Act of March 3, 1879

Yearly Subscription, $6.00.
Single Copies, Fifty Cents.

FIGURE 10. Table of contents of the *New York Medical Journal and Medical Record*, volume 115, May 3, 1922.

homes, their savings have been depleted, their incomes reduced."[31] Since the journal was a subscription driven publication its economic viability was closely tied to the number of subscribers. As physician subscribers, hard hit by the depression, curtailed their journal subscriptions, the fortunes of the *Journal* fell. The publishers tried to deal with this by reducing the number of pages per issue. In an attempt to give bulk to each number they sequenced the supplements in with the regular editorial content. As a strategy it worked for awhile.

The A. R. Elliott Publishing Company of New York City, which had published the *Journal* since 1900 after acquiring it from D. Appleton and Company, ceased publishing it with the September 30, 1933, issue. The new publisher, The Medical Journal and Record Publishing Company, then took over publication and continued to publish the *Journal* until December 1946 when it was discontinued. This new company was headquartered in New York City.

No announcement of this ownership transfer was made in the *Journal*. However, the president and other officers of the new company were listed on the masthead. In bringing the history of the *Journal* full circle as far as ownership was concerned, the president was the then editor, Gregory Stragnell, MD. He would remain president of the company and editor until the *Journal* finally ceased publication in December 1946. Based on available evidence, it appears that Stragnell and others acquired the *Journal* from the A. R. Elliott Publishing Company through their own company, The Medical Journal and Record Publishing Company.

In January 1934, three months after the transfer of ownership was effected, both the name of the journal and its format were changed. The new name, *Medical Record*, was actually an old one, having been that of a famous medical journal that had been taken over by the *New York Medical Journal* in 1922.[8] Stragnell authored a brief statement on the cover of the January 3, 1934, issue, describing the changes and noting the new simplified name. He cited the *Journal's* principal aim as providing the most progressive and modern material in medicine and surgery.[32] The table of contents page noted that the *Journal* incorporated the *New York Medical Journal*, *Medical Record*, *Philadelphia Medical Journal*, and the *Medical News*. However, the subtitle, *A Semi-Monthly Review of Medicine and Surgery*, in use since 1921, was dropped. Although the *Medical Record*, as it was now called, represented a lineal descendant of the *New York Medical Journal*, it was also a descendant of the old *Medical Record* which had been published between 1866 and 1922 by William Wood and Company. However, its owners saw it as a continuation primarily of the *New York Medical Journal*, and used that journal's volume sequence. Thus the first issue of the new *Medical Record* in January 1934 was volume 139 following on volume 138 for 1933 for the *Medical Journal and Record*. The layout of the new *Medical Record* did not appreciably differ from that of its predecessor, the *Medical Journal and Record*, nor did the categories of articles. The trim size of the *Journal* was slightly reduced from 8½" × 11½" to 8" × 11".

Ownership of medical journals by their editors was not uncommon during the 19th century. However, it was a waning phenomenon in the first half of the 20th century. It is probable that Stragnell was not the sole proprietor of the *Journal's* publisher and that others had invested in the company. It is of interest that Stragnell's father-in-law, Smith Ely Jelliffe, MD, both edited and owned the *Psychoanalytic Review* and the *Journal of Nervous and Mental Diseases* (personal communication, Robert Stragnell, MD, Prescott, Ariz, March 15, 1989).

The cover of the *Medical Record*, which remained unchanged from 1934 until the *Journal* ceased publication in 1946, consisted of a solid color which varied with each issue and an inserted illustration. The illustrations were either of famous physicians or medical scientists or of hospitals and other medical care facilities. For example, former *Journal* co-editor Dr Smith Ely Jelliffe was featured on the December 1942 cover (vol. 155, no. 16) and Dr Charles E. De M. Sajous, a former journal editor, on the July 16, 1941 (vol. 154, no. 15), cover. In the former instance, Jelliffe's picture appeared in conjunction with a symposium on neuropsychiatry.

To Stragnell's credit, he maintained the quality of the *Journal's* content through the early 1940s. The categories of articles appearing did not differ much from those that had appeared in the *Journal's* predecessors. He published special symposia, printed on yellow paper, in virtually every issue through the end of 1942. Only six symposia were published in 1943 and three in 1944. The *Journal* was published twice a month until May 1942, when it became a monthly. In an editorial in the May 1942 issue, Stragnell explained that the reason for this was to conserve paper and aid in the war effort. He explained that articles would be terse and that all the material that appeared in two monthly issues would now be put out in one issue.[33] A number of other medical journals reduced their annual number of issues at the time in order to conserve paper.

However, even prior to the war, the *Journal's* volumes were steadily shrinking in size. Volume 139 (January–June 1934) had 680 pages whereas volume 148 (July–December 1938) had 462 pages. Volume 156 for all of 1943 had 754 pages, slightly more than the number of pages that were published in a six-month period during the 1920s. Beginning in 1943, the stacked advertising pages appearing at the beginning and the end of the *Journal* numbers were no longer separately numbered but sequenced in with the editorial content. Thus the actual size of the annual editorial well was substantially smaller given that there were from 20 to 30 pages of advertising per issue.

Stragnell regularly published the *Medical Mentor* as a supplement within the *Journal* for several years. This journal was the official publication of the American Medical Editors' and Authors' Association which had been a free standing journal between 1929 and 1934.

Stragnell did not devote all of his time to editing the *Journal*. In 1929 he became medical director in charge of research at Schering Corporation. He remained at Schering until 1944 when he joined the Heyden Chemical Corporation, a major producer of penicillin at the time (personal communication, Robert Stragnell, MD, Prescott, Ariz, February 6, 1989). Heyden had its plant in Princeton, NJ, where in 1946 it first began production of crystalline penicillin, then a new form that did not require refrigeration. It is of interest that Heyden advertised its penicillin products on the back cover of the last issue of the *Journal* (vol. 159, no. 12) in 1946. This issue also contained an unsigned article describing Heyden's new crystalline penicillin.[34]

During the 13-year life of the *Medical Record* the journal staff remained relatively stable, as did the subscription price, which was $4.00 annually. Stragnell's assistant editor from 1934 through October 1946 was A. M. Marlatt. Ms N. F. Hanf served as business manager for the *Journal* from 1934 through April 1946. She played a major role in running the daily affairs of the *Journal* (personal communication, Robert Stragnell, MD, Prescott, Ariz, March 15, 1989). Stragnell and his staff produced the *Journal* on what appears to be acid-free paper as a half century later the sheets are in an excellent state of preservation. The *Journal's* editorial offices remained in New York City for most of Stragnell's tenure, except for a brief period when they were moved to Bloomfield, New Jersey.

The *Journal* issues for 1946 show evidence of decline in the scope, quality, and amount of material published. The reasons for this are not clear. It may be that Stragnell was devoting less time to the *Journal* and also that he was unable to position it favorably in a medical publishing market in which new competing journals were appearing, such as the *American Journal of Medicine*. Whatever the reasons, the *Journal* ceased publication with the December 1946 issue, bringing to an end a medical publishing endeavor that had spanned 82 years. Stragnell, who had during the time of his editorship worked with various pharmaceutical companies, eventually became president of Organon Pharmaceuticals (personal communication, Robert Stragnell, MD, Prescott, Ariz, February 6, 1989). He later moved to California and died in Los Angeles in December 1963 at the age of 75 years.

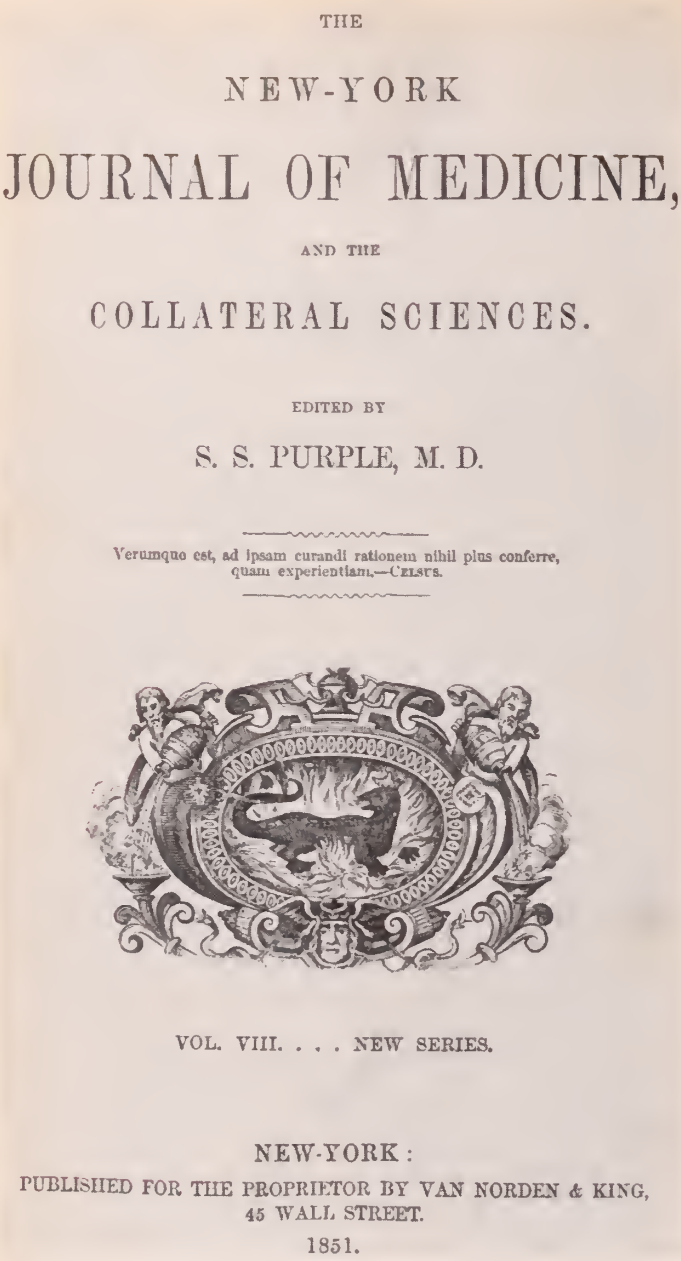
The *New York Medical Journal* was a unique medical publication in that it was a general medical journal which was not a benefit of membership of medical society affiliation. It was initially created by a man of vision, William Alexander Hammond, MD, who in addition to the *Journal* left a legacy that includes not only his medical writings but the Armed Forces Institute of Pathology.[35-37] The latter emerged from the Army Medical Museum which he founded in 1862. Hammond's creation was brought to maturity by a succession of distinguished editors. That the *Journal* played a major role in scientific medical publication for eight decades was a reflection of the excellence of its editorial leadership and the skills of its publishers who demonstrated an ability to adapt form and content to changing needs without sacrificing scientific excellence.

TWO OTHER MEDICAL JOURNALS WITH SIMILAR NAMES

The *New York Medical Journal* is often thought to have been associated in some way with the *New York State Journal of Medicine*. However, there was no association between the two journals, except on library shelves where they were filed side by side.

The *New York State Journal of Medicine*, published by the Medical Society of the State of New York (MSSNY) since 1906, began publication under that name in January 1901 under the auspices of the New York State Medical Association,[37] a medical society formed in 1884. This latter society was formed among disaffected MSSNY members who unlike the MSSNY favored a continued association with the American Medical Association. The MSSNY's medical journal, *Transactions of the Medical Society of the State of New York*, began publication in 1807. The MSSNY continued to publish it under that name until 1905. In that year the MSSNY and the splinter group, the New York State Medical Association, voted to merge and were unified under the name Medical Society of the State of New York.[39] The MSSNY then ceased publishing the *Transactions* and adopted the *New York State Journal of Medicine* as its official medical journal. There was a five-year overlap, 1901–1905, between the two publications. Although the *New York State Journal of Medicine* is now in its 89th year, its predecessor, the *Transactions*, goes back 182 years.

The *New-York Journal of Medicine* was another scientific medical journal with a similar name that had no relationship to either the *New York Medical Journal* or the *New York State Journal of Medicine*. It was first published in July 1843 and continued publication until May 1860 when it was superseded by the *American Medical Times*, which was then published from July 1860 to September 1864.[40] The *New-York Journal of Medicine* was subtitled *and the Collateral Sciences*.



THE

NEW-YORK

JOURNAL OF MEDICINE,

AND THE

COLLATERAL SCIENCES.

EDITED BY

S. S. PURPLE, M. D.

Verumque est, ad ipsam curandi rationem nihil plus conferre,
quam experientiam.—CELSUS.

VOL. VIII. . . . NEW SERIES.

NEW-YORK:
PUBLISHED FOR THE PROPRIETOR BY VAN NORDEN & KING,
45 WALL STREET.
1851.

FIGURE 11. Title page of the *New-York Journal of Medicine*, volume 8, 1851.

As this brief history indicates the lives of medical journals are more complex than one might suspect.

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Nonfatal pulmonary edema following cocaine smoking

LINDA EFFEREN, MD; DAVID PALAT, MD; JAY MEISNER, MD

As the illicit use of cocaine increases, and as the methods of ingestion vary, the number and types of complications multiply. While in some instances the mechanism by which cocaine induces various medical complications can be presumed, in other instances the mechanism is less well defined.[1,2] Reports of pulmonary complications have concentrated on the diffusion impairment seen in chronic cocaine smokers[3,4] as well as episodes of pneumomediastinum and pneumothorax following cocaine inhalation.[1] A recent case report implicated an immunologically mediated process in a nonfatal reaction to the inhalation of cocaine,[5] a mechanism that had been postulated to explain the rapidity of fatal reactions occasionally seen in narcotics abusers in the absence of documented toxic levels.[6] While pulmonary edema secondary to narcotic abuse has been well described,[7,8] there has been little evidence in the literature implicating cocaine. We report a patient who presented with nonfatal pulmonary edema following the smoking of cocaine. This clinical entity has been noted premortem in only two prior reports,[9,10] though the postmortem finding of pulmonary congestion and edema is felt to be a universal consequence of cocaine-related deaths.[2] Only one patient previously reported has survived.[9]

CASE REPORT

A 33-year-old woman presented with dyspnea, cough productive of slightly blood-tinged sputum, fever, and a feeling of chest tightness approximately 30 hours after smoking cocaine. She had a history of mild asthma treated only with an isoproterenol metered dose inhaler as needed, and she admitted to habitual recreational use of cocaine intranasally as well as by smoking. She denied intravenous drug abuse and had no other identifiable risk factors for acquired immunodeficiency syndrome (AIDS).

The patient delayed seeking medical attention for 48 hours, during which time she experienced persistent dyspnea and nonproductive cough. On presentation to the emergency room, she was found to be febrile to 101°F, but was afebrile thereafter. Her respiratory rate was 38 breaths per minute. Examination of the chest revealed scattered rales in the left lung and occasional expiratory wheezing anteriorly. Laboratory studies revealed a white blood cell count of 20.2 $\times 10^3/\mu L$ with 85% neutrophils, 10% lymphocytes, 3% monocytes, and 2% eosinophils. The hemoglobin was 9.1 g/dL with a mean corpuscular volume of 58 fL. Blood chemistry studies were within normal limits. Arterial blood gas obtained while the patient was breathing room air showed a pH of 7.44, P_{CO_2} of 34 mm Hg, and a P_{O_2} of 57 mm Hg with 90.6% saturation. Pulmonary function testing revealed a forced vital capacity of 1.06 liters, a forced expiratory volume (FEV_1) of 0.88 liters with no improvement after bronchodilator therapy. Diffusing capacity of the lung for carbon monoxide was 45% of predicted. The patient's chest film was markedly abnormal with diffuse bilateral alveolar infiltrates (Fig 1). The electrocardiogram was significant for inverted T waves throughout the precordial leads. A toxicology screen was not performed due to the delay between the patient's last reported use of cocaine and the time of presentation.

The patient was empirically started on trimethoprim-sulfamethoxazole (for presumed *Pneumocystis carinii* pneumonia (PCP)) and erythromycin, and underwent bronchoscopy with bronchoalveolar lavage and transbronchial biopsy 36 hours after admission. A postbronchoscopy chest film showed marked resolution of the alveolar infiltrates (Fig 2). By the fifth hospital day, the patient's chest film was normal, hypoxemia had resolved with a return of exercise tolerance to baseline, and the electrocardiogram had normalized.

Pathologic examination of the biopsy specimens revealed mild interstital inflammatory changes and did not show PCP, acid-fast bacilli (AFB), or fungal pathogens. Antibiotics were discontinued, and the patient was discharged in good condition.

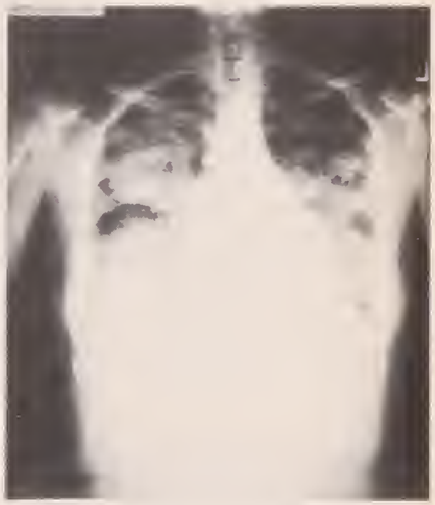

FIGURE 1. Admission posteroanterior chest film showing diffuse bilateral alveolar infiltrates.

DISCUSSION

The radiologic appearance of a pat-

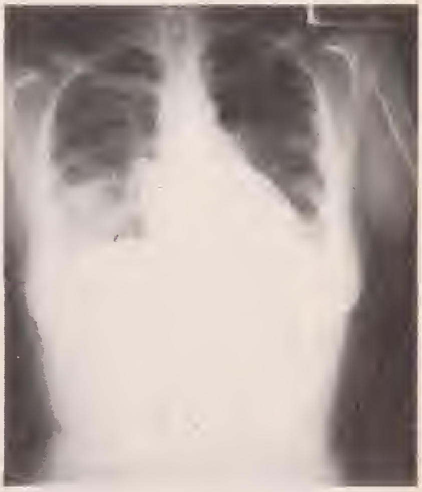

FIGURE 2. Postbronchoscopy posteroanterior chest film showing resolution of alveolar infiltrates.

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tern of ground-glass density that is diffuse and bilateral, followed by rapid and complete clearance of that density, is consistent with a diagnosis of pulmonary edema, temporally related to cocaine smoking. This diagnosis is supported by the patient's history of cocaine use as well as the rapid and clinical response in the absence of any other identifiable pathogenic agent. The pathophysiology of this phenomenon is not clear. Although the patient's electrocardiogram could suggest a cause of cocaine-induced ischemia with subsequent cardiogenic pulmonary edema, inverted T waves are a nonspecific finding and could be due to primary central nervous system or pulmonary processes. Other potential pathophysiologic mechanisms include neurogenic pulmonary edema induced by cocaine, direct pulmonary toxicity, or a hypersensitivity phenomenon. In addition, it is possible that some other substance used to dilute the drug prior to sale is responsible for the clinical findings of pulmonary edema. We were unable to obtain a specimen for analysis from this patient.

Other authors have found a high frequency of diffusion impairment in chronic cocaine smokers.[3,4] It is interesting to speculate that this patient's interstitial changes on transbronchial biopsy could be the pathologic correlate of this diffusion impairment. Of note is that in the previous case report of nonfatal pulmonary edema, diffusion was normal, as was the biopsy specimen. This is in contrast to the case of presumed hypersensitivity to cocaine, which documented both an abnormal diffusion and an abnormal lung biopsy in the presence of eosinophilia and an elevated IgE level. It seems possible that there may be multiple pathophysiologic responses of the lung in response to the inhalation of cocaine.

There currently exists a need to define the pathophysiology of cocaine-induced nonfatal pulmonary edema as well as other possible mechanisms. There is also a need for increased clinical cognizance of this entity, so that the diagnosis is considered in patients who present with similar symptoms and findings. If there is a high suspicion of cocaine-induced pulmonary edema in an otherwise stable patient, we suggest close clinical and radiographic follow-up prior to more invasive diagnostic procedures.

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Leiomyomatous tumors: Presentation of a giant gastric leiomyoma and a review of the literature

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More than 90% of tumors of gastric origin diagnosed prior to autopsy are malignant.[1] Among these, the overwhelming majority are of mucosal origin. The most common benign gastric neoplasm is the leiomyoma, a smooth muscle tumor comprising 41.7% of the total.[2] Most of these are incidental findings at autopsy or during other surgical procedures, and they carry with them an excellent prognosis. Gastric leiomyosarcomas are considerably less common, accounting for less than 3% of all malignancies of the stomach.[3,4] They hold a much less favorable prognosis.

CASE REPORT

A 52-year-old woman was admitted to Mount Sinai Hospital with a one-day history of hematemesis and melena, a 10-lb weight loss over the previous five months, and a history of early satiety. She also noted an enlarging upper abdomen over the past few months. Four days prior to admission crampy periumbilical pain developed which spontaneously resolved in two hours. Following that she felt lethargic and experienced several syncopal episodes. The patient denied any previous episodes of hematemesis, melena, or hematochezia. Her medical history was noncontributory; her surgical history included an uncomplicated appendectomy performed 20 years earlier.

Physical examination on admission demonstrated a flat, nontender abdomen containing a firm, nonmobile 25 × 25 cm mass located in the midline, with its center just above the umbilicus. Laboratory results were significant for a hematocrit of 18%, a blood urea nitrogen of 36 mg/dL, and a creatinine of 1.0 mg/dL. Her stools were positive for occult blood. Endoscopy showed a large clot located approximately 10 cm below the gastroesophageal junction along the greater curvature of the stomach. An abdominal computed tomographic (CT) scan revealed a large, round, intrabdominal mass compressing the liver and right kidney, and displacing the stomach along the left side of the abdominal wall (Fig 1).

Following admission, gastrointestinal bleeding continued intermittently until morning, requiring transfusion of three units of blood. On the day of surgery, the

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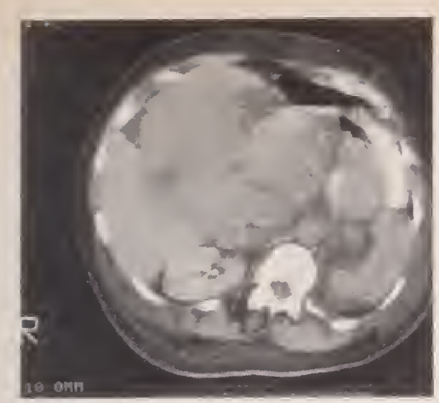

FIGURE 1. Computed tomography of the upper abdomen. A large exogastric mass is shown compressing the liver and right kidney. In addition, displacement of the stomach anteriorly is noted.

patient began to bleed again profusely, and was transfused with an additional three units. She underwent an exploratory laparotomy on the third hospital day, with a preoperative diagnosis of abdominal mass of undetermined nature. A gastric leiomyoma was considered the most likely diagnosis.

At surgery, a large mass arising from the posterior wall of the stomach next to the lesser curvature and eroding into the branches of the left gastric artery was found. It expanded posteriorly to occupy the entire lesser sac, displacing all adjacent organs. The mass was carefully isolated and excised together with a 3-cm gastric wedge. The frozen section yielded a diagnosis of leiomyoma. Careful inspection revealed no metastases. The postoperative course was uneventful, and the patient was discharged on the eighth postoperative day.

The excised specimen weighed 2,800 grams and measured 24 cm in greatest diameter. Its gross appearance was that of an ovoid, encapsulated, red-tan mass. Sectioning revealed multiple cystic spaces measuring up to 1.0 cm in diameter containing clear serous fluid within homogeneous tan tissue (Fig 2).

On histologic examination, the mass consisted of whorled, uniform bundles and intersecting fascicles of smooth muscle cells with minimal pleomorphism (Fig 3). The cells were fusiform shaped and contained centrally located vesicular nuclei with rounded ends. Mitotic activity was less than one per ten high power fields. Rare foci of nuclear palisading were present in addition to occasional areas of hypercellularity containing little intervening connective tissue. Hydropic degeneration and necrosis were scattered throughout the mass.

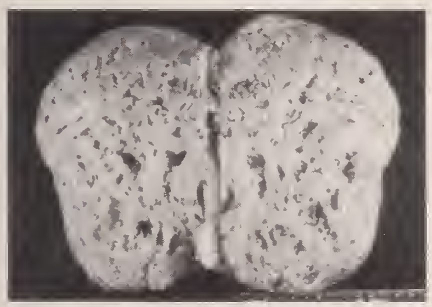

FIGURE 2. Gross appearance of sectioned mass shows multiple cystic spaces in a background of smooth muscle.

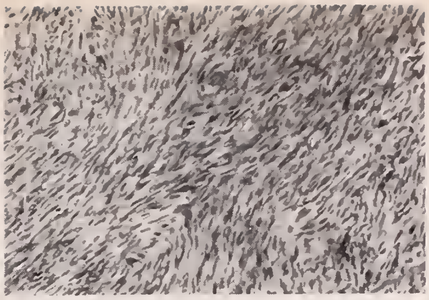

FIGURE 3. Histology of leiomyoma: intersecting bundles of uniform smooth muscle cells (hematoxylin-eosin stain, original magnification × 100).

DISCUSSION

Leiomyomas, leiomyoblastomas, and leiomyosarcomas are all included under the category of leiomyomatous tumors. Together, they present a diagnostic dilemma for the clinician, the radiologist, and the pathologist. It is nevertheless very important to distinguish among them, for they differ in terms of both prognosis and therapy.

Leiomyomas. Leiomyomas comprise the majority of smooth muscle neoplasms in the gastrointestinal tract. Most are well-circumscribed masses, frequently arising in the mid to distal stomach, especially along the anterior wall.[5] Sizes range from under 0.5 cm to upwards of 30 cm, with the mean diameter varying among the different subtypes. Less than 2% of tumors identified histologically as leiomyoma metastasize, with those that do probably being misdiagnosed leiomyosarcomas.[6] The number of mitoses is the most reliable indicator of benign status, with the frequency of mitoses for benign neoplasms generally accepted to be less than one per ten high powered fields (HPF). Although some studies have found men and women to be equally affected,[7] most have shown the tumor to have a slight predilection towards men.[5,8] The peak incidence occurs during the sixth decade of life.[6,9]

Although the majority of leiomyomas are asymptomatic, because of their relatively high frequency, they are more often implicated in causing symptoms than are their less common malignant counterparts, the leiomyosarcomas. Of those tumors that do cause patients to seek medical attention, they are often mistaken for adenocarcinoma of the stomach or, if no mass is palpable, for peptic ulcer disease. Abdominal pain independent of eating is the most frequent presentation, followed closely thereafter by melena and/or hematemesis. Although bleeding is present in up to 50% of leiomyomas, benign smooth muscle tumors account for less than 1% of cases of upper gastrointestinal bleeding.[12] A palpable mass, weakness, weight loss, and abdominal tenderness are other principal signs and symptoms. Less common manifestations include dysphagia, early satiety, vomiting, dyspnea, and dizziness. Almost all benign gastric smooth muscle tumors are found in the mid to distal stomach, with the antrum being the most common location.[5] Treatment involves local resection including adequate margins (>2 cm), with some studies recommending a more extensive resection for the larger masses.[7]

Leiomyomas are commonly divided into one of three categories based on microscopic appearance. This histopathologic classification was first presented by Stout[13] and later updated by Martin et al[14] and Stout.[8] They are as follows.

Ordinary leiomyoma. This group represents the majority of the benign smooth muscle tumors. They show varying degrees of differentiation towards smooth muscle tissue, with interlacing bundles of spindle shaped cells. Tumors in this category tend to be slightly smaller than other leiomyomas and, consequently, a smaller percentage of patients present with clinical evidence of a tumor. The five-year mortality ranges from 20% in some studies[15] to 0% in others.[7]

Cellular leiomyoma. In these tumors, the nuclei are prominent when compared to the cytoplasm, giving the neoplasm a cellular appearance. Although these tumors are somewhat larger (median size of 4 cm vs 1 cm) and cause more symptoms than ordinary leiomyomas, the overall prognosis is similar.[7,15]

Leiomyoma with regimentation. These are tumors which, in addition to the paucity of mitotic figures and lack of invasion common to all leiomyomas, show a prominent regimentation of their nuclei. This is the least common of the leiomyomas. They too carry an auspicious prognosis; Salmela and Tallqvist[15] reported a 0% mortality in eight recorded cases.

Leiomyoblastoma. The term leiomyoblastoma was first proposed by Martin et al[14] and Stout[8] to describe an unusual group of smooth muscle tumors of the stomach which macroscopically resembled leiomyomas, but which displayed completely different histologic features. This term carries no malignant implications, and all leiomyoblastomas should be referred to as either benign or malignant leiomyoblastomas. The latter group should be treated the same as the leiomyosarcomas.

Over the years, these neoplasms have been given a number of different names such as bizarre leiomyoma, epithelioid leiomyoma, epithelioid leiomyosarcoma, etc. However, in 1977, the World Health Organization reclassified them as leiomyoblastomas.[16] These tumors are characterized by a bizarre histologic appearance. They consist of sheets (rather than bundles) of slightly eosinophilic, spindle shaped cells lacking distinct myofibrils, and round or polygonal "epithelioid" cells containing clear cytoplasm.[5] Tumors having more than four to five mitoses per 50 HPF are considered to be malignant, and carry a five-year survival rate similar to leiomyosarcomas.[17]

The clinical presentation of leiomyoblastoma is similar to that of leiomyoma with respect to age, type and quality of symptoms, and radiographic findings.[7] However, these tumors are usually slightly larger than ordinary leiomyomas, and consequently are more often symptomatic. Approximately 80% of all leiomyoblastomas occur in the stomach, with 75% being found in the antrum.[18] In Stout's original series, only two of 69 patients developed metastases.[8] Abramson's[19] review of 190 patients with leiomyoblastoma revealed that only 12% went on to develop malignant disease as evidenced by documented metastases. However, Laven et al[20] found that a disturbing 38% of patients with gastric leiomyoblastomas died within three years. Most available literature has thus far supported Stout's data, showing the overwhelming majority to be benign and without metastases. The treatment for the benign tumors is the same as for leiomyomas.

Leiomyosarcoma. Patients with gastric leiomyosarcomas are similar to patients with gastric leiomyomas in having the same age and sex distribution. The tumors are considerably larger than leiomyomas, averaging about 10 cm in diameter. Almost all patients are symptomatic, with the most frequent findings being abdominal pain, a palpable mass on physical examination, recent weight loss, and melena. Few patients have their symptoms for more than a year prior to hospitalization.

Exogastric tumors outnumber both intramural and endogastric tumors combined. They tend to be larger and less differentiated than the other two, and consequently hold a worse prognosis.[17]

Approximately 40% of gastric leiomyosarcomas arise in the body of the stomach, although a disproportionate percentage involve both the fundus and cardia. They occur most commonly on the posterior wall, as opposed to leiomyomas, which have a propensity to involve the anterior wall.[5] No known association exists between the anatomic location of the tumor and a patient's age, sex, or subtype of malignancy. While the prognosis of leiomyosarcoma is better than that of gastric adenocarcinoma because of its slower growth, lack of submucosal infiltration, and lack of lymphatic spread, it is not innocuous. Previous studies show tumor invasion of adjacent organs and structures to occur in a significant number of cases, with published reports ranging from 25%[2] to 76%.[21] The five-year mortality rate correlates well with size. Appelman et al[5] found 85% of tumors 6 cm or more metastasized, whereas only 20% of tumors under 6 cm did. Metastatic spread for malignant gastric smooth muscle tumors is usually hematogenous, with the liver and peritoneum being the most frequent sites.[10,17,22,23] The size, stage, and degree of differentiation determine the prognosis, with an average five-year survival rate ranging from 30% in some studies[23] to 65% in others.[10]

The diagnosis of leiomyosarcoma can be very difficult. Weight loss and a large, tender mass on physical examination support the diagnosis of leiomyosarcoma, but these symptoms are also present in benign tumors. The majority of leiomyosarcomas show central ulceration and necrosis of the overlying mucosa with subsequent hemorrhage, but these are not specific findings. Microscopically, most leiomyosarcomas contain at least one mitotic figure per HPF, and in the absence of gross infiltration or metastases, mitotic frequency is the most consistent and reliable index for the diagnosis of leiomyosarcoma. Treatment of leiomyosarcoma is a function of tumor size. A wedge or limited partial resection is adequate for smaller tumors, while larger ones require either a total or subtotal gastrectomy.[17] There is no need to obtain extra wide margins on the stomach, as is done in treating gastric carcinomas. The efficacy of chemotherapy has not been established, and the response to radiotherapy has thus far been poor.

Summary. Nonepithelial tumors of gastric origin are infrequently encountered, with only a minority of these being clinically significant. Of those seen in a clinical setting, the most common by far is the leiomyoma. The differential diagnosis depends on the presentation, with the symptoms being proportional to the size of the tumor. Pain and bleeding are the most common symptoms. Physical examination is noncontributory in most cases of benign leiomyomatous tumors, although a mass is not infrequently palpable. A low hematocrit may or may not be present.

Sonography is the procedure of choice for the evaluation of abdominal masses.[24] With most exophytic masses, the gastric origin can be definitively established, and in a large percentage of these, the correct preoperative diagnosis is suggested.[25,26] Other advantages of ultrasound include establishing the nature of the mass, documenting the presence of necrosis, cavitation, and ascites, and determining whether or not hepatic metastases are present. CT evaluation may provide valuable additional information regarding the potential success of surgery, and CT is more sensitive than ultrasound in detecting lung metastases and determining the extent of exophytic infiltration.[27]

The sine qua non of diagnostic tests is the upper gastrointestinal series, which reveals an abnormality in more than 80% of cases.[5,6] The most consistent feature seen is a gastric filling defect. On the basis of roentgenologic findings, a diagnosis of smooth muscle tumor of the stomach can be made in more than 50% of cases.

While smooth muscle tumors of the stomach are by no means common, they are certainly not rare. These tumors may present in a myriad of ways that the astute clinician should be aware of, and surgical intervention is recommended for all leiomyomatous tumors producing symptoms. The histopathologic diagnosis of these tumors can be very difficult on limited biopsy specimens, and frozen section examinations are unreliable for the diagnosis of malignancy. The surgeon confronted with such a tumor at the time of surgery must perform a surgical procedure that would be adequate if the tumor indeed proved to be a sarcoma, but not unreasonably excessive if it proves to be benign. Because the prognosis for even the malignant tumors is clearly superior to that for other gastric malignancies, a quick and accurate preoperative diagnosis is of the utmost importance.

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Chest wall deformity following expansion of irradiated soft tissue for breast reconstruction

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Tissue expansion is a common technique for postmastectomy breast reconstruction in patients with local soft tissue deficiency. Irradiated soft tissue poorly tolerates tissue expansion. Therefore, the method is generally not recommended in this setting.

This case report describes a patient in whom tissue expansion in an irradiated field resulted in persistent deformity of the bony chest wall. No soft tissue complications were noted.

CASE REPORT

A 48-year-old postmenopausal woman was found to have a 3-cm right upper outer quadrant breast mass at the age of 39, in 1979. An incisional biopsy disclosed adenocarcinoma. She then underwent a lower axillary sampling procedure. This was followed by radiation therapy of 5,000 rads to the right breast, supraclavicular area, and axilla. In addition, she received interstitial irradiation in the form of iridium implantation in the region of the original gross tumor. Five years later, in 1984, local recurrence was suspected. A wedge-shaped excisional biopsy verified this to be an adenocarcinoma, similar in histology to the original tumor. Modified radical mastectomy was recommended. However, the patient refused to accept this unless breast reconstruction could be done at the same time. The inherent risks of using local tissue for reconstruction in an irradiated field were explained in detail to her and her husband. In spite of this, distant tissue transfer was declined. The opposite breast was estimated to be approximately 300 cc in volume and was mildly ptotic in shape.

At the time of the modified radical mastectomy, an inflatable tissue expander was introduced in a totally submuscular pocket. Very slow, gradual expansion followed during the ensuing six months, eventually reaching a volume of 455 cc. At this point, a diminution in the size of the right breast mound was noted. Mechanical failure of the expansion device was suspected. This was confirmed at surgical exploration and, indeed, a hole was found in the posterior aspect of the expander (far away from the remote feeding port). A new expander was introduced and expansion continued at a slow rate over another six months, reaching a total volume of 650 cc. It was noted at this point that the size of the breast mound that was created was not proportionate with this volume of expansion. A leak, however, was not suspected. No further expansion was carried out. During the next two months, the mound remained stable in size and became softer to the touch.

Subsequently, the expander was removed and, indeed, it was found to be intact and contained approximately 650 cc of saline. A concave depression at the undersurface of the expander was found deforming the rib cage in an inward direction, of equal depth as the height of the projection of the breast mound. A double lumen breast implant of 400 cc total volume was introduced. The postoperative course was uneventful. Though judged by the surgeon to be less than optimal, the aesthetic result of the reconstruction was satisfactory to the patient.

During the following two years, the reconstructed right breast mound became fairly soft to the touch and remained so. In the fall of 1987, a mass in the left breast was noted. Despite a normal mammogram, a biopsy proved the mass to be an adenocarcinoma.

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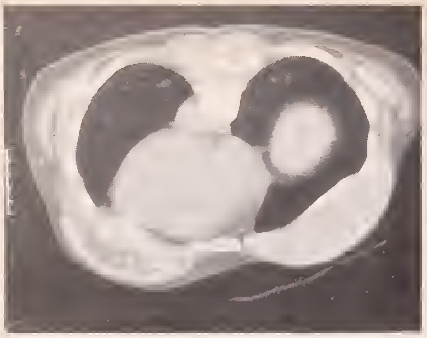

FIGURE 1. Computed tomographic scan from February 1988, two years after surgery, illustrating bilateral breast reconstruction. Note concave right chest wall deformity adjacent to silicone gel prosthesis.

The patient elected to undergo a left modified radical mastectomy. An immediate tissue expansion technique breast mound reconstruction was also carried out. The postoperative course was uneventful. The reconstructed breast domes are similar in size but not symmetric in shape. In spite of this, the patient remains content and is awaiting bilateral nipple areolar complex reconstruction.

A computed tomographic (CT) scan from February 1988 (Fig 1) clearly illustrates the persistent concave deformity of the left side of the thoracic cage adjacent to the left breast prosthesis. The right breast, which was also reconstructed by tissue expansion but received no irradiation, shows no abnormality.

DISCUSSION

Tissue expansion, a technique first reported by Neumann,[1] was subsequently popularized for clinical use by the pioneering works of Radovan[2,3] and many others. Though its application extends to practically all body regions, it is most commonly employed for postmastectomy breast reconstruction.

Research information on tissue expansion is scanty in comparison with the amount of clinical experience gained with the technique over the last ten years. Though histologic and ultrastructural changes in expanded human tissues have been studied, the data obtained are still sporadic and do not answer many questions.[4-6] It was found that the epidermis became significantly thicker during expansion but returned later to its preoperative thickness.[7] In contrast, the subcutaneous tissues and dermis were found to become thinner during expansion and thickened back to their preoperative state two years after removal of the expander. Skeletal muscles, soon after expansion, were found to undergo some atrophy. There have been no significant differences in animal research studies in the surviving length between expanded and delayed skin flaps.[8]

Various complications, such as hematoma, infection, wound dehiscence, thinning of expanded tissues, erosion, contracting capsule formation around the expander, and mechanical failure of the device have been well documented in patients undergoing tissue expansion.

Successful tissue expansion of the scalp in the pediatric and adult age groups has been reported for correction of scalp defects and male pattern baldness.[9-13] Though the method has not been recommended at an age prior to fusion of the cranial sutures, at the age of 60 days, craniopagus twins were successfully separated using scalp expansion.[14] At the time of surgery, only mild discrepancy of the bony plates adjacent to the expanders was noted. Chest wall bony deformity, not unlike the one reported in this paper, was recently described[15] in a postmenopausal woman in connection with tissue expansion for breast reconstruction. The combination of severe osteoporosis, chronic steroid use, and peripheral vascular disease present in that patient was thought to be a contributing factor. Also, in a recent letter to the editor of *Plastic and Reconstructive Surgery*,[16] another similar case was mentioned in a 43-year-old woman. Presence of "thick periexpander fibrous capsule" was thought to be responsible for the bone deformity noted. Neither of these two patients apparently received radiotherapy prior to their breast reconstruction.

Prior to these recent reports, no cranial or other osseous defects had been reported as complications of tissue expansion.

No report in the literature could be found describing the complication of bony chest wall deformity in connection with tissue expansion in a patient with past history of radiotherapy. The unyielding nature of radiation-induced soft tissue fibrosis resulted in deformation of the ribs in this case, in spite of a very slow rate of expansion.

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Psoas abscess in the neonate

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Primary psoas abscess is a distinct clinical entity.[1] It is not frequently considered in patients with signs and symptoms referable to the hip and lower extremity, particularly in neonates. A high index of suspicion is required because of the associated high morbidity and mortality of this condition if not diagnosed early and treated appropriately.

This report describes a case of primary psoas abscess in a newborn, which was diagnosed by computerized axial tomography.

CASE REPORT

A preterm male infant, 2,355 grams and 36 weeks' gestation, was born to an intravenous drug user following poor prenatal care. The infant had drug withdrawal symptoms requiring treatment. On day 18, swelling over the right groin and upper thigh with several doughy masses and overlying telangiectasia was observed.

On day 24, fever and marked leukocytosis developed. A right pelvic and hip sonogram showed echogenic masses in the pelvis and thigh with a normal hip joint.

On day 26, right hip flexion and external rotation and pain on extension were noted. A plain film of the lower extremities (Fig 1) showed marked soft tissue swelling of the right thigh. The vertebrae and other bony structures were radiologically examined and found to be normal. Computed tomography of the lower pelvis (Fig 2) showed a soft tissue mass with low attenuation areas in the psoas region, findings consistent with a psoas abscess or resorbing hematoma.

An immediate extraperitoneal drainage of the cavity was performed, yielding 35 mL of greenish-yellow pus, which grew *Staphylococcus aureus* on culture. Prompt recovery occurred within 24 hours; the appropriate antibiotic (oxacillin) was continued for 16 days.

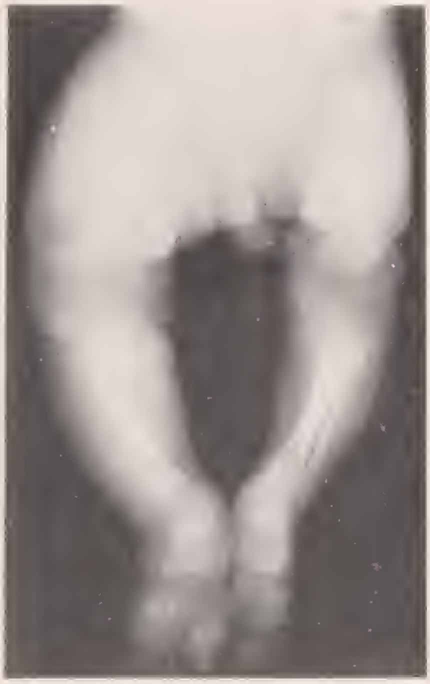

FIGURE 1. Plain film of the lower extremities showing marked soft tissue swelling of the right thigh.

DISCUSSION

Primary psoas abscess is relatively uncommon in children and adults. About 50% of reported cases occur in children below 15 years of age.[2] Most psoas abscesses are secondary to direct extension of inflammatory processes from adjacent structures, such as appendicitis or osteomyelitis of the spine. In the neonate described here, no etiologic factor could be ascertained. The infant did not have any antecedent clinical signs of sepsis, nor did he require any intravenous or arterial access.

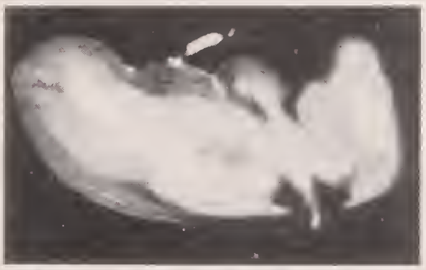

FIGURE 2. Computed tomographic section of the lower pelvis showing a soft tissue mass with areas of low attenuation in the region of the psoas.

Very rarely do neonates have primary psoas abscess; only two other neonatal cases[3,4] have previously been reported, both diagnosed by intravenous pyelography. The first case presented at 20 days of age with a five-day history of fever and a large abdominal mass extending from the right subcostal area to the pelvis; the preoperative diagnosis was a neuroblastoma.[3] The second case[4] was an infant with hyaline membrane disease, who at eight days of age developed fever, irritability, abdominal distention, and edema of the lower extremities. In both cases, adequate surgical drainage of the abscess and administration of appropriate antibiotics led to complete recovery.

The predominant causative organism of primary psoas abscess is *Staphylococcus aureus*, which was cultured in 75–100% of the reported cases.[1,5] The portal of entry is usually unknown, and a history of antecedent infection or trauma is often lacking. Among the neonatal cases, this organism was cultured from the patient presented here and from the three-week-old infant who presented with a large abdominal mass. *Klebsiella* was recovered from the eight-day-old infant.

Primary psoas abscess is a difficult diagnosis to establish, unless a high index of suspicion is raised. Consequently, treatment is often delayed. Smith et al[5] reported eight children ranging in age from 12 to 15 years with psoas abscess. Among these, the correct diagnosis was suspected in only one patient,

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and the delay in instituting appropriate therapy averaged five days.

The clinical presentation of the neonates mentioned in this report demonstrates diversity in manifestations, except for fever, which was found in all three. One infant presented with a large abdominal mass simulating neuroblastoma; the second had abdominal distention and edema of the lower extremities. In the patient described here, swelling developed initially over the groin and upper thigh with overlying telangiectasia, giving an impression of hemangioma. Six days later, fever and marked leukocytosis developed. In two days, the infant maintained a position of constant flexion and external rotation of the involved hip, signs strongly indicating a psoas abscess.

In the past, retroperitoneal abscess, as in the first two reported neonatal cases, was diagnosed using an excretory urogram, which would show displacement of the ipsilateral kidney and ureter and compression of the urinary bladder. Improved imaging techniques obviate the need for intravenous pyelography. The case reported here is the first neonatal case of psoas abscess diagnosed by computed tomography to our knowledge.

Surgical extraperitoneal incision and drainage, in addition to administration of appropriate antimicrobial therapy, has been the traditional management of iliopsoas abscess. Recently, percutaneous needle and catheter drainage guided by ultrasound or computed tomography[6] has been successfully used in adults; this nonoperative alternative has not been reported in infants and children.

In conclusion, primary psoas abscess should enter into the differential diagnosis of primary hip disease such as septic arthritis in the neonate. With a high index of suspicion, one should also consider this diagnosis in patients presenting with a tender mass in the groin or abdomen and swelling of the lower extremities. Prompt diagnosis can be made from a careful history and physical examination, aided by computed tomography, thereby avoiding other unnecessary diagnostic studies and delay. Immediate extraperitoneal drainage leads to rapid recovery.

Acknowledgment. The authors thank Roseann Bertino for typing the manuscript and Dr Jack Haller for interpreting the radiologic studies.

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Pulmonic valve replacement for pulmonic insufficiency one year after mitral valve replacement

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Pulmonary hypertension is common in patients with mitral stenosis. However, the incidence of pulmonic valve insufficiency, its reversibility with resolution of pulmonary hypertension after mitral valve replacement, and its contribution to right ventricular failure are not well understood. We describe a patient who underwent mitral valve replacement for mitral stenosis. Despite a significant reduction in the pulmonary artery pressure after surgery, the patient subsequently needed a pulmonic valve replacement for severe residual pulmonic insufficiency associated with progressive right ventricular failure.

CASE REPORT

A 67-year-old woman with a history of rheumatic fever at age 13 and again at age 19 was admitted to New York University Hospital on January 6, 1986, with severe shortness of breath. She had had atrial fibrillation and orthopnea and progressive exertional dyspnea for many years without edema. On admission, she was short of breath at rest (New York Heart Association class IV).

On examination, the patient's blood pressure was within normal limits, and her heart rhythm was irregular at a rate of 85 beats/min. Her respirations were unlabored and her chest was clear. She had neck vein distention at 30° and a right ventricular lift. The first heart sound was accentuated, and the pulmonic component of the second heart sound was loud. There was a grade III/VI diastolic rumble at the apex as well as a grade III/VI diastolic, decrescendo blowing murmur at the left upper sternal border. There was no hepatomegaly or edema.

Cardiac catheterization showed mitral stenosis with a calculated valve area of 1.5 cm^2, minimal aortic insufficiency, a mean right atrial pressure of 11 mm Hg, a pulmonary artery pressure of 78/30 mm Hg, normal coronary arteries, and a well-contracting left ventricle. The calculated pulmonary vascular resistance was 3.2 Wood units (256 dyne-cm sec^{-5}). In addition, Doppler echocardiography documented significant pulmonic insufficiency with a rapid decay in velocity.

The patient underwent uncomplicated mitral valve replacement with a size 29 Carpentier-Edwards porcine prosthesis. The excised mitral valve showed fibrosis, calcification, and neovascularization.

The patient was lost to follow-up until 15 months later, when she presented with profound fatigue on minimal exertion and a 10-lb weight gain. The examination was notable for a pulsatile liver, severe lower extremity edema, and a pulmonic insufficiency murmur that had become louder and harsher.

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Doppler echocardiography showed normal prosthetic mitral valve function, as well as pulmonary artery, right atrial, and right ventricular dilation, severe pulmonic regurgitation, and severe tricuspid regurgitation. Cardiac catheterization showed a mean right atrial pressure of 20 mm Hg, a pulmonary artery pressure of 50/26 mm Hg, a mean left atrial pressure of 17 mm Hg, and a left ventricular end diastolic pressure of 22 mm Hg. Her cardiac output was 3.6 L/min, her cardiac index was 2.3 L/min/m^2, and her calculated pulmonary vascular resistance had fallen to 1.7 Wood units (136 dyne-cm sec^{-5}).

The patient underwent pulmonic valve replacement with a size 27 Carpentier-Edwards porcine prosthesis, and tricuspid valve annuloplasty. At surgery, the pulmonary artery was markedly dilated. Two of the pulmonic valve leaflets had a normal appearance. The third leaflet was somewhat fibrotic, but histopathologic examination did not suggest rheumatic valvulitis. After surgery, the patient's cardiac output increased to 6 L/min, and the patient experienced a marked increase in her exercise tolerance and sense of well-being. Echocardiography showed normalization of the internal dimensions of the right heart chambers. When the patient was examined six weeks after the pulmonic valve replacement, she had no diastolic murmur, less hepatomegaly, and no lower extremity edema.

DISCUSSION

In patients with mitral stenosis complicated by pulmonary hypertension, mitral valve surgery often results in profound decreases in pulmonary artery pressure and pulmonary vascular resistance as early as 24 hours after surgery.[1,2] In those patients whose condition deteriorates despite valve surgery, persistent pulmonary hypertension is often the cause.[3]

Despite the marked pulmonary hypertension frequently associated with mitral stenosis, pulmonic insufficiency complicating mitral stenosis seems to be relatively rare (approximately 1% in one study).[4] In most cases it results from dilation of the pulmonary artery secondary to pulmonary hypertension. In the majority of patients who undergo mitral valve replacement, resolution of the pulmonic insufficiency parallels improvement in the pulmonary artery pressure.[4] In the patient presented here, the pulmonary vascular resistance decreased significantly after mitral valve replacement. Nonetheless, the pulmonic insufficiency progressed, and right ventricular failure ensued.

Pulmonic insufficiency requiring operative repair is very uncommon. In some patients who have undergone repair of tetralogy of Fallot, pulmonic valve replacement is necessary to correct significant residual pulmonic insufficiency and progressive right ventricular failure. In these cases, pulmonic valve replacement is associated with significant improvement in right ventricular function.[5-7]

A search of the literature has not revealed any previously reported cases of post-mitral-valve-replacement pulmonic insufficiency requiring pulmonic valve replacement. In the patient presented here, the pulmonic insufficiency was due to pulmonary artery dilation rather than primary valvular pathology. The patient, who presented with severe right ventricular failure after surgery for mitral stenosis, had marked symptomatic, hemodynamic, and echocardiographic improvement after pulmonic valve replacement. The patient also had tricuspid insufficiency. We believe this was caused by right ventricular dilation secondary to progressive and severe pulmonic insufficiency, and it was treated with annuloplasty at the time of pulmonic valve replacement. Although pulmonic insufficiency was significant prior to mitral valve replacement, it did not remit with postoperative reduction in the pulmonary pressures. In fact, the murmur of pulmonic insufficiency got louder and dramatically harsher, which prompted consideration of pulmonic valve replacement. In patients with ongoing or worsening right ventricular failure after surgery for mitral stenosis and reversal of pulmonary hypertension, unremitting pulmonic insufficiency may be the cause and may mandate replacement of this valve.

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LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Omphalocele at 15 weeks

TO THE EDITOR: The case report appearing in the November 1988 issue of the *Journal* regarding sonographic detection of an omphalocele in a 15-week fetus[1] and the accompanying commentary[2] should give us all cause to consider the implications. This is indeed an instance in which rapidly improving technology may force us into a number of ethical and moral dilemmas before we are fully ready to deal with them.

There is no question that present day sonographic equipment will allow the elegant studies demonstrated by this well-documented case report. However, I noted in the case history that no other sonographic abnormalities were found. On the basis of the finding of an omphalocele as an isolated congenital defect, it would seem that interruption of the pregnancy was recommended. I inferred that only at postmortem examination were the cardiac anomalies noted and that chromosomal studies were completed only after termination.

Termination on the basis of a non-lethal congenital defect may lead us down a path which we do not wish to follow. We have all heard anecdotal reports of termination on the basis of fetal sex. However, more central to this issue is the problem only alluded to in the commentary by Dr Smerud.[2] Many of us in pediatric surgery have seen babies whose mothers had been referred prenatally with the diagnosis of omphalocele. At birth, the baby is found to have only a very large base of the cord or a very small hernia of the cord. Gastroschisis, with implications of very few associated anomalies, has also been the outcome. Termination in these instances would have been tragic.

As the marginal training of some ultrasonographers has caused a variety of conditions to be missed, it may also lead to unnecessary terminations. I realize that being a pediatric surgeon makes what I write seem somewhat biased, but as a human being I have great difficulty accepting such definitive therapy based on frequently uneven diagnostic ability.

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1. Romano KR, Manfredi OL, Farley T, et al: Omphalocele: Sonographic detection at 15 weeks' gestation. *NY State J Med* 1988; 88:596-597.
2. Smerud MJ: Omphalocele at 15 weeks. *NY State J Med* 1988; 88:569-570.

In reply. I agree that technology's acceleration has potential for enormous benefit as well as for presenting morally difficult decisions. However, providing the patient with maximal diagnostic information outweighs the negative potential for which Dr Ratner justifiably has concern.

In the case of the fetus found to have an omphalocele, the patient was extensively informed about the nature of the abdominal wall defect (ie, an omphalocele vs gastroschisis) and the possible associated congenital defects. Based on all available information and counseling from the department of obstetrics and gynecology, the patient reached an independent decision to terminate the pregnancy.

I believe, as does Dr Ratner, that unnecessary terminations would be tragic. Therefore, serial sonographic follow-up and the use of other diagnostic adjuncts are essential; this includes ultrasound guided amniocentesis, alluded to by Dr Smerud.[1]

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In reply. Dr Ratner has raised an important point which I did not address. I wrote of the tragedy of missing anomalies at prenatal obstetrical ultrasonography, but equally important and sometimes more tragic is the case of diagnosing an abnormality which is not present. Modern-day ultrasound equipment allows us to visualize the fetus and its parts at an earlier stage than ever before. Because of this, it is extremely important that these examinations be performed by well-trained and experienced individuals who can differentiate between normal variation and subtle abnormality. In difficult cases, repeated studies and second opinions rendered by other skilled sonographers can minimize errors.

The ethical question of pregnancy termination on the basis of ultrasound-detected fetal anomalies has no easier answer than the ethical question of pregnancy termination in general. That significant fetal abnormalities can be documented in utero is without question. Some of these are lethal, others will result in mental or physical handicaps, and others are of little consequence or easily correctable. Termination on the basis of a nonlethal congenital defect is, in the final analysis, a decision of the parents. It is more important than ever that we help them in their decision by providing the best possible characterization of the abnormality.

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New York State's do-not-resuscitate law

TO THE EDITOR: Robert Swidler's welcome discussion of New York State's do-not-resuscitate (DNR) law in the February 1989[1] issue leaves major questions unanswered.

Mr Swidler contends that because the DNR law simply permits resuscitation attempts in the "non-DNR" patient there is no explicit or implicit duty to perform CPR. He correctly notes that permission to perform a procedure does not imply a duty to perform the procedure. The logical conclusion is that CPR need not be attempted if it is clearly going to be ineffective. This is of course in keeping with standard medical practice, which dictates that physicians are under no obligation to provide clearly ineffective therapy.[2-4]

What, then, constitutes clearly ineffective CPR? Although the law states that this is a medical determination based on the statute's definition, Mr Swidler only mentions a narrow application. He refers to an "on the scene decision" in an emergency room or delivery room. One can presume he is referring to the sudden presentation of a patient in a state incompatible with survival (ie, decapitation). Most physicians, however, believe there are other circumstances in which CPR would be futile. There is an expanding body of medical literature documenting the failure of CPR in certain patient groups.[5,6] There are some patients for whom any physician will agree CPR will be futile in advance of the emergency situation cited by Mr Swidler. He does not address this possibility and one assumes DNR orders in such cases still require patient or surrogate consent.

If medical futility cannot be invoked unilaterally before an emergency, then the physician is left with the absurd option of offering a procedure to the patient (or surrogate) which will not work. Despite the stated premise that patients and surrogates will make rational decisions when provided with a realistic assessment of burdens and benefits, the mere act of offering a choice presupposes there is a possible benefit.[6] This is confusing at best and a cruel hoax.

In summary, the new legislation gives the illusion of permitting medical decisions regarding medical futility of CPR. In fact, this is restricted to narrow applications, as a consideration for surrogates or a decision by the physician only where there is no surrogate for a patient who lacks capacity. We are now told that an "emergency exception" also exists for a patient who happens to arrest in front of his physician. Simple logic and traditional medical practice dictate that the first question to ask is, "Could CPR possibly work?" Medical futility should not be relegated to the bottom of a decision tree for those few solitary incapacitated patients with no friends or family.

On the other hand, if the writers of the law believed that it is not medically possible to predict futility, they should have left it out of the law entirely. If it is truly a question of medical judgment as the law indicates, then the whole matter should end whenever two physicians certify that CPR will not work.

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[1] Swidler RN: The presumption of consent in New York State's do-not-resuscitate law. *NY State J Med* 1989; 69-72.

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The do-not-resuscitate law

TO THE EDITOR: Dr Phillip Swender is obviously a compassionate physician. Unfortunately, many of his reflections about the do-not-resuscitate (DNR) law in New York, published in the February issue, are premised on a misunderstanding of the law.[1] Dr Swender acknowledges that the duty to obtain the patient's consent to a DNR order orally or in writing is "not new," but he bemoans the need to reaffirm the patient's wishes every three days. The law imposes no such requirement. Physicians must review the order every three days for hospital patients, but the patient, or others who consent on the patient's behalf, need only consent once.[2]

Dr Swender voiced concern that it would be "tragic" if a patient with a valid DNR order were to be denied life saving measures when he or she choked on a piece of food. The legislature agrees. In defining cardiopulmonary resuscitation (CPR), the law explicitly states that "cardiopulmonary resuscitation shall *not* include measures to improve ventilation and cardiac function in the absence of an arrest."[3,4]

Dr Swender also deplored the use of blue dots on the patient's medical chart to indicate "no code" status, describing the dots as a stigmatizing symbol. The law requires what common sense and sound medical practice would suggest: that physicians inform other members of the health care team that a DNR order has been issued for the patient.[5] Presumably, the hospital devised the blue dot system. Since Dr Swender states that many employees, when asked, had no idea what the dot meant, the system obviously does not work. Dr Swender should advocate within his hospital for a policy he finds more suitable, and for better communication among health care professionals, to ensure that the patient's or family's wishes about CPR are not abrogated by administrative failure.

Finally, Dr Swender laments that the DNR law makes "passive consent" from patients and family members a thing of the past. Here he has accurately interpreted, and touches upon a major ethical and policy judgment embodied in the law. Although Dr Swender asserts that, "To a sensitive and compassionate caregiver, feelings and wishes are rarely ambiguous . . . ," studies consistently suggest otherwise. One study found that physicians treating the same patient frequently did not agree about the patient's preferences regarding a DNR order.[6] The same study showed that physicians' assumptions and patients' wishes often diverge; of 25 patients who were resuscitated and left the hospital, eight stated that they had not wanted CPR.[6] A more recent study echoed these findings; physicians incorrectly predicted patients' wishes about CPR 18.2% to 48% of the time, despite the fact that physicians believed in 183 out of 233 (78.5%) cases that their judgments were accurate.[7] While physicians frequently underestimated patients' desire for CPR, patients' spouses consistently erred in the other direction, making clear the limitations of passive consent.[7]

These studies do not point to physicians' insensitivity but to a persistent reluctance to discuss CPR and the wide margin for human error in assessing the feelings of others about decisions as intensely personal as those made at the end of life.[8-11] When physicians do not have the kind of long-standing relationship with patients that Dr Swender recounted, the margin for error expands into simple guesswork.

Discussing CPR with patients is not always easy for physicians. But it is often welcomed by patients ready to accept the limits of medical technology but still desiring the human contact and respect that a dialogue entails. In the hands of compassionate physicians, that dialogue need not destroy hope, but can convey caring for the patient and recognition of the special partnership between patient and physician.[11]

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[1] Swender PT: Reflections on the New York do-not-resuscitate law. *NY State J Med* 1989; 89:57-58.

[2] NY Public Health Law, Art 29-B, § 2970.

[3] NY Public Health Law, Art 29-B, § 2961(4).

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In reply. Ms Miller's response to my reflections appears to miss the point of my commentary. I am quite aware that the law does not require reaffirmation of a patient's wishes every three days.[1] It would be irresponsible, however, to renew an order of such seriousness without periodic discussion with the patient or family. If consent to a do-not-resuscitate (DNR) order is a one-time event, then renewal every three days is meaningless, except as an exercise in control. The resolution of issues of life, terminal illness, and death is not a single event, but rather a process, the intervals and nature of which should be determined by the participants, not a law.

Regarding the hypothetical choking patient with a valid DNR order, Ms Miller quotes the law, "cardiopulmonary resuscitation shall *not* include measures to improve ventilation and cardiac function in the absence of an arrest."[2] Sometimes, choking people experience an arrest. To expect clinicians to apply all due measures to the choking patient until the momentary cessation of heart or lung function evokes the DNR order reflects a misunderstanding of the intense environment in which these events occur. Arrests do not proceed in a predictable fashion. No matter how the law defines cardiopulmonary resuscitation, it will share actions in common with "measures to improve," and the moral dilemma with the choking patient will be intensified, not lessened.

I agree that the "blue dot" system was an ineffective means of communication. It is also reassuring to know that the law expects common sense and sound medical practice in such communication.[2] I find little to suggest that this was not the case, prior to the law. I am unaware of data to suggest that patients with written DNR orders were frequently resuscitated in error. Common sense and good medical practice seem to have prevailed, albeit in a way less demonstrable to a reviewer. Do we need legislation to establish things that already exist?

Ms Miller cites several references in support of the limitations of passive consent.[4–8] I interpret these studies somewhat differently. They do not speak to the limits of passive consent, but rather the perils of noncommunication. Passive consent requires greater communication, trust, and understanding than a single active affirmation. While not appropriate in every instance, passive consent should not be forbidden because of an erroneous perception that it is synonymous with noncommunication. The studies speak to the need for a greater emphasis on communication skills in medical training, not necessarily the need for a law.

The controversy surrounding the DNR legislation reflects a deeper conflict between those who practice medicine and those who regulate it. No reasonable person would suggest that medicine should be practiced without constraints. In the regulatory extreme, however, protocol replaces judgment, regulation replaces trust, and decisions are made based on fear of the consequences of noncompliance, rather than the best interest of the patient. This law will neither force compassionate communication nor reduce human error and it is naive to think that it can accomplish these lofty aims. The relationship between a physician and a dying patient is a special partnership. The State of New York should not be one of the partners.

I have repeatedly been told that I do not understand the law. I humbly suggest that those who are responsible for the law may not completely understand the practice of medicine. Perhaps our continued dialogue will serve to further enlighten each of us.

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Typhlitis

TO THE EDITOR: The possible pathogenetic mechanisms of typhlitis (neutropenic enterocolitis, ileocecal syndrome) in patients with natural or induced neutropenia were discussed fairly comprehensively by Kunkel[1] in his commentary in the February 1989 issue of the *Journal.* While he mentioned the use of antineoplastic drugs and broad spectrum antibiotics as probable inciting agents, and the bacterial and fungal organisms associated with typhlitis, Kunkel neglected to include viral infections of the cecum. Cytomegalovirus (CMV) in particular is known to be an opportunistic invader of this region of the gastrointestinal tract in immunodeficient patients. This member of the herpes group tends to infect vascular endothelial cells, promoting thrombosis and hemorrhage. CMV infection has been documented in the gastrointestinal tracts of renal and bone marrow transplant recipients.[2,3] Acute ulcerative typhlitis with intense hemorrhage requiring ileo-colic resection in a 29-year-old leukopenic woman who was receiving multiple drug chemotherapy for myelogenous leukemia was reported from our institution.[4] Pathologic study of the cecal ulcer disclosed CMV inclusions within vascular endothelium and smooth muscle cells. Appropriate culture or endoscopic biopsy for the identification of CMV as an etiologic agent in acute ulcerative typhlitis in immunodeficient and/or neutropenic patients may permit the institution of timely and effective new antiviral therapy. This has the potential of reducing morbidity and mortality in a subset of these highly vulnerable individuals.

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Judging medicine

Judging Medicine. By George J. Annas, JD. 448 pp. Clifton, NJ, The Humana Press, Inc, 1988. $24.95 (hardcover)

George Annas, a law professor at Boston University School of Public Health, is a prominent figure in the field now commonly called bioethics. His books, lectures, participation in governmental commissions, and frequent media appearances have greatly influenced professional and public views about ethical and legal issues in medicine. But Professor Annas' most accessible work, and the work for which he is probably best known, is his bimonthly column in the *Hastings Center Report*, a leading bioethics journal. Since 1976, his column has concisely informed readers about the latest developments in law and medicine, and eloquently argued why those developments should be welcomed or deplored.

Judging Medicine is a selection of essays from Professor Annas' column, with a few other Annas pieces included for good measure. The essays are grouped into eight categories: patient's rights; conception, pregnancy and birth; reproductive liberty; medical practice; the mentally retarded and mentally ill patient; death, dying and refusing medical treatment; government regulation; and transplants and implants.

Throughout his essays, Annas advocates the rights of hospital patients to greater self-determination in treatment decisions, to equal care regardless of wealth, to a more humane environment. He is often sharply critical of physicians and hospitals for not taking patients' rights seriously, or for elevating their own professional and institutional interests over those of their patients.

Indeed, Annas' depiction of how hospitals and doctors treat patients is certain to irritate many readers. He writes of patients turned away at the emergency room, or transferred for financial reasons while still critically ill. He describes doctors' disregard of, and sometimes contempt for, the principle that a patient's consent should be informed. He denounces experimental procedures that impose enormous burdens on patients with little possibility of therapeutic benefit. He exposes hospitals and doctors who provide futile, unwanted treatment to dying patients because of their belief, almost always unwarranted, that the law requires them to do so.

This is harsh stuff. But Professor Annas' critique of medical practice, though sometimes polemical, is never irresponsible. One can debate the extent of such problems but not their existence. Nor does Annas limit himself to judging medicine: he is often equally critical of lawyers, legislators, and judges, especially when they purport to act as doctors. (In fact, several of the essays might more aptly have been compiled under the rubric "Judging Judges.")

The lodestar for Professor Annas is the patient's own view of his or her interests. Once that view is identified, Annas' assessment of doctors, lawyers, and others depends on whether they respect or obstruct it. Thus, in an essay entitled "Prisoner in the ICU: The Tragedy of William Bartling," Annas supports the doctors who humanely wanted to honor a competent, dying cancer patient's wish to forgo mechanical ventilation, and castigates both the hospital lawyers who opposed the request and the lower court judge who ordered the ventilation continued. (That decision was later reversed by the California Court of Appeal.[1]) In contrast, in two other essays Annas applauds legislators and courts for defending a woman's right to be informed about alternatives to a mastectomy and to opt for home birth; he faults medical organizations for resisting those rights.

In some instances, Annas sees medicine and law aligned in opposition to patients. For example, in "the Unkindest Cut of All," Annas argues that a woman's refusal to undergo a Cesarean should be honored, even when that decision places her fetus and herself at greater risk. He denounces doctors who sought, and courts that permitted, the performance of forced Cesareans on objecting women.

Similarly, Professor Annas takes the part of Elizabeth Bouvia, a 26-year-old paraplegic who entered a California hospital for comfort care while she carried out her plan to stop eating and die. Bouvia's physician sought to feed her by nasogastric tube, but she resisted and commenced a widely publicized lawsuit. In his essay, "When Suicide Prevention Becomes Brutality," written after the lower court authorized the hospital to force-feed Bouvia, Annas criticizes both the doctors and the court. (This decision, too, was reversed by the California Court of Appeal.[2]) To Annas, the case presented a straightforward application of a longstanding legal principle: Competent adults have the right to refuse any medical intervention, including tube feeding. While the lower court found that permitting Bouvia to starve would have a devastating effect on the medical staff, nurses, and other patients at the hospital, Annas rejects the relevance of that finding. He contends that the patient has a far greater stake in the decision than "unnamed others," and suggests that the prospect of hospitals force-feeding patients poses a greater threat to medical integrity and morale than honoring patient choices.

As Annas states, "medical care must be consensual or it loses its meaning."

When he is unable to rest his position on patient self-determination, Professor Annas is more cautious and circumspect. For example, in his essays on life sustaining treatment decisions for incompetent patients, he acknowledges his concern about the application of the "substituted judgment" principle (the principle that a surrogate decision maker should strive to make the treatment choice that the patient, if competent, would make) to cases in which there is no basis for ascertaining what the patient would have decided. In particular, his essay "Mary Hier: When Substituted Judgment Becomes Sleight of Hand," attacks the doctors and court for characterizing their decision to withdraw treatment from a demented patient as a "substituted judgment" without adequate knowledge of what Mrs Hier would choose.

On the other hand, Annas endorses the general principle that decisions about life sustaining treatment for incapacitated patients should be based on a substituted judgment, when the individual "was previously competent and had a value system that a close family member or friend could reasonably understand," or, when there is no basis for making a substituted judgment, an evaluation of the patient's best interests. Consequently, in these essays and other writings,[3,4] Annas is highly critical of New York Court of Appeals decisions which hold that life sustaining treatment may be withdrawn only when there is "clear and convincing evidence" that the patient would want it withdrawn.[5,6] In Annas' opinion, the court set a burden of proof that effectively denies patients the right to refuse treatment, without allowing their families and physicians to make humane decisions to stop treatment based on the patient's best interests.

Annas' patient-centered perspective leads him into another dilemma when patients compete with one another. One of the most intriguing essays in the collection is "The Prostitute, the Playboy, and the Poet," which compares alternative schemes for allocating scarce life saving medical resources. The essay captures the difficulty in framing an ethical answer to the simple question: Who should get the next organ? The next person on line? The most medically urgent? The best matched? The patient who can pay the most? The patient with the greatest "social worth?" Should the rules of the game be explicit, or finessed somehow to avoid the intolerable symbolism of publicly denying life saving resources to identifiable patients? Annas, acknowledging the inadequacy of every approach, argues for a system of first come, first served, with allowance for urgent patients to jump queue.

Toward the end of his essay on recipient selection, Annas suggests that the organ shortage would be alleviated if, among other measures, "individuals can be persuaded not to join the pool." While there may well be a serious allocation-of-resource problem on the horizon, the statement is still jarring. It conjures up images of *Soylent Green*, the film about an overcrowded future society, in which citizens are given incentives to volunteer to be put to death. There is no doubt that some informed patients decline to undergo life saving transplants, but the notion of persuasion, with an implicit gatekeeping role assigned to the patient's physician, is indeed troubling.

Annas also contends, sometimes rather stridently, that transplant surgeons have cajoled patients or their families into consenting to highly experimental procedures that pose more burdens than benefits. He is particularly incensed by the Baby Fae case, in which a baboon heart was transplanted into an infant, and by the implantation of mechanical hearts into William Schroeder and Barney Clark. Annas' primary concern is that these experiments became an end in themselves, divorced from any sober assessment of the patient's interests. At a recent lecture, Annas commented that the participants in the mechanical heart experiments came to view the patient as keeping the heart alive, rather than the other way around.

Another recurrent theme in *Judging Medicine* is an emphasis on legislation as a remedy for problems in medical practice. In various essays, Professor Annas endorses legislation, among other purposes, to ensure patient access to records, to guarantee women greater information and choice in treatment of breast cancer, to settle legal questions raised by the cryopreservation of embryos.

Interestingly, though, Annas sees no general need for brain death legislation or living will legislation. According to him, a determination of death by generally accepted medical standards is necessarily lawful, and brain death statutes are superfluous and potentially mischievous. As for living wills, Annas emphasizes that adult patients have a firm legal right to refuse treatment which must be honored, irrespective of whether the jurisdiction has a living will statute. These observations seemed particularly accurate with regard to New York: Although there is no New York statute regarding brain death, a Court of Appeals decision,[7] state task force report,[8] and subsequent Health Department regulation[9] unequivocally confirm the legality of determining death by brain-based criteria in New York. Similarly, while there is no legislation governing the use of living wills in New York, the Court of Appeals has consistently held that life sustaining treatment may—indeed, ordinarily must—be withdrawn or withheld when there is clear and convincing evidence that a patient would want it withdrawn or withheld.[5,6] In its recent decision, *Matter of O'Connor*,[5] the Court explained that a living will would be "ideal" proof of a now incapacitated patient's wish to forgo life sustaining treatment.

Overall, *Judging Medicine* is a highly readable, excellent presentation of the patient-centered perspective of medical practice and medical law. There are, however, two weaknesses in the book, both of which are inherent in collections of essays. First, short essays inevitably exhibit a "hit and run" character. With only a few pages allotted per topic, Annas typically has only enough space to describe an issue, offer his brief analysis, and move on. Frequently, one is left frustrated by the absence of further development. For example, Professor Annas deplores the fact that in some cases operations are performed not by the surgeon that the patient engaged, but by a resident under the surgeon's supervision. After reading that accusation, one expects some recognition and discussion of the complex problem of clinical education; how best to balance the need to train surgeons with the need to protect and respect patients. But the essay moves on to another topic. (Admittedly, book reviews exhibit the same weakness.)

A second criticism, actually more observation than criticism, concerns the age of some of the essays. While all are insightful and informative, some essays now have the flavor of history rather than current events. For instance, Annas' essay about the original Baby Doe regulations is interesting, but no longer timely since those regulations have been invalidated by the Supreme Court and replaced by different federal regulations. Similarly, Annas' discussion of some of the older life sustaining treatment cases, like *Quinlan* in New Jersey and *Saikewicz* in Massachusetts, is overshadowed by important subsequent court decisions in those states, some of which are addressed in other essays. Of course, the human dilemmas raised by these cases are timeless.

A final observation is in the nature of a warning. While Professor Annas clearly has great respect for physicians and medicine, he sees much wrong with the way health care is delivered. His essays are briefs in support of change, and some will flinch at their adversarial, activist tone. Indeed, in *Judging Medicine*, Annas sometimes seems to take on the role of a civil rights worker circa 1964, with American health care as his Mississippi. He speaks for patients who are thwarted, even oppressed, by doctors and hospitals, lawyers and courts. Correspondingly, readers who practice medicine or administer the system may see Annas as an "outside agitator" who is stirring up trouble, upsetting their basically good relationship with patients.

Today, the civil rights movement has completely vindicated its role: we look back at that cause as one of the clearest examples in US history of right against wrong. It is unlikely that Professor Annas' judgment of medicine, where questions of right and wrong are so complex, will ever secure such complete acceptance. On the other hand, Professor Annas' contribution to many of the progressive developments in medical practice today is clear, and the powerful essays in *Judging Medicine* are the centerpiece of that contribution.

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Haemophilus influenzae type b prevention

Haemophilus influenzae type b (Hib) is a leading cause of serious systemic bacterial illness in the United States. It is the most common cause of bacterial meningitis, with an estimated 12,000 cases occurring annually. Most of these cases are seen in young children under five years of age. Serious Hib disease is most common in children between the ages of six months and one year with attack rates declining thereafter. Approximately 35–40% of the disease occurs in children 18 months of age or older, and 25% occurs among those 24 months of age and older. The mortality rate from the disease is 5% with neurologic sequelae observed in 25–35% of the survivors. Approximately one in every 200 children develops a systemic infection due to Hib by the age of five. *Haemophilus influenzae* type b is also responsible for invasive diseases including epiglottitis, sepsis, cellulitis, septic arthritis, osteomyelitis, pericarditis and pneumonia, in addition to bacterial meningitis.[1]

In 1987, the number of reported Hib cases in New York State was 346. Of these cases, 80 (23%) occurred in New York City; 266 (77%) occurred in the rest of the state. A total of 265 cases (77%) occurred in children five years of age and younger.

Hib Vaccines

In 1985 the Food and Drug Administration (FDA) licensed *Haemophilus* b Polysaccharide Vaccine for routine use in children 24 months of age or older and at 18 months for those in high-risk groups. These high-risk groups include children attending day care centers, those with anatomic or functional asplenia (eg, sickle cell disease or splenectomy) and those with malignancies associated with immunosuppression. This vaccine is composed of purified capsular polysaccharide from *Haemophilus influenzae* type b.[1] A 1974 study in Finland determined that the vaccine was 90% effective in preventing invasive Hib disease in children vaccinated at 18-71 months of age.[2] However, in this study, the efficacy could not be demonstrated in children 18 to 23 months of age because of the small sample size of this age group.[3] In addition, the vaccine was not shown to be effective in preventing the disease in infants, the age group at highest disease risk.[2] The development of a new vaccine was aimed at improving the immunogenicity in infants and younger children. The search led researchers to covalently link the capsular polysaccharide to a protein carrier.[4] The first conjugate vaccine was licensed by the FDA on December 22, 1987, for routine use in children 18 months of age or older and consists of Hib capsular polysaccharide linked to diphtheria toxoid, a protein carrier.[3]

Several studies conducted compared the immunogenicity of both the conjugate and polysaccharide vaccines. The conjugate vaccine was shown to exhibit a greater immunogenic effect than the latter form. A randomized, double-blind study was conducted in the United States in which children 15–24 months of age were given a single dose of either conjugate or polysaccharide vaccine. Over 90% of the children who received the conjugate vaccine responded with antibody levels considered to be protective. This compares to a 50% overall response level in the children vaccinated with the polysaccharide vaccine. In addition to studies concerning immunogenicity, the biological activity of the conjugate vaccine has also been addressed. Several studies have shown a correlation between the functional activity and serum levels of the antibody to the *Haemophilus influenzae* type b polysaccharide and suggest that antibody produced in response to the conjugate vaccine is biologically equivalent to that produced in response to the polysaccharide vaccine.[2]

On December 22, 1988, the FDA licensed an additional conjugate vaccine for routine use in children 18 months and older. The administration of this new vaccine (Diphtheria CRM_{197} Protein Conjugate) follows the recommendations applied to the Diphtheria Toxoid Conjugate.[5] This new conjugate vaccine is composed of oligosaccharides from the Hib capsular antigen directly linked to CRM_{197} protein, a nontoxic derivative of diphtheria toxin. This vaccine was also shown to be more immunogenic than the polysaccharide vaccine. Studies are currently in progress to evaluate the efficacy of this vaccine and other conjugate vaccines in children under 18 months of age. The aim of such research is to target the youngest possible group of children for Hib immunization.[6]

The New York State Department of Health reminds health care providers to educate parents of young children about the potential dangers of Hib disease. Parents should be aware of the availability of vaccine to protect their children from Hib. Conjugate Hib vaccine is available from private health care providers and is provided without charge by local health unit clinics statewide. For further information on Hib prevention, contact the Immunization Program at (518) 473-4437.

Recommendations

The following guidelines for the administration of *Haemophilus influenzae* type b (Hib) conjugate vaccine are endorsed by the New York State Department of Health and are recommendations of the Immunization Practices Advisory Committee (ACIP) and the American Academy of Pediatrics.[3,7]

- All children should receive conjugate vaccine at 18 months of age. For those 24 months of age and older who have not yet been immunized against Hib disease, immunization is recommended through the fifth year of life. For these children, administration of conjugate vaccine is preferred to polysaccharide vaccine because of its greater immunogenicity.

- A special effort should be made to immunize children at high risk of invasive Hib disease. Children at high risk include those attending day care centers, those with anatomic or functional asplenia and those with malignancies associated with immunosuppression.
- Currently, revaccination is not recommended for children receiving conjugate vaccine at 18 months of age or older. Further information is needed concerning the duration of immunity after a single dose of conjugate vaccine.
- Because many children who received the polysaccharide vaccine between 18 and 23 months may have had a less than adequate response to the vaccine, they should be revaccinated with a single dose of conjugate vaccine. Revaccination should take place a minimum of two months after the initial dose of polysaccharide vaccine. There is no need to routinely revaccinate children who received polysaccharide vaccine at 24 months of age or older.
- Children who had invasive Hib disease when they were less than 24 months of age should receive the vaccine according to the above recommendations. Most children younger than 24 months fail to develop adequate immunity following natural infection.
- The conjugate vaccine should not be considered an immunizing agent against diphtheria, although increases in serum diphtheria antitoxin levels can follow administration of conjugate vaccine. No changes in the schedule for administration of diphtheria toxoid (generally DTP) should be made secondary to the use of conjugate vaccine.
- Chemoprophylaxis of household or day care contacts of children with Hib should be directed at vaccinated and unvaccinated contacts. Vaccination with either type of vaccine probably does not inhibit asymptomatic carriage of the organism. Vaccinated children may be protected from invasive disease but may nevertheless transmit the organism to susceptible children.
- Conjugate vaccine and DTP may be given simultaneously at different sites. Data are lacking with respect to concomitant administration of conjugate vaccine and measles-mumps-rubella (MMR) or oral polio (OPV) vaccines. If the recipient is unlikely to return for further vaccination, simultaneous administration (DTP, OPV, MMR and Hib conjugate) is recommended according to the recipient's age and previous vaccination status.

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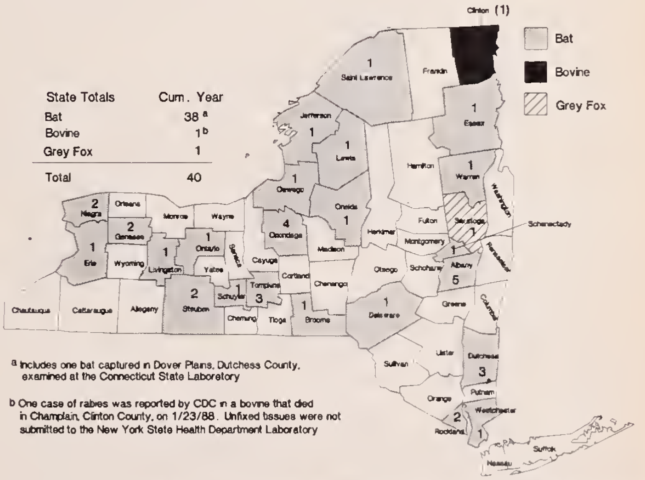
NYS Rabies Summary 1988

During 1988, 40 cases of rabies in animals occurred in New York State (exclusive of New York City) (Figure 1). Bats comprised the largest number of cases (38), followed by one case each in a grey fox and a bovine.

The red fox rabies outbreak that has occurred cyclically in the northern counties for the last three decades was not evident during 1988. This was the first year since 1980 that fox rabies was not confirmed in that area. The outbreak was interrupted on previous occasions for a year or more and recurred on islands of the St Lawrence River or in townships bordering the river.

The mid-Atlantic raccoon rabies epizootic, which currently affects Virginia, West Virginia, Maryland, the District of Columbia, Delaware and Pennsylvania, continued to spread slowly toward New York during 1988. Pennsylvania reported 543 rabid animals, including 385 raccoons for the year. The problem moved only about 25 miles northward this year, and is currently about 45 miles south of Elmira, New York. The eastward spread toward New Jersey was more dramatic, with numerous cases in the Pennsylvania counties bordering that state.

During 1988, the New York State Health Department Rabies Laboratory examined 3,213 animals for evidence of rabies infection (Table I). Bats comprised the largest single category of specimens (1,177), followed by domestic cats (609) and dogs (473). The raccoon was the most commonly submitted animal (309) among terrestrial wildlife. A total of 245 rodents and lagomorphs (rabbits and hares) were submitted for examination despite the fact that in the United States they are relatively rabies-free. Ferrets submitted to the Rabies Laboratory to exclude rabies infection totaled 57, the same number as in 1987. Ninety-one percent of the ferrets were involved in a biting incident.



State Totals	Cum. Year
Bat	38[a]
Bovine	1[b]
Grey Fox	1
Total	40

[a] Includes one bat captured in Dover Plains, Dutchess County, examined at the Connecticut State Laboratory

[b] One case of rabies was reported by CDC in a bovine that died in Champlain, Clinton County, on 1/23/88. Unfixed tissues were not submitted to the New York State Health Department Laboratory

FIGURE 1. Laboratory confirmed rabies in New York State (excluding New York City), 1988.

TABLE I. Animals Submitted to the New York State Rabies Laboratory—1988

Animal	No. Submitted	No. Positive	% Positive
Dogs	473	0	0
Cats	609	0	0
Cattle	50	1	2*
Other Domestic	30	0	0
Skunks	89	0	0
Foxes	127	1	0.79†
Bats	1177	38	3.14‡§
Raccoons	309	0	0
Rodents/Lagomorphs	245	0	0
Ferrets	57	0	0
Other Wild	47	0	0
Total	3213	40	1.24

* Includes 1 bovine reported positive by the Centers for Disease Control after examination of formalin-fixed tissues by an experimental technique.

† 1 Grey Fox, July 14, 1988, Northumberland, Saratoga County.

‡ 1 Myotis keenii, June 19, 1988, Deposit, Delaware County. 36 Big Browns, *Eptesicus fuscus*, March 10-December 2, 1988, from the following counties: Albany (5), Broome (1), Dutchess (2), Erie (1), Essex (1), Genesee (2), Jefferson (1), Lewis (1), Livingston (1), Niagara (2), Oneida (1), Onondaga (4), Ontario (1), Oswego (1), Rockland (2), St. Lawrence (1), Schenectady (1), Schuyler (1), Steuben (2), Tompkins (3), Warren (1), Westchester (1).

§ Includes one rabid bat captured in Dover Plains, Dutchess County, examined by the Connecticut State Laboratory.

Bat rabies was once again widespread geographically, with the 38 confirmed cases occurring in 22 counties from all regions of the state. All but one case occurred in the big brown bat, *Eptesicus fuscus*, one of the two common commensal species. The other case occurred in *Myotis keenii*, a relatively rare species. Overall, 3.2% of submitted bats were rabid, while 5.7% of big brown bats were rabid. A total of 95 (8.7%) of all bats submitted had bitten a human, 452 (38%) had contact with one or more cats. Among rabid bats, 21.8% had bitten a human while 16.2% had contact with one or more cats. Several of the human bites inflicted by rabid bats received widespread media attention this year.

In January, a bovine in Clinton County, suspected of listeria infection, was euthanized and tissues were sent to the New York State College of Veterinary Medicine at Cornell. Formalin-fixed tissues were sent to the US Centers for Disease Control where an experimental avidin-biotin amplified histochemical test was performed and the cow was reported as rabid. Attempts to confirm the result of this test have been unsuccessful.

A rabid grey fox captured in Saratoga County had rabies virus widespread throughout its tissues. The virus isolated from the fox was identified by monoclonal antibody panel analysis at the Wadsworth Center for Laboratories and Research to be the strain commonly found in big brown bats in New York State. This is the 13th occurrence since 1978 of bat-to-terrestrial mammal transmission of rabies.

These cases confirm the need to suspect rabies in an animal exhibiting signs of the disease anywhere in the state, since rabid bats are reported from all areas and do occasionally infect other species. The pattern of reported contacts with bats emphasizes the value of rabies vaccination for pets throughout the state, especially cats. Vaccination should also be considered for valuable livestock housed in buildings with a history of bat infestation. Ferrets must be considered as a wild animal and submitted for rabies examination subsequent to a biting incident of humans.

Cancer incidence in five continents

The fifth volume in the series *Cancer Incidence in Five Continents* has recently been prepared and published jointly by the International Association of Cancer Registries, a nongovernmental body in official relations with the World Health Organization (WHO), and the International Agency for Research on Cancer. The volume contains information on the incidence of cancer as it is distributed throughout many parts of the world and, for some regions, among various ethnic groups. With the increasing homogeneity of the environment in many parts of the world, it is important to cover as much of the globe as possible with a network of cancer registries able to provide reliable, comparable information on risk differentials. Populations covered by this volume vary in size from 68,900 in the Northwest Territory and Yukon of Canada to a population of 49,203,000 in England and Wales. Cancer incidence data for New York State, excluding New York City, is included. Separate data for the population of New York City is included for the first time in Volume V.

The problems involved in comparing incidence data from different parts of the world are discussed in depth in the commentary and data on the indices of reliability are presented for each registry by sex, site and broad age groups. These indices include the percent of cases histologically verified, the percent of cases derived from death certificates only and the ratio of mortality to incidence.

Separate chapters are devoted to Techniques of Registration, Classification, Coding Practices, Processing of Data and Comparability and Quality of Data: Reliability of Registration. Included is a discussion of the variations in definitions of multiple primary neoplasms between registries. Almost all registries count each independent primary separately if these primaries occurred in different organs. However, many registries also count separately independent primaries occurring in the same organ, such as colon, and many registries also count cancers occurring in paired organs twice. The New York State Registry tends to be more conservative in counting multiple primaries than many other registries.

One of the principal objectives of this series is to provide data that will permit comparisons of incidence that may lead to the formulation of etiologic hypotheses. This comparison may be international, inter-regional or secular. The differences observed can result from exposure to a variety of environmental factors, modified by differences in genetic background and cultural patterns. They may also be influenced by purely technical procedures of registration. The book provides no interpretation of the data and leaves the reader to draw conclusions and build hypotheses on the basis of this unique data set.

Data continue to support the effect of changes in lifestyle that occur following migration. This includes but is not limited to changes in dietary habits. Table I shows incidence rates for four types of cancers among Japanese

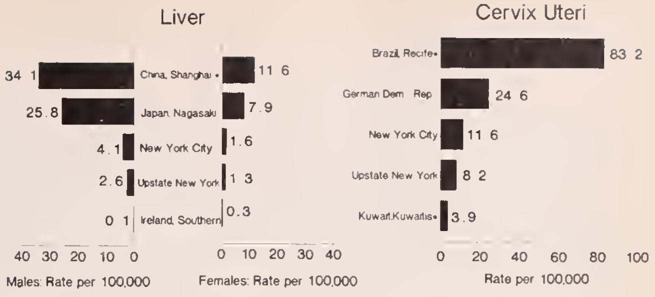
TABLE I. Cancer Incidence Rates per 100,000,* 1978–1982

	Stomach		Colo-rectal	
	Males	Females	Males	Females
Japan (Nagasaki)	82.0	36.1	31.1	17.4
Hawaii (Japanese)	28.4	14.1	52.0	31.1
San Francisco Bay Area (Japanese)	24.3	10.8	43.4	33.2
San Francisco Bay Area (Whites)	10.4	4.8	46.0	34.7
	Prostate		Female Breast	
Japan (Nagasaki)	8.8		20.3	
Hawaii (Japanese)	31.2		50.1	
San Francisco Bay Area (Japanese)	16.5		48.9	
San Francisco Bay Area (Whites)	50.0		87.0	

* Age-adjusted to world population.

living in Japan and those who have migrated to the United States. The rates for certain types of cancer change markedly among Japanese who have migrated to Hawaii or to the San Francisco Bay area. Stomach cancer declines dramatically but not to the low level of whites living in the San Francisco Bay area. Colorectal cancer has increased and now equals or exceeds that of the white population. Marked increases have occurred in the Japanese rates for female breast and prostatic cancer but still do not equal the rates observed for whites.

Figure 1 illustrates some of the differences found between registries. Cancer incidence rates are given for two selected sites (liver and cervix uteri) from several registries worldwide to illustrate differences between New York State and other parts of the world. In each case, the highest and lowest reported rates are included. All incidence rates are age-adjusted to the standard world population. The world population gives more weight to younger ages in the age-adjustment process and, thus, produces somewhat lower rates for New York State than when adjusted to the 1970 United States population. For both liver and cervical cancer the rates for New York City, even with its extremely heterogenous population which includes many migrants, are higher than for the rest of the state. Liver cancer remains elevated among Far Eastern migrants. Cancer of the cervix uteri remains higher in New York City as it is more common in lower socioeconomic groups including black and Hispanic populations.



Cancer in Five Continents Volume V
Age-Adjusted to World Population

• Some data may be incomplete as evidenced by the indices of reliabilty and/or the conditions in which the registry operates

FIGURE 1. Age-adjusted incidence rates, 1978–1982, liver (ICD 9th, 155) and cervix uteri (ICD 9th, 180), selected international cancer registries.

Additional publications are expected to be published based on Volume V. *Cancer Incidence in Five Continents: A Picture Book* will depict data in graphic form and will be published in 1989. Included will be histograms showing the world age-standardized rates for 40 representative populations in rank order by site and sex, and age-specific rate graphs for selected sites and populations. *Cancer Incidence in Five Continents, Volume V* is distributed by Oxford University Press, New York, and may be obtained through local bookstores. For additional information contact: Dr William S. Burnett, Director, New York State Cancer Registry, (518) 474-2255.

GENERAL SURGERY: REVIEW AND ASSESSMENT

Third Edition. By Richard M. Stillman, MD. 438 pp, illustrated. East Norwalk, Conn, Appleton & Lange, 1988. $24.95 (paperback)

This is the third edition of a softcover book first published in 1983 to help students and residents develop and maintain the "mental data bank" required to care for surgical patients and to pass the NMB, Flex, ECFMG, and In Training, Qualifying and Certifying examinations of the American Board of Surgery.

The book covers general surgery as well as some of the subspecialties such as pediatric surgery, peripheral vascular surgery, cardiovascular surgery, and anesthesia. Because of its small size, it can easily fit in a coat pocket, and its outline format makes information readily accessible.

Each chapter begins with a concise and to-the-point outline of anatomy followed by a discussion of pathophysiology, diagnosis, clinical findings, treatment, and complications. Numerous tables, illustrations, and mnemonics are used to facilitate the memorization process. A list of multiple choice questions and answers designed to aid the reader in self-examination completes each chapter.

In this new edition, the author has added a chapter on the diagnosis and management of the acute abdomen and revised the chapters on fluid and electrolytes, shock, and thyroid. His attempt to keep pace with the recent advances in various surgical fields has, however, been only partially successful. The book contains some statements that are either outdated or not widely accepted. For example: "Paget's disease of the nipple has a very poor prognosis," (page 53); "The treatment of Zollinger-Ellison: Cimetidine for two weeks then total gastrectomy," (page 163); "Familial polyposis Rx.: total colectomy and ileorectal anastomosis and F/U of rectum," (page 195).

Although the author indicates that the book will "expose areas that the reader does not fully comprehend," the student will quickly realize that he has to consult the selected bibliography quite often, for the book touches only lightly on the detailed treatment of many clinical entities. For example: "The RX of primary aldosteronism is: spironolactone and potassium to correct metabolic abnormalities. Operation." The author does not indicate which modality to use if the primary aldosteronism is due to an adenoma or hyperplasia, nor the type of operation required.

The treatment of melanoma is based on Clark's histologic classification without any mention of Breslow's classification. The treatment of ulcerative colitis fails to indicate total colectomy with submucous resection of rectum and ileoanal anastomosis as an alternative.

Overall, the special value of this book lies in its format: an outline text and a question-answer section. Medical students will find it very useful as an overview and as a reference guide during their clinical rotations, as well as a valuable study aid for the surgical part of the National Board Examinations. The house officer may also find it useful in preparing for the in-training examination, assuming a previously acquired data base is adequate.

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SURGICAL INFECTIOUS DISEASES

Second Edition. Edited by Richard J. Howard, MD, Richard L. Simmons, MD. 913 pp, illustrated. East Norwalk, Conn, Appleton & Lange, 1988. $159.00 (hardcover)

Although there are many excellent infectious disease texts written by internists, there are few currently available texts which are written by surgeons and which deal with surgical infections. The purpose of this text is to address the clinical management of those types of infections with which the surgeon deals: polymicrobial infections in hosts who have compromised resistance to these organisms. Until *Surgical Infectious Diseases*, no such text had been published since 1949, when Frank Meleney published *Clinical Aspects of Treatment of Surgical Infections*. Since 1949 much has been learned about humoral immunity, cellular immunity, as well as phagocytic cells and the complement system. Additionally, a broad array of antimicrobial agents are available to the surgical practitioner, as well as new surgical approaches to the treatment of certain infections. This text is a comprehensive review of the pathogenesis, diagnosis, and treatment of surgical infectious diseases, which is intended to serve both the senior surgeon and the surgical resident.

The book has divided the understanding of surgical infections into eight main categories. The first section deals with the microbiology of surgical infections. Part II is an overview of host resistance to infection. Part III, entitled "The Systemic Responses to Infection," provides a review of the metabolic response to injury. Part IV, "Principles of Diagnosis," addresses the role of the ancillary laboratories in the diagnosis of infections. Part V, "Antimicrobial Therapy," discusses not only standard antibiotic therapies but also immunotherapy as well. Part VI, "Wound Infections," provides in-depth discussions on antibiotic prophylaxis, epidemiology of wound infections, technical management of wounds, and nosocomial infections. Part VII is a survey of infections categorized by the region of the body. Part VIII covers special problems such as tetanus, burns, and infections in the immunocompromised host.

The editors are both recognized authorities in the field of infection, and leaders in the area of resistance to infection in the surgical hosts. They have gathered together an impressive number of authorities in their respective fields to write specific chapters. This provides for individual authoritative reviews.

This is the second edition of this text. Since the first edition was published, many new antibiotics have been placed on the market and some new infectious diseases have appeared, including Lyme disease and human immunodeficiency virus infections. Although some chapters were omitted in the second edition, the loss of these chapters has little impact on the overall excellent quality of the text. Many handbooks of surgical infection have been published since the first edition of *Surgical Infectious Diseases*. However, these newer books lack the breadth and depth of Howard and Simmons' book.

In summary, this is a beautifully presented book which provides the only complete textbook of surgical infection available on the market. It is comprehensive and authoritative and is the first text I would go to if I were faced with a difficult surgical infectious disease problem.

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CLINICAL IMPERATIVES IN SURGERY AND ORTHOPAEDICS

By B. G. P. Lindeque. 450 pp, illustrated. Pretoria, South Africa, Haum Educational Publishers, 1988. $67.00 (hardcover)

The authors and contributors have performed a valuable service in writing this concise compendium of orthopedics and the general surgical and basic science foundation of musculoskeletal diseases and their treatment. Perhaps written with house officers and those reviewing for certifying examinations in mind, the 49 chapters have excellent graphics and consistency of form, with a clarity useful to the practitioner as well.

Although the longest chapter, on the subject of endocrinology in surgery and anesthesia, is only 20 pages, it is well written and contains much authoritative information, particularly on the management of hypo- and hyperthyroidism and the diabetic patient. There is no mention of inappropriate antidiuretic hormone states, or the interrelationship of calcium metabolism with renal function, although hypercalcemic and hypocalcemic states are dealt with in the text.

Almost a quarter of the book is devoted to the spinal area. Excellent diagrams augment the lucid discussion of the spine examination, injury, deformity, developmental and acquired disease, including bracing, and surgical and neurological aspects. Growth and development, hematologic aspects, immunology, and the management of shock and the acutely injured patient, are sections that deserve particular praise. There is clarity and completeness in these areas. Biomechanics, prosthetics, and orthotics, however, are given short shrift. Hand and amputations receive only scant and therefore wanting coverage.

Another admirable feature of this 400-page text is the excellent bibliographies at the end of each chapter and frequent references in the book to detailed classics when more information is sought by the reader.

The included material has continuity, making the book easily readable. Credit is due to the senior editor and principal contributor, G. F. Dommisse, for the fluidity and uniformity of the text which emanates principally from the University of Pretoria. The University of Liverpool is also represented. Four American authors who are well known and widely published in their fields are also contributors.

The title of *Clinical Imperatives in Surgery and Orthopedics* is apt. Much of the information is invaluable, "must know" material. A certain amount of repetition of important facts through some of the chapters is actually an advantage in recalling some of the pearls of orthopedic wisdom. It is far superior to many handbooks, outlines, and compendiums of recent vintage aimed in the same direction. The publisher, Haum Education Publishers, deserves applause for excellence in printing, paper quality, and binding as well as the moderate price.

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OCULOPLASTIC, ORBITAL AND RECONSTRUCTIVE SURGERY

Volume 1. Edited by Albert Hornblass, MD. 717 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1988. $125.00 (hardcover)

The purpose of this textbook is to offer extensive information on the subject of oculoplastic surgery with an adequate reference list for each topic. The first volume of the well-written text covers eyelid surgery; the orbit and the lacrimal system will be covered in the second volume. All of the 92 authors are recognized authorities in their fields including a large number of ophthalmologists, plastic surgeons, dermatologists, and others. Seventy-six subjects are discussed in nine sections and most chapters have good black and white photographs and clear illustrations.

Section I discusses the anatomy and physiology of the eyelids and includes general principles of oculoplastic surgery, such as specialized instruments, wound healing, type and application of anesthesia, and specific surgical techniques. This section ends with a review of psychological and medicolegal issues, areas not always well addressed by non-ophthalmological surgeons. The congenital and developmental anomalies, including congenital ptosis, is well covered in section II. The discussions of diseases of the eyelid, including information on connective tissue diseases, are interesting in section III and are not usually available in oculoplastic literature. Section IV deals with neoplastic lesions of the eyelids and adnexa, including different types of treatment. Eyelash and eyebrow problems are reviewed in section V, and in section VI ectropion and entropion as well as acquired ptosis and the different repairs available are discussed. The treatment of lagophthalmos with springs and silastic rods, nerve anastomosis, and fascia lata slings is mentioned. Telecanthus, post-traumatic and in relation to blepharophimosis syndrome and blepharospasm, is also reviewed in this section.

The principles of eyelid trauma are well discussed in section VII. This section covers chemical and thermal burns as well as trauma to the lacrimal system. It is difficult to cover completely the diversity of eyelid trauma in 50 pages. Approximately 20% of the volume is dedicated to cosmetic surgery of the eyelid (five chapters). Many different techniques are described including the transconjunctival approach of herniated periorbital fat of the lower eyelids. Especially well written chapters are the ones on lower blepharoplasties and on the importance of the balance between levator and the orbicularis muscle. The last section deals with eyelid reconstruction including repair of a variety of lid and canthal defects. Removal of basal cell carcinoma with the Moh technique and the argon laser treatment of port wine stains and other blood vessel tumors are also included in this section.

There is a need for this type of encyclopedic textbook. The field of oculoplastic surgery has grown tremendously in the last 20 years, and several surgical specialties share interest in this anatomical area. Volume I is one of the first complete textbooks of eyelid disease and eyelid surgery, and it is very valuable for anyone who operates in that area. It is especially useful to the aesthetic plastic surgeon requiring an introduction to multiple disease manifestations affecting the eyelid and the orbit.

In summary, Volume I of *Oculoplastic, Orbital and Reconstructive Surgery* provides the reader with a well-organized, encyclopedic textbook of eyelid disorders and ophthalmic plastic surgery technique. I would imagine that Volume II dealing with the orbit and lacrimal system will be of interest to a lesser number of surgeons. However, I am looking forward to its publication.

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ATLAS OF CO_2 LASER SURGICAL TECHNIQUES

Edited by Raymond J. Lanzafame, MD, and J. Raymond Hinshaw, MD. 297 pp, illustrated. St Louis, Ishiyaku Euro-America, Inc, Publishers, 1988. $145.00 (hardcover)

This is a well-constructed and well-illustrated book on CO_2 laser surgery by the capable laser surgical group of the Rochester General Hospital. It is a welcome addition to the increasing group of color atlases for this form of treatment. These colorful photographic aids show surgical techniques almost as well as the expensive color VHS tapes. This atlas is superior to the usual textbook on the subject. Laser physics is explained in many easily understood color prints and comparable tissue ablation pictures are also available illustrating the value of controls. The section on laser safety is also well illustrated. The section on laser nursing is excellent, especially for anyone designated as the laser safety officer. For the postoperative laser gynecologic patient, nursing is important. A laser trained nurse can be more understanding and responsive to the patient. In the instrument section, the Sharplan CO_2 laser and its accessories are shown in detail; no other lasers are shown. The clinical treatment sections start with cutaneous and subcutaneous benign and malignant tumors and infections. Definite indications for laser surgery are shown for malignant and infected lesions. Occasionally, there are follow-up pictures. Laser surgery of the breast is presented, as is laser abdominal surgery. The latter is uncommon so far in the US, but extensive in the USSR, China, and Japan. Laser abdominal surgery includes the pancreas and the gall bladder. The authors admit that hepatic surgery is controversial but hepatic surgery in dogs has been shown to be effective. An interesting case of an echinococcus cyst of the liver is presented and head and neck surgery as well as rectal surgery are also covered. Similar to laser surgery of decubiti, laser surgery of gangrene is presented. Orthopedic laser surgery of rheumatoid arthritis and hemophilic arthritis of the knee are pictured. In Europe, there is extensive use of orthopedic laser surgery in hemophilia. Also included is a laser liver biopsy in a patient with hemophilia with suspected Laennec cirrhosis. A selected laser surgery reading list and a good glossary conclude the textbook. A table of contents probably substitutes for an index. This book is a beautiful and colorful practical guide for many areas of current CO_2 laser surgery.

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ATLAS OF SURGICAL OPERATIONS

Sixth Edition. By Robert M. Zollinger, Jr, MD, and Robert M. Zollinger, MD. 273 pp, illustrated. New York, Macmillan Publishing Co, 1988. $120.00 (hardcover)

This is the sixth edition of this now classic atlas by Zollinger and Zollinger. The authors' stated theme "to review the anatomy in technical steps of major surgical procedures in order to avoid problems from unexpected anatomic anomalies or pathologic processes" is well fulfilled. To this new edition the authors have added 14 surgical procedures with 20 new plates, including a section on stapling devices, modified radical mastectomy, Shouldice herniorrhaphy, ileoanal anastomosis, saphenous vein in situ arterial bypass, and parathyroidectomy. A new chapter covering ambulatory surgery has also been added. The book's contents and organization are basically unchanged from previous editions and thoroughly detail the anatomy and technical considerations of numerous general surgical, vascular, and gynecologic procedures. Each plate is organized with respect to indications, preoperative preparation, anesthesia, positioning, operative preparation, incision and exposure, and details of the specific procedure, including closure and postoperative care. The anatomical graphics of the operative procedures are meticulously drawn and correspond sequentially to the descriptive text. Fine points of surgical techniques are clearly pointed out, both in the text and the graphic displays. The sixth edition of the *Atlas of Surgical Operations* by Zollinger and Zollinger represents a significant contribution to the field of surgery and remains a classic surgical text which will enrich the education process for generations of surgeons to come.

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CLINICAL PROBLEMS, INJURIES AND COMPLICATIONS OF GYNECOLOGIC SURGERY

Second Edition. Edited by David H. Nichols, MD. 332 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1988 (hardcover)

The second edition of this book follows the format of the first. The topics are introduced in individual chapters and reinforced by one or two case abstracts. The author uses the abstract to open the discussion, and moves to various treatment options, often with comments on the relative value of each. Most chapters close with pertinent references. Dr Nichols has selected an extremely talented group of contributors to address an extraordinary range of topics. The number of chapters has been increased to 97, and 25 authors have been added. The chapters are grouped into ten sections, focusing on such issues as intestinal injuries, urinary tract injuries, fertility surgery, and vaginal surgery. The section on pelvic cancer surgery, with 17 chapters, is new to this edition, including such diverse problems as trauma to the vena cava during para-aortic node dissection and unexpected carcinoma of the fallopian tube at vaginal hysterectomy. The section on breast surgery includes chapters on the solitary breast nodule and pneumothorax during mastectomy. These are examples of the unusual range and diversity of the problems addressed.

This is not a book which lends itself to reading in continuity, but rather to selective reading, somewhat analogous to a volume of essays or short stories. It will also serve as a quick reference for those clinical problems, complications, and injuries included. The discussions are, for the most part, of very high quality. The depth of discussion varies, depending on the topic. A chapter on the missing needle would, for instance, require less elaboration than that covering accidental cystotomy during vaginal hysterectomy. Doctor Nichols, reflecting his interest, has included 18 chapters in the section on vaginal surgery alone, and several other complications of vaginal surgery in the section devoted to urinary tract problems and injuries. These are uniformly excellent.

This is a very useful book appealing to attending physicians and house officers. It provides a quick review of a wide variety of clinical problems in gynecology. While many of the complications are rare and may never be encountered, their inclusion adds to the book's value.

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FROM THE LIBRARY

REPORT ON THE IMPROVEMENTS IN THE ART AND SCIENCE OF SURGERY IN THE LAST FIFTY YEARS

... I will here introduce my opinions as to the improvement in surgery by the use of anaesthetic agents, as applicable to different cases in which we are called on to operate. I shall not enter into the discussion of their discovery or who first used them, but of their value to the surgeon, and who should use them, and under what circumstances they can be employed with benefit. In the use of chloroform, the most common agent, I hold it to be an established fact, that it is always hazardous to give it to its fullest extent, for to say the least, it is giving, *unnecessarily*, two chances for death to the patient, the one by the knife of the surgeon, the other by the anaesthetic agent. Hundreds of cases have occurred in which death or permanent insanity has been the result of its use. Consult CHURCHILL on the use of it in obstetrics, and his confession should be a warning to every woman who has the happiness of becoming a mother; we would exhort her to give birth to her child as she got it: in the natural way.

In surgery, men do not often confess their sins, and when they do it is to a priest whose obligations compel him to secrecy. Although I have never been so unfortunate as to kill any one with chloroform, still I must confess that in the early use of it, and in the absence of experience, I have in a few instances come very near destroying my patients, by a total suspension of life and by convulsions, the result doubtless of its use; and I do know that my neighbors have killed some of their patients outright. Had I done so in one instance I should have been mobbed, because I did not belong to the church, or because I should have been charged with experimenting on a Catholic Irishwoman, in using chloroform for the trifling operation of opening a felon. Anaesthetic agents certainly produce insensibility by compression, or an apoplectic condition of the brain, and under such circumstances, the rupture of a vessel in the ventricles or soft parts of the brain is death. That this is the condition of the patient, I have satisfied myself both by experiments on animals, and by surgical operations on the human body, some of which I shall never perform again with the aid of chloroform, with my present experience of its effects. In all my experiments with chloroform on animals, they sink into insensibility as the pupils of the eyes dilate, or the brain is overcharged with blood, and die if it is long continued. ...

... I have determined never to give chloroform in any operation but when diluted with sulphuric ether, and then never to its fullest extent; but if my patients will have an anaesthetic agent, will give them as much good whiskey as they will drink, which, after all the boasting of the advocates of chloroform and ether, is the very best agent of the kind that can be used, and answers all the purposes of the others without the dangers attending their use.

I am advised of the preponderance of professional opinions on this subject in favor of the use of chloroform, but notwithstanding, cannot hesitate to give my honest convictions, and were I an autocrat in surgery I would banish from my dominions all anaesthetic agents but opium and alcohol. Chloroform is too indiscriminately used, and by persons unfitted by the absence of physiological knowledge to judge correctly of the propriety of its use. Every quack boasts of his skill in administering it, and its perfect safety in all cases, while the most ignorant tooth carpenter administers it freely even in his most simple operations, and which cause but little pain to his patient. I have in almost every instance extirpated the parotid gland without chloroform, and in the case of Mrs. Charles North, of Johnson County, Mo., extracted a tumor as large as an infant's head from the upper jaw, extending to the base of the skull, and carrying with it nearly all the upper teeth, successfully, without the use of chloroform, and I cannot again be persuaded to administer it in such cases, for I am convinced that my patients bleed less and recover sooner without the use of this agent in surgery. The patient is safer in the hands of the skillful anatomist and surgeon, with nothing in his reach but the knife, the tenaculum and the sponge. ...

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(*Trans AMA* 1860; 13:425–466)

Survey assesses AIDS on college campuses

A nationwide survey of blood samples from students at 19 campuses confirmed the presence of infection with the AIDS virus among college students (*NY Times*, May 23, 1989, p C12). The American College Health Association, which performed the survey, reported that "the infection rate was slightly higher than scientists had expected but below the estimated average rate for the nation."

While estimates of the rate of AIDS infection in the US range from 4/1,000 to 6/1,000, approximately two of every 1,000 students tested showed evidence of infection with human immunodeficiency virus (HIV). Of the 30 HIV antibody-positive samples, 28 were from men, even though a majority of the overall samples were from women. The blood samples tested for HIV were not obtained at random; rather, they had been collected at student health centers for other testing procedures, including cholesterol screening, pregnancy tests, or mononucleosis diagnosis.

In the view of Dr Richard P. Keeling, chief researcher on the study, "Because of a suspected high incidence of sexual and drug activity, including alcohol abuse, among college students, the figures portend a rising problem with AIDS among this age group if strong preventive measures are not taken."

Unpublished research data may obscure important findings

Participants at a recent American Medical Association meeting to evaluate the peer review process voiced their concerns that many researchers and journals have a bias against publishing inconclusive or negative results (*NY Times*, May 16, 1989, p C5). They expressed the fear that decisions concerning treatment protocols could be based on skewed and incomplete information because the results of many clinical studies are never published.

Using a study of the treatment of ovarian cancer as an example of the potential danger of not publishing negative findings, one participant at the meeting described how "while published trials led to the conclusion that one treatment regimen was clearly superior to another, a similar analysis that included unpublished studies did not show clear differences."

One solution might be for funding agencies and research laboratories to register all studies from their inception, thereby assuring that both unpublished and published data are available for scrutiny. Understanding why some clinical studies fail can provide valuable information for clinical application and for shaping future trials.

What's ahead for malpractice premiums?

After several years of skyrocketing malpractice premiums, insurance rates nationwide have stabilized, in general, with more moderate increases and premiums even dropping in some states (*Medical Economics* 1989; 66:52–63). Rate increases have decreased by about half among the physician-owned carriers, which handle nearly 60% of all medical liability insurance. "We'll have another year or two of relative calm, and then we'll see a major upward spiral," predicts the president of the Physician Insurers Association of America.

Various financial and legislative strategies have contributed to the recent stability in premium rates. In New Jersey, rates have not increased for the past three years, an achievement attributed to fewer claims having been filed and the increasing awareness among physicians of malpractice risks and preventive measures. Educational efforts and risk-management programs have raised the "malpractice consciousness" of many physicians.

In New York, legislative accomplishments have imposed strict limits on rate increases. "New York started clamping down on premiums in 1985, after five years of steep increases," with rate hikes of 14% in fiscal year 1985–1986, 9% in 1986–1987, and 9% in 1987–1988. "The law has been extended for three years, with the cap reduced to 5 percent." Tort reform, adopted in 1975, has also been responsible for keeping the lid on malpractice premiums in California. Measures included "a cap of $250,000 on non-economic damages, graduated contingency fees, a collateral-source provision, and periodic payouts."

Competition among insurance carriers helps to lower rates, although companies often offer artificially low teaser rates to attract business, and then raise the premiums.

Insurance industry experts pinpoint underfunding of reserves as a major cause of the recent upward surge of premiums. When claims progressively increased in frequency and severity, the reserve funds rapidly dwindled, leaving insurance carriers with the need rapidly to rebuild the reserves. Hopefully, most rates are now sufficient to handle the current frequency and magnitude of malpractice claims, but only time will tell.

New findings confirm early-warning sign of cancer relapse

For two years a controversy has been brewing over the claims of Dennis Slamon and colleagues at the UCLA School of Medicine that they discovered a genetic signal in tumors that could help predict the prognoses of patients with breast cancer (*Science* 1989; 244:654–655). Slamon and coworkers' initial study involved a relatively small patient population, and some researchers were unable to confirm the findings.

However, the UCLA group has now supported its claims with the results of an expanded study of breast cancer patients and, furthermore, has demonstrated the prognostic capability of this same genetic signal for ovarian cancer, suggesting that a common genetic or biochemical abnormality may have a role in the development of breast and ovarian cancer (*Science* 1989; 244:707–712).

The discovery of this genetic abnormality, which signals the increased likelihood of a relapse, will hopefully lead to better approaches to treating breast and ovarian cancer, by enabling physicians to predict more accurately which patients are at greater risk of a relapse and should receive more aggressive therapy. In addition, this discov-

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NEWS BRIEFS are compiled and written by Vicki Glaser.

ery could serve as the basis for developing new chemotherapeutic agents aimed at preventing cancer recurrence.

In their earlier study, Slamon and colleagues demonstrated amplification of the HER-2/*neu* proto-oncogene in 28% of 189 primary human breast cancers. (A proto-oncogene is a normal gene that is similar in make-up to genetic sequences with known tumorigenic potential.) The authors found that gene amplification—multiple copies of the HER-2/*neu* gene—in breast tumor cells correlated with a more active disease process, evidenced by a shorter time to relapse and a shorter overall survival.

In their more recent, expanded study, Slamon et al reported on 526 patients with breast cancer. Twenty-seven percent of the 345 patients in whom the cancer had spread to the lymph nodes had evidence of HER-2/*neu* amplification. Statistical analysis "showed amplification of the HER-2/*neu* gene to be a significant predictor of both disease-free survival and overall survival for these patients." Of the 181 patients with node-negative disease, 25% had specific gene amplification, but there was no correlation between amplification and disease outcome in this patient population. The authors proposed that because disease recurrence occurred in only 15–25% of the node-negative patients, there may not have been enough relapses to reveal a statistical association. Other recent studies have demonstrated a correlation for node-negative cases.

A reliable prognostic indicator for patients with node-negative breast cancer would greatly aid in treatment planning. Whereas women with node-positive disease generally receive chemotherapy to halt progression of the cancer, women with node-negative disease were commonly spared the risks and adverse side effects associated with chemotherapy because the rate of recurrence was relatively low. However, about 30% of patients with node-negative breast cancer suffer a relapse within five years, according to an article accompanying the Slamon paper. "Clinical trials indicating that chemotherapeutic drugs can reduce this relapse rate led officials of the National Cancer Institute to recommend last year that all node-negative women receive the drugs." A genetic signal for increased risk of recurrence would help physicians target patients for more intensive therapy.

Slamon et al screened DNA from other human malignancies to search for the same or a similar genetic sequence as is found in the HER-2/*neu* gene. They found significant gene amplification only in samples of ovarian cancer. This finding prompted a study of a greater number of ovarian cancer samples. The results supported a correlation between gene amplification and survival for ovarian cancer.

The striking similarities between breast and ovarian cancer add to the importance of this finding and lead to the theory that similar or the same cellular abnormalities may play a role in the initiation of both diseases. Both breast and ovarian cancer are of epithelial rather than stromal origin, and both tumor types contain cells with estrogen receptors. In addition, women with either ovarian or breast cancer are at increased risk of developing the other type of cancer. HER-2/*neu* gene amplification occurred in 25–30% of the breast and ovarian tumor samples.

Studies in animals and cultured cells have suggested that amplification of the HER-2/*neu* gene and overproduction of its protein product can cause malignancy. One theory holds that this protein is a growth factor receptor—as has been found with other known oncogenes—and that it may cause tumor cells to grow more rapidly and infiltrate surrounding tissue aggressively.

Slamon et al concluded that "this oncogene should be a focus of attention for diagnosis and therapy in human breast cancer; our current findings would extend the idea to ovarian cancer."

Possible role of TNF in nutritional disorders

Based on evidence to suggest that tumor necrosis factor (TNF) has a role in nutritional status, Chollet-Martin et al postulated a relationship between TNF levels and obesity (*Ann Intern Med* 1989; 110:666).

Previous studies have demonstrated that recombinant human TNF leads to reduced food intake and body weight in rodents and produces anorexia and increased whole body lipolysis and fat utilization in humans when administered as antineoplastic chemotherapy.

The authors measured plasma TNF levels in 16 obese subjects, whose body weight exceeded their ideal weight by more than 135%. Comparable measurements were performed on plasma obtained the same day from 16 age- and sex-matched control subjects of normal weight.

TNF values, determined by radioimmunoassay, "were below the detection limit in 13 of the 16 plasma samples from the obese group compared with only 5 of 16 in the control group ($p < 0.01$)." The authors proposed several ways in which TNF could be related to obesity, including modified adipocyte lipoprotein lipase activity in obese persons. Given the antineoplastic capability of TNF, reduced plasma levels of TNF could be implicated in the increased risk of certain types of cancer in obese persons. Furthermore, TNF's beneficial effect on the outcome of some infectious diseases suggests a relationship between TNF deficiency and the heightened susceptibility of obese persons to infection.

Heimlich maneuver successful for clearing esophageal obstruction

The Heimlich maneuver, an effective noninvasive means of removing an object that is obstructing the airway, may also be a safe, effective method for relieving esophageal obstructions. A physician recently reported his experience using the Heimlich maneuver to dislodge a candy from the esophagus of his eight-year-old daughter (*N Engl J Med* 1989; 320:1016).

"She came to me retching, but neither coughing nor in respiratory distress. Her speech was clear, and she described a pain to the right of her cricothyroid region. Application of the Heimlich maneuver while the girl was retching produced an immedicate projectile expulsion of the candy, seemingly by raising the intraesophageal pressure. "Presumably, this pressure was transferred as a result of increased intraabdominal and perhaps intrathoracic pressure as the diaphragm elevated." Hopefully the Heimlich maneuver can be used effectively in future cases of upper esophageal obstruction.

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Daniel K. Adler, MD, Syracuse. Died March 19, 1989; age 75. Dr Adler was a 1939 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a Diplomate of the American Board of Internal Medicine, a Fellow of the American College of Cardiology, and a Fellow of the American College of Physicians. He was a member of the New York Academy of Medicine, the Onondaga County Medical Society, and the Medical Society of the State of New York.

Edwin Newman Beery, MD, New York. Died April 17, 1989; age 79. Dr Beery was a 1934 graduate of Columbia University College of Physicians and Surgeons. He was a Diplomate of the American Board of Ophthalmology and a Fellow of the American College of Surgeons. His memberships included the American Academy of Ophthalmology and Otolaryngology, the American Academy of Compensation Medicine, Inc, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Paul Chester Clark, MD, Fayetteville. Died April 9, 1989; age 84. Dr Clark was a 1930 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a Diplomate of the American Board of Internal Medicine and a Fellow of the American College of Physicians. His memberships included the New York Academy of Medicine, the New York State Society of Internal Medicine, the American Society of Internal Medicine, the Onondaga County Medical Society, and the Medical Society of the State of New York.

Edwin Leopold Demuth, MD, Scarsdale. Died April 20, 1989; age 81. Dr Demuth was a 1938 graduate of the University of Michigan Medical School, Ann Arbor. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the Association for Research in Nervous and Mental Disease, the American Academy of Neurology, the American Psychiatric Association, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Harold Lloyd Ellis, MD, New York. Died April 18, 1989; age 95. Dr Ellis was a 1920 graduate of the McGill University Faculty of Medicine, Montreal, Quebec, Canada. He was a Diplomate of the American Board of Psychiatry and Neurology and a Fellow of the American Psychiatric Association. His memberships included the New York Academy of Medicine, the American Psychiatric Association, the American Academy of Neurology, the New York County Medical Society, and the Medical Society of the State of New York.

Otto Alois Faust, MD, Rochester. Died April 5, 1989; age 101. Dr Faust was a 1915 graduate of the Johns Hopkins University School of Medicine, Baltimore, Maryland. He was a Diplomate of the American Board of Pediatrics and a Fellow of the American Academy of Pediatrics. He was a member of the Medical Society of the County of Albany and the Medical Society of the State of New York.

Philip L. Forster, MD, Albany. Died March 19, 1989; age 90. Dr Forster was a 1925 graduate of the Albany Medical College of Union University. He was a Diplomate of the American Board of Orthopedic Surgery, a Fellow of the American College of Surgeons, a Qualified Fellow of the International College of Surgeons, and a Fellow of the American College of Angiology. His memberships included the New York Academy of Medicine, the American Academy of Orthopaedic Surgeons, the Pan American Medical Association, the American Occupational Medical Association, the American Academy of Compensation Medicine, Inc, the Medical Society of the County of Albany, and the Medical Society of the State of New York.

William M. Glantz, MD, Boca Raton, Florida. Died April 22, 1989; age 77. Dr Glantz held Registrable Qualification Granted by the Scottish Conjoint Board in 1939. He was a member of the International Academy of Proctology, the Bronx County Medical Society, and the Medical Society of the State of New York.

Arnold Grunfeld, MD, New York. Died February 15, 1989; age 88. Dr Grunfeld was a 1924 graduate of the Universitaet Wien, Medizinische Fakultaet, Wien, Austria. He was a Diplomate of the American Board of Radiology and a Fellow of the American College of Radiology. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Alfred A. Imperato, MD, Wantagh. Died April 3, 1989; age 86. Dr Imperato was a 1930 graduate of the Cornell University Medical College. He was a member of the American Geriatrics Society, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Louis T. Imperato, MD, Brooklyn. Died April 23, 1989; age 62. Dr Imperato was a 1951 graduate of the State University of New York Health Science Center at Brooklyn. He was a Diplomate of the American Board of Internal Medicine. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Alexander Kleinman, MD, Bronxville. Died December 10, 1988; age 75. Dr Kleinman was a 1942 graduate of the University of Arkansas School of Medicine, Little Rock. He was a Fellow of the American College of Allergists. His memberships included the New York Cardiological Society, the American Academy of Allergy, the American Academy of Family Practice, the American Association for Cancer Research, the Bronx County Medical Society, and the Medical Society of the State of New York.

Joel Liebermann, MD, Middle Village. Died March 2, 1989; age 77. Dr Liebermann was a 1939 graduate of the Facolta di Medicina e Chirurgia dell'Universita di Bologna, Bologna, Italy. He was a member of the American Academy of Family Physicians, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Mack Lipkin, MD, Chapel Hill, North Carolina. Died April 4, 1989; age 82. Dr Lipkin was a 1930 graduate of the Cornell University Medical College. He was a Diplomate of the American Board of Internal Medicine and a Fellow of the American College of Physicians. His memberships included the New York Academy of Medicine, the Academy of Psychosomatic Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Charles Balthasar Livoti, MD, Holliswood. Died March 18, 1989; age 75. Dr Livoti was a 1939 graduate of the St Louis University School of Medicine, St Louis, Missouri. He was a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Arthur J. Mannix, Jr, MD, New Rochelle. Died April 27, 1989; age 67. Dr Mannix was a 1946 graduate of the State University of New York Health Science Center at Brooklyn. He was a Fellow of the American College of Surgeons. His memberships included the New York Academy of Medicine, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Myron A. Sallick, MD, New York. Died April 16, 1989; age 88. Dr Sallick was a 1924 graduate of Yale University School of Medicine, New Haven, Connecticut. He was a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons. He was a member of the New York Academy of Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Rosine M. Silberer, MD, Palm Beach Shores, Florida. Died April 10, 1989; age 82. Dr Silberer was a 1934 graduate of the Universitaet Wien, Medizinische Fakultaet, Wien, Austria. She was a member of the Broome County Medical Society and the Medical Society of the State of New York.

Angelo Ferrara, MD, PhD
1931–1988

Dr Angelo Ferrara, Medical Director of the New York City Infant Transport Service and Director of Neonatology at New York University-Bellevue Hospital, and a peer reviewer for the *Journal*, died October 15, 1988. He was 57 years old.

Dr Ferrara was an internationally respected neonatologist who made major research and service contributions in the areas of the sick newborn and high risk neonates. Born in New York City on July 15, 1931, he graduated from City College of the City University of New York with a BS degree in 1953 and received his MD degree from the University of Rome in 1958. He did a rotating internship at St Vincent's Hospital and Medical Center from 1958 to 1959, followed by two years of residency training in pediatrics at the same institution. Following this he served as a fellow in allergy at Roosevelt Hospital in New York City.

Combining an interest in pediatric allergy and general pediatrics, he was associated with a number of hospitals in New York City following his fellowship training. In 1971, he became Medical Director of the New York City Infant Transport Service which soon became a model for other such services around the country. In 1982, he became director of neonatology at New York University-Bellevue Hospital, and continued to direct the Infant Transport Service.

In 1977, Dr Ferrara received a PhD degree from New York University's Graduate School of Public Administration. The title of his doctoral dissertation was *Causal Model of an Urban* (*New York City*) *Emergency Neonatal Transport System.* An extremely productive scholar, he co-authored with Sophie Pierog, MD, several editions of a standard text, *Medical Care of the Sick Newborn.* He also co-authored several other books, including *Emergency Transfer of High Risk Neonates* (1980) and *Neonatal Protocols for Emergency Transport and Neonatal Intensive Care Unit Care* (1985). He was a major contributor to medical journals and texts, having published some 40 papers.

While devoting much time to neonatology, Dr Ferrara also continued to be active in pediatric allergy. He served as Director of the Pediatric Allergy Clinics at Bellevue Hospital beginning in 1971. He was a Diplomate of the American Board of Pediatrics and of the American Board of Pediatric Allergy and a Fellow of the American Academy of Pediatrics and of the American Academy of Allergy. He was also Professor of Pediatrics at New York University School of Medicine and Attending Physician in Pediatrics at St Vincent's Hospital and Medical Center.

Dr Ferrara was a dedicated and caring physician and a meticulous scholar with a broad range of interests. His warm personality, dedication, and sense of concern were a source of comfort to both patients and their families. His high standards of scientific excellence won him the respect and admiration of his colleagues. The courage he showed during the final months of his illness were an inspiration to all who knew him.

Dr Ferrara is survived by his wife, Joyce, and by his five children, Lisa Ciardi, Angela, Christine, Joseph, and Carl.

PASCAL JAMES IMPERATO, MD
Editor

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1988; 88:456-462). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith[2] reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al.* Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83:925-927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics*, ed 12. Philadelphia, WB Saunders Co, 1983, pp 337-338.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Pascal James Imperato, MD, Editor, *New York State Journal of Medicine*, PO Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

Medical Society of the State of New York

ANNUAL CME ASSEMBLY

CASE MANAGEMENT & CLINICAL CARE DILEMMAS

September 15–16, 1989

The New York Hilton
Avenue of the Americas at 53rd/54th Streets
New York City

HIGHLIGHTS . . .

24 Sessions — Panel Discussions
Technical Exhibits — Hospitality Functions

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☐ 2-4 p.m. ☐ 4-6 p.m. ☐ 6-8 p.m. ☐ after 8 p.m.

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CONTINUING MEDICAL EDUCATION ASSEMBLY
MEDICAL SOCIETY OF THE STATE OF NEW YORK

FRIDAY, SEPT. 15–SATURDAY, SEPT. 16, 1989

Single:	$150	$165	$180
Double/Twin:	$175	$190	$210
Triple:	Additional $25		
EXECUTIVE TOWER:			
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Rates subject to 8¼% N.Y. State sales tax; $2.00 per room per night, and 5% N.Y.C. occupancy taxes. (If room at the rate requested is unavailable, one at the nearest available rate will be reserved.)

GENERAL INFORMATION

Don't miss this opportunity to gain Category 1 credits over a weekend when you are free from office hours. Bring your family to New York City to enjoy the many sights.

All physicians, members of the allied professions, and their guests are invited to attend the Medical Society of the State of New York's Annual CME Assembly to be held September 15–16, 1989, at the New York Hilton. The two days of meetings will provide the individual physician with a possible 14 credits out of a total of 68 Category 1 CME credit hours being offered. The Medical Society of the State of New York is accredited by the Accreditation Council for Continuing Medical Education to sponsor CME for physicians.

The theme of the CME sessions will be "Case Management and Clinical Care Dilemmas." The chosen subjects are varied and topical, e.g., the battered woman syndrome (Psychiatry)—child abuse (Pediatrics/Forensic Medicine)—cocaine (Substance Abuse)—osteoporosis (American Medical Women's Assoc./Orthopaedic Surgery)—malpractice (Mock Trial, Medical Liability Mutual Insurance Co.) are but a few. . . . Our programs are designed to be interesting and informative.

HOTEL THEATRE DESK

Located in the main lobby of the New York Hilton to accommodate those persons wishing to purchase tickets to any of the various New York theatres.

TECHNICAL EXHIBITS

Technical exhibits include pharmaceutical products, medical and surgical suppliers, health and pension plans, office management systems and more. Representatives from several companies will be in the Rhinelander Gallery Exhibit Hall on Friday and Saturday to introduce you to their products and services.

HOTEL RESERVATIONS

Physicians must make their own reservations with the New York Hilton, no later than August 31, 1989. After that date, rooms are on an availability basis only. If making reservations by telephone, please mention that you are attending the Medical Society Convention for billing at the special rate.

AWARDS TO BE GIVEN AT THE CME ASSEMBLY

Suiter Lectureship presented during the Internal Medicine Program on Saturday, 9/16. Suiter Lectureship is given for a non-surgical subject. The recipient of this award will be announced.

Albert F. R. Andresen Memorial Lecture presented during the Gastroenterology/Colon and Rectal Surgery Program on Friday, 9/15. The Albert F. R. Andresen Memorial Lecture is for a lecture on Gastroenterology. Adrian J. Greenstein, M.D., Professor of Surgery, Mount Sinai School of Medicine, NYC will receive this award.

Lucien Howe Prize presented during the Neurology/Ophthalmology Program on Saturday, 9/16. Lucien Howe Prize is given for the best original contribution to our knowledge of some branch of surgery, preferably ophthalmology. Myles Behrens, M.D. of Columbia University, NYC will be the recipient of this award.

1989
PRELIMINARY PROGRAM
Annual CME Assembly
September 15–16, 1989

The New York Hilton
New York City

INDEX

Hotel Reservation Form 9A
General Information 10A
Scientific Program Committee 13A
Scientific Program 14A
Advance Registration 28A

REGISTRATION & MEETING HOURS

CME Assembly

Registration is open to MSSNY members, spouses, invited guests, non-member physicians, and members of the allied professions. There is a $25 registration fee for MSSNY active members and members of other state medical societies. There is no registration fee for residents/interns, speakers, medical students, medical assistants and spouses. There is a registration fee of $50 for non-member physicians, and a $10 fee for the allied professions.

An advance registration form will be found in this section. For those who register in advance, your identification badge will be held at the CME Registration Desk located in the Rhinelander Gallery Exhibit Hall on the second floor of the hotel. For those who have not registered in advance, registration will take place on Friday, September 15, and Saturday, September 16, from 8:00 a.m. to 5:00 p.m.

All registrants are required to show identification at the registration desk before a badge is issued. Identification badges are required for admittance to all CME Meetings and exhibits.

CME Meetings will be held on Friday, September 15, and Saturday, September 16, from 8:30 a.m. to 5:00 p.m.

CME CREDITS

The Medical Society of the State of New York is accredited by the Accreditation Council for Continuing Medical Education to sponsor CME for physicians. The Medical Society of the State of New York designates all CME activities in this CME Assembly as meeting the criteria for Category 1.

Each program is acceptable for prescribed or elective credits by the American Academy of Family Physicians (AAFP) as designated. The Medical Society's CME credit forms will be issued at the entrance to each meeting room at the opening of each session.

EXHIBITS

Technical exhibits will be located in the Rhinelander Gallery on the second floor of the New York Hilton. Exhibits will be on view Friday, September 15, from 9:00 a.m. to 6:00 p.m. and Saturday, September 16, from 9:00 a.m. to 6:00 p.m.

PHYSICIAN MESSAGE CENTER

Messages for the physicians attending the CME sessions may be left at the MSSNY Information Desk located in the Exhibit Hall on the second floor. Telephone: (212) 865-7000.

HOSPITALITY

FRIDAY, SEPTEMBER 15

Coffee service in exhibit area throughout the meeting days.

Morning and afternoon coffee breaks to foster contact between attendees and exhibitors.

5:00 p.m.–6:30 p.m.: Medical Society of the State of New York to host Cocktail/Reception in Exhibit Hall for attendees and exhibitors.

SATURDAY, SEPTEMBER 16

One hour Continental Breakfast in Rhinelander Gallery for attendees and exhibitors.

Medical Society of the State of New York to host a buffet luncheon in Rhinelander Gallery for attendees and exhibitors.

Periodic prize drawings through the two-day meetings.

Visit the Exhibits

1989 ANNUAL CME ASSEMBLY

Medical Society of the State of New York
The New York Hilton, New York City
September 15–16, 1989

CASE MANAGEMENT AND CLINICAL CARE DILEMMAS

FRIDAY, SEPTEMBER 15

8:30 a.m.–9:30 a.m.	Medical Technology, *Nassau A*
9:30 a.m.–12:30 p.m.	Gastroenterology/Colon & Rectal Surgery, *Sutton Center*
	Computers in Medicine, *Nassau A*
	Occupational & Environmental Health/Family Practice, *Murray Hill B*
	Physical Medicine & Rehabilitation/Neurology, *Murray Hill A*
	Medical Liability Mutual Insurance Company, *Sutton South*
2:00 p.m.–5:00 p.m.	Chest Diseases/Radiology, *Sutton Center*
	Cardiovascular Diseases, *Sutton South*
	Women's Medical Societies of New York State/Orthopaedic Surgery Society, *Murray Hill A*
	Neurology/Ophthalmology, *Nassau A*
	Neurosurgery, *Murray Hill B*
	Emergency Medicine/Critical Care, *Nassau B*

SATURDAY, SEPTEMBER 16

8:30 a.m.–9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m.–12:30 p.m.	Substance Abuse, *Sutton North*
	Internal Medicine (All Day), *Sutton Center*
	Neurology/Ophthalmology (All Day), *Beekman Parlor*
	Psychiatry (All Day), *Nassau B*
	Obstetrics & Gynecology/Urology, *Murray Hill A*
	General Surgery, *Sutton South*
	Dermatology/Plastic & Reconstructive Surgery (All Day), *Nassau A*
2:00 p.m.–5:00 p.m.	Internal Medicine (All Day), *Sutton Center*
	Neurology/Ophthalmology (All Day), *Beekman Parlor*
	Psychiatry (All Day), *Nassau B*
	Pediatrics/Forensic Medicine, *Sutton North*
	Otolaryngology/Neurosurgery/Plastic Surgery, *Sutton South*
	Dermatology/Plastic & Reconstructive Surgery (All Day), *Nassau A*

CME CREDITS: The Medical Society of the State of New York (MSSNY) is accredited by the Accreditation Council for Continuing Medical Education to sponsor CME for physicians. MSSNY designates all CME activities in this CME Assembly as meeting the criteria for Category 1 of the AMA, PRA. The credits are enumerated for each session. Those programs approved for credit by the American Academy of Family Physicians (AAFP) are so designated.

□

Technical Exhibits

1989 ANNUAL CME ASSEMBLY

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A. W. MARTIN MARINO, Jr., M.D., Brooklyn

Hand Surgery

THOMAS PALMIERI, M.D., New Hyde Park
New York State Society for Surgery of the Hand

Internal Medicine

SIDNEY WEINSTEIN, M.D., Rochester
New York State Society of Internal Medicine

Neurology

RICHARD SATRAN, M.D., Rochester
New York Neurological Society

Neurosurgery

KALMON POST, M.D., New York City
New York Society of Neurosurgery

Obstetrics and Gynecology

JOHN PARENTE, M.D., Bronx
American College of Obstetricians and Gynecologists

Occupational & Environmental Health including Workers' Compensation

RICHARD L. KLEIN, M.D., New York City

Ophthalmology

SAMUEL PACKER, M.D., Manhasset
New York State Ophthalmological Society

Orthopaedic Surgery

DAVID HIRSH, M.D., Scarsdale
New York Society of Orthopaedic Surgery

Otolaryngology

STEVEN PARNES, M.D., Albany
New York State Society of Otolaryngology—Head and Neck Surgery

Pathology

HENRY SIMPKINS, M.D., Staten Island
New York State Society of Pathology

Pediatrics

MARVIN BLUMBERG, M.D., Jamaica
American Academy of Pediatrics, District 11

Physical Medicine and Rehabilitation

SAMUEL SVERDLIK, M.D., New York City
New York Society of Physical Medicine and Rehabilitation

Psychiatry

RICHARD B. DROOZ, M.D., New York City
New York State Psychiatric Association

Radiology

RITA GIROLAMO, M.D., Valhalla
New York State Chapter of the American College of Radiology

Surgery

LESLIE WISE, M.D., New Hyde Park
New York State Society of Surgeons

Urology

PETER J. PUCHNER, M.D., New York City
New York State Urological Society

Women's Medical Society of New York State

LESLIE KOHMAN, M.D., Syracuse
New York State Women's Medical Society

FRIDAY, SEPTEMBER 15, 1989

Medical Technology, 1989

8:30 A.M.–9:30 A.M.
NASSAU A

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 1 Hour, Category 1; AAFP, Hour, Prescribed

William K. Weissman, M.D., New York City, *Moderator*

Objective: To advance the physician's understanding of the scientific principles which underlie emerging technologies in modern medicine.

8:30 A.M. **The Mathematics of Medical Imaging**

Lawrence Shepp, Ph.D., New Jersey

This introduction, for a general medical audience, explores the strengths and weaknesses of such technologies as CAT scanning, emission tomography and Magnetic Resonance Imaging.

Gastroenterology/Colon and Rectal Surgery

9:30 A.M.–12:30 P.M.
SUTTON CENTER

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 2.75 Hours, Prescribed

WHAT THE NON-GI CLINICIAN SHOULD KNOW ABOUT THE MANAGEMENT OF INFLAMMATORY BOWEL DISEASE IN 1989

John S. Davis, M.D., Cooperstown, *Moderator*

Director of Medical Education, The Mary Imogene Bassett Hospital, Cooperstown; Associate Professor of Clinical Medicine, College of Physicians and Surgeons of Columbia University, New York City

Objective: Attendees will see how each speaker decides on specific medical and/or surgical therapeutic approaches to four actual difficult cases. Each speaker will attempt to distinguish what is new, as opposed to established therapy. Each case report will last 20 minutes, with a 10-minute question and answer discussion. The Albert F. R. Andresen Memorial Lecture will cap the program by a discussion of "A Century of Surgery for Inflammatory Bowel Disease: Have We Reached the Bottom Line?"

9:30 A.M. **Introduction**

John S. Davis, M.D., Cooperstown

9:35 A.M. **Ulcerative Colitis of 20 Years Duration**

George Flint, M.D., Great Neck

Attending Surgeon, Long Island Jewish Medical Center, New Hyde Park, NY; Attending Surgeon, Queens Hospital Center, Jamaica, NY; Attending Surgeon, Consulting Surgeon, Mid-Island Hospital, Long Island, New York

9:55 A.M. **Questions and Answers**

10:05 A.M. **Acute Bloody Diarrhea in an 18-Year-Old**

William Mitchell, M.D., Cooperstown

Attending Physician, Department of Medicine, The Mary Imogene Bassett Hospital, Cooperstown

10:25 A.M. **Questions and Answers**

10:35 A.M. **Coffee Break to View Exhibits**

10:55 A.M. **26-Year-Old Male with a Perineal Fistula**

Richard Burg, M.D., White Plains

Attending Surgeon, White Plains Hospital, White Plains, New York

11:15 A.M. **Questions and Answers**

(continued)

11:25 A.M. **Weight Loss and Abdominal Pain Despite Steroids in a 42-Year-Old Woman with Previous Surgery for Crohn's Disease**

William H. Ramsey, M.D., Farmington, Connecticut

Assistant Professor of Medicine, Division of Gastroenterology, University of Connecticut Health Center, Farmington, Connecticut

11:45 A.M. **Questions and Answers**

11:55 A.M. **Albert F. R. Andresen Memorial Lecture: A Century of Surgery for Inflammatory Bowel Disease—Have We Reached The Bottom Line?**

Adrian J. Greenstein, M.D., New York City

Professor of Surgery, Mount Sinai School of Medicine, New York City

12:15 P.M. **Questions and Answers**

Medical Liability Mutual Insurance Company
Joint Meeting With
Medical Society of the State of New York

9:30 A.M.–12:30 P.M.
SUTTON SOUTH

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 2.75 Hours, Prescribed

MOCK TRIAL: "THE DOCTOR AS A DEFENDANT"

Ralph M. Schwartz, M.D., New York, *Chairman*

Vice President-Secretary, Medical Liability Mutual Insurance Company, New York

Objective: The purpose of this seminar is to provide physicians in attendance with "hands on" information about problems and procedures of a trial. During this Mock Trial a case will be tried before a Judge and Jury by Plaintiff and Defense counsels with expert witnesses representing patient and physician. Sufficient time will be devoted for questions and answers from the attendees at any point during the seminar.

9:30 A.M. **Introductory remarks**

Ralph M. Schwartz, M.D., New York

Direct Examination of Defendant and Expert Witness
George F. Mould, Esq., Syracuse
Partner—Martin, Ganotis & Brown, New York

Cross-Examination of the Defendant and Expert Witness

David E. Brown, Esq., Rochester
Partner—Martin, Ganotis & Brown, New York

Program will be moderated by:
Donald J. Fager, Esq., New York
Vice-President, Medical Liability Mutual Insurance Company, New York

There will be a coffee break and opportunity to visit exhibits from 10:30 to 10:45 A.M.

Occupational and Environmental Health
Joint Meeting
Family Practice

9:30 A.M.–12:30 P.M.
MURRAY HILL B

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 2.75 Hours, Prescribed

TIGHT BUILDING SYNDROME: A PRIMARY CARE PERSPECTIVE

John Falencki, Jr., M.D., New York City, *Moderator*

Family Physician, New York City

Objective: Indoor Air Pollution will be elucidated and provide the primary care physician with tools for diagnosis and management.

9:30 A.M. **Definition and Detection of Tight Building Syndrome**

Philip Morey, Ph.D., Pennsylvania

Indoor Air Quality Consultant, Clayton Environmental Consultants, Wayne, Pennsylvania

10:05 A.M. **Diagnosis and Management of Tight Building Syndrome**

Mark Wilkenfeld, M.D., New York City

Department of Community Medicine, Mt. Sinai Medical School, New York City

(continued)

10:40 A.M. **Diagnosis and Management of Tight Building Syndrome**

Patricia Hagendorn, M.D., New York City

Medical Director, Chase Manhattan Bank, New York City; Attending Physician in Medicine, St. Vincent's Hospital & Medical Center, New York City

11:00 A.M. **Coffee Break to View Exhibits**

11:15 A.M. **Workers' Compensation Aspects of Occupational Diseases**

Speaker, to be announced, will be a representative of the Workers' Compensation Board

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Physical Medicine and Rehabilitation
Joint Meeting
Neurology

9:30 A.M.–12:30 P.M.
MURRAY HILL A

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1;
AAFP, 2.75 Hours, Prescribed

THE PROS AND CONS OF COGNITIVE REHABILITATION

Samuel S. Sverdlik, M.D., New York City, *Moderator*

Emeritus Director, Department of Rehabilitation Medicine, St. Vincent's Hospital and Medical Center, New York City; Clinical Professor of Rehabilitation Medicine, New York Medical College, Valhalla

Objective: The long term management of brain injured patients is a costly and often frustrating challenge. The scope of the problem in New York State and our present state of knowledge regarding management of these patients will be described. The merits and limits of these efforts will be critically discussed.

9:35 A.M. **The Problems of Head Injury**

William E. Reynolds, D.D.S., Albany

Director, Bureau of Development and Standards, New York State Health Department, Albany

10:00 A.M. **The Nature of Head Injury**

David Goldblatt, M.D., Rochester

Clinical Attending, Strong Memorial Hospital, Rochester; Professor of Neurology, University of Rochester School of Medicine and Dentistry, Rochester

10:25 A.M. **Coffee Break To View Exhibits**

10:40 A.M. **Contemporary Issues in Cognitive Rehabilitation**

Leonard Dillar, Ph.D., New York City

Chief, Psychology, Rusk Institute of Rehabilitation Medicine; Professor, Clinical Rehabilitation Medicine, New York University School of Medicine, New York

11:15 A.M. **A Neurologist's Views of Cognitive Rehabilitation**

Joshua Hollander, M.D., Rochester

Associate Profesor, Clinical Neurology, University of Rochester School of Medicine and Dentistry; Chief, Neurology, Rochester General Hospital, Rochester

11:40 A.M. **A Physiatrist's Views of Cognitive Rehabilitation**

Loren M. Fishman, M.D., Flushing

Associate Professor, Rehabilitation Medicine, New York City; Director, Department of Physical Medicine, Flushing Hospital, Flushing

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Computers in Medicine, 1989

9:30 A.M.–12:30 P.M.
NASSAU A

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 2.75 Hours, Prescribed

William Bauman, M.D., Connecticut, *Moderator*

Director of Medical Education, Danbury Hospital, Danbury, Connecticut

Objective: Explaining how the methods of information technology lead to the goals of quality assurance in patient care.

9:30 A.M. **The Automation of Clinical Experience**

Gilbert Mellin, M.D., New York

Director of Quality Assurance, Presbyterian Hospital, New York. Medical care requires timely medical information and access to it. The decompensation of the classical medical record obliges us to develop new systems to communicate this information.

10:45 A.M. **Refined DRG's—The Computer Quality Care**

William Bauman, M.D., Connecticut

Modification of DRG's serves as an index for measuring quality of care for hospitalized patients.

(continued)

11:00–11:15 A.M. **Coffee**

11:15–12:00 P.M. Peter D. Esser, Ph.D., New York City

Associate Professor, Columbia University Small computers have had a significant impact in the clinical utilization of radiologic images. Modern processing techniques allow data enhancement and analysis in Nuclear Cardiology. Nuclear Medicine and general body imaging. Future enhancements may broaden the clinical scope.

12:00–12:30 P.M. **Panel discussion**

Neurology (Neuro-ophthalmology) Joint Meeting with Ophthalmology

2:00 P.M.–5:00 P.M.
NASSAU A

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 2.75 Hours, Prescribed

NEURO-OPHTHALMOLOGIC ADVANCES IN MOVEMENT

Joshua Hollander, M.D., Rochester, *Moderator*
Attending, Neurology, Rochester General Hospital; Associate, Neurology, Strong Memorial Hospital

Objective: Physicians will learn to use observations made on the patient's face and head as a window to the general problem of movement disorders.

2:00 P.M. **Movement Disorders in General**
Ira Shoulson, M.D., Rochester
Professor of Neurology, University of Rochester Medical Center, Rochester

2:40 P.M. **Facial Dyskinesis**
Stanley Fahn, M.D., New York City
H. Houston Merit Professor of Neurology, Columbia-Presbyterian Medical Center Neurological Institute, New York City

3:20 P.M. **Coffee Break to View Exhibits**

3:40 P.M. **The Eye In Movement Disorders**
Jeffrey Odel, M.D., New York City
Assistant Professor of Clinical Ophthalmology, Institute of Ophthalmology, New York City

4:40 P.M. **Panel Discussion**

Neurosurgery

2:00 P.M.–5:00 P.M.
MURRAY HILL B

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 2.75 Hours, Prescribed

NEUROSURGICAL TREATMENT OF SPINAL TUMORS

Kalmon D. Post, M.D., New York City, *Moderator*
Professor and Vice Chairman, Department of Neurological Surgery, Columbia University, College of Physicians and Surgeons, New York City.

Objective: Update neurosurgeons on spinal tumors.

2:10 P.M. **Intramedullary Spinal Cord Tumors**
Paul R. Cooper, M.D., New York City
Associate Professor of Neurological Surgery, New York University Medical Center, New York City

2:45 P.M. **Intradural Extramedullary Spinal Cord Tumors**
Charles Hodge, M.D., Syracuse
Professor and Chairman, Department of Neurosurgery, Syracuse University, Syracuse

3:30 P.M. **Coffee Break to View Exhibits**

3:45 P.M. **Tumors of the Spinal Cord**
Martin B. Camins, M.D., New York City
Associate Clinical Professor of Neurological Surgery, Mt. Sinai Medical Center, New York City

4:30 P.M. **Panel Discussion**

Check for Messages at the Message Center

(continued)

Cardiovascular Diseases
Joint Meeting With
New York Cardiological Society
In Association with
The New York Cardiac Center

2:00 P.M.–5:00 P.M.
SUTTON SOUTH

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 2.75 Hours, Prescribed

CLINICAL DILEMMAS IN CARDIOLOGY

RICHARD GORLIN, M.D., New York City, *Moderator*
Murray M. Rosenberg Professor of Medicine; Chairman, Department of Medicine,
Mt. Sinai Medical Center

Objective: Clinicians will learn how to handle clinical dilemmas in four types of cardiological problems: arrhythmias, myocardial infarction, valvular heart disease and congestive heart failure.

2:00 P.M. **Introduction**
RICHARD GORLIN, M.D., New York City, *Moderator*

2:05 P.M. **Current Status of Arrhythmias**
STEPHEN S. SCHEIDT, M.D., New York City
Assistant Dean for CME, Professor of Clinical Medicine, The New York Hospital—Cornell Medical Center, New York

2:35 P.M. **Thrombolysis in Acute Myocardial Infarction**
RICHARD GORLIN, M.D., New York City

3:05 P.M. **Coffee Break to View Exhibits**

3:20 P.M. **Management of Valvular Heart Disease: Valvuloplasty vs. Surgery**
ARTHUR C. FOX, M.D., New York City
Professor of Medicine, Chief, Cardiovascular Section, Department of Medicine, New York University School of Medicine, Bellevue Medical Center, New York

3:50 P.M. **Congestive Heart Failure; Current Approaches**
EDMUND H. SONNENBLICK, M.D., Bronx
Olson Professor of Medicine; Chief, Division of Cardiology, Albert Einstein College of Medicine, New York

4:15 P.M. **Panel Discussion**

Women's Medical Societies of New York State
Joint Meeting With
Orthopaedic Surgery Society

2:00 P.M.–5:00 P.M.
MURRAY HILL A

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1, AAFP, 2.75 Hours, Prescribed

OSTEOPOROSIS—PREVENTION, DILEMMAS AND NEWER MODALITIES OF TREATMENT

ADELAIDE T. SCANLON, M.D., Glen Cove, *Moderator*
Attending Pediatrician, Community Hospital of Glen Cove, N.Y.; American Women's Medical Societies of New York

Objective: This program is directed toward primary care physicians as well as Orthopaedic Surgeons and Gynecologists in order to increase their awareness of methods of early treatment to prevent osteoporosis as well as to update them on the occurrence and treatment of fractures and the possibility of newer therapy for the future.

2:05 P.M. **Role of Hormones in Prevention and Treatment of Osteoporosis**
LILA A. WALLIS, M.D., New York City
President of American Medical Women's Association; Clinical Professor of Medicine at Cornell Medical Center

2:25 P.M. **Reproductive Disorders In Ballet Dancers and their Effect on Bone**
MICHELLE P. WARREN, M.D., New York City
Associate Attending, Obstetrician/Gynecologist, St. Luke's/Roosevelt Hospital Medical Center; Director—Gonadotropin Laboratory, St. Luke's/Roosevelt Hospital Medical Center, New York City

(continued)

2:45 P.M. **Beyond Calcium—Nutrition and Osteoporosis**

SUSAN BROWN, Ph.D., Syracuse

Director of Nutrition Education and Consulting Service

3:15 P.M. **Coffee Break to View Exhibits**

3:30 P.M. David M. Hirsh, M.D., Bronx, *Moderator*

Associate Clinical Professor, Department of Orthopaedic Surgery, Jack D. Weiler Hospital of the Albert Einstein College of Medicine

3:35 P.M. **Fracture Incidence and Treatment in Osteoporosis**

NEIL J. COBELLI, M.D., Bronx

Assistant Clinical Professor, Albert Einstein College of Medicine, New York

3:55 P.M. **Definitive Diagnosis and Treatment**

CHARLES CORNELL, M.D., New York City

Assistant Professor—Orthopaedic Surgery, Cornell University Medical College; Hospital for Special Surgery, New York

4:15 P.M. **Newer Modalities for Treatment of Osteoporosis**

DAVID ZACKSON, M.D., New York City

Assistant Professor of Medicine, Cornell University Medical Center, Director of Bone and Mineral Metabolism Section of Rogosin Institute

4:35 P.M. **Panel of all speakers for questions and answers**

Emergency Medicine/Critical Care
Joint Meeting With
New York Chapter of American College of Emergency Physicians

2:00 P.M.–5:00 P.M.
NASSAU B

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 2.75 Hours, Prescribed

CLINICAL DILEMMAS IN THE MANAGEMENT OF THE ACUTE CARE PATIENTS

LORRAINE M. GIORDANO, M.D., FACEP, Jamaica, *Moderator*

Chairperson, Dept. of Emergency Medicine, The Jamaica Hospital, Jamaica, New York

Objective: All physicians, regardless of specialty, should be aware of the problems presented by the patient who suddenly becomes acutely ill or injured, such as the management of the difficult airway, the differential diagnosis of the patient who presents in shock, the victim of poisoning and the patient with an acute abdomen. Attendees at this program will be made aware of the various new technologies of and Indications for physiological monitoring in the critical care unit.

2:00 P.M. **Management of the Difficult Airway**

NINA MAZUR, M.D., Brooklyn

Director, Emergency Services, The Brooklyn/Caledonian Hospital, Brooklyn, New York

2:20 P.M. **Evaluation and Treatment of Shock**

WILLIAM GOETZ, M.D., Bronx

Associate Director, Emergency Department, Our Lady of Mercy Hospital, Bronx, New York

3:00 P.M. **Coffee Break**

3:15 P.M. **Management of the Poisoned Patient**

PAUL ADLER, D.O., Bronx

Director, Emergency Services, North Central Bronx Hospital, Bronx, New York

3:35 P.M. **Evaluation, Differential Diagnosis and Treatment of the Acute Abdomen**

LYNNE RICHARDSON, M.D., New York City

Associate Chief, Emergency Services, Harlem Hospital Center, New York, New York

3:55 P.M. **Physiologic Monitoring in the Critical Care Units**

CARLOS R. ORTIZ, M.D., Rochester

Medical Director, Intensive Care Units, The Genesee Hospital, Rochester, New York

4:15 P.M. **Questions and Answers**

Chest Diseases/Radiology
Joint Meeting
American College of Chest Physicians—New York State Chapter of the American College of Radiology

2:00 P.M.–5:00 P.M.
SUTTON CENTER

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 2.75 Hours, Prescribed

(continued)

ADVANCES IN DIAGNOSIS AND TREATMENT OF LUNG CANCER—1988

Frederick B. Parker, Jr., M.D., Syracuse, *Moderator*
President, American College of Chest Physicians, New York State Affiliate

Objective: The objectives of this program are to make all physicians (specialists and non-specialists) aware of recent advances in diagnosing and treating cancer of the lung. Special attention will be directed to the overlapping responsibilities of the radiologist, oncologist and surgeon in the case of the patient with lung cancer.

2:00 P.M. **Howard Lilienthal Memorial Lecture: Pulmonary Spread of Bronchogenic Carcinoma**
E. Robert Heitzman, M.D., Syracuse
Professor and Vice-Chairman of Radiology, SUNY Health Science Center at Syracuse, University Hospital

2:35 P.M. **Radiologic Staging of Bronchogenic Carcinoma**
David P. Naidich, M.D., New York City
Associate Professor of Radiology, New York University

3:05 P.M. **Coffee Break to View Exhibits**

3:25 P.M. **Current Therapy of Small Cell Carcinoma**
Stephen L. Graziano, M.D., Syracuse
Chief, Oncology Section, Veterans Administration Medical Center, Syracuse, New York

4:00 P.M. **Current Surgical Management of Non-Small Cell Carcinoma of the Lung**
Stanley W. Dziuban, M.D., Albany
Associate Professor of Surgery, Division of Thoracic Surgery, Albany Medical Center

4:30 P.M. **Advances in Laser Therapy in Bronchogenic Carcinoma**
Richard H. Feins, M.D., Rochester
Associate Professor of Surgery, Division of Thoracic Surgery, University of Rochester School of Medicine and Dentistry

SATURDAY, SEPTEMBER 16, 1989

Medical Technology

8:30 A.M.–9:30 A.M.
MURRAY HILL B

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 1 Hour, Category 1; AAFP Hour, Prescribed

MEDICAL TECHNOLOGY, 1989

William K. Weissman, M.D., New York City, *Moderator*

Objective: To be announced

8:30 A.M. **Integration of Medical Information**
Mr. John Skerchock, District Manager, Hewlett Packard Health Care Information Systems

Internal Medicine

9:30 A.M.–5:00 P.M.
SUTTON CENTER

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 6 Hours, Category 1; AAFP, 5.25 Hours, Prescribed

NEW FRONTIERS IN GASTROENTEROLOGY

Robert Scheig, M.D., Buffalo, *Moderator*
Professor of Medicine and Biological Sciences, State University of New York at Buffalo; Head of the Department of Medicine, Buffalo General Hospital, Buffalo

Objective: This meeting is designed for primary care physicians and all other physicians interested in an update of a variety of Gastroenterology disorders, especially peptic ulcer disease and infectious disease issues.

9:30 A.M. **Introductory Remarks**
Robert Scheig, M.D., Buffalo, *Moderator*

9:35 A.M. **Overview of Peptic Ulcer Disease**
James Freston, M.D., Farmington, Connecticut
Professor and Chairman, Department of Medicine, University of Connecticut, School of Medicine

(continued)

10:05 A.M. **Recent Therapeutic Advances In Peptic Ulcer Disease**

Thomas J. Humphries, M.D., Pennsylvania

Director, Worldwide Clinical Research, Merck, Sharp, Dohme Research Laboratories

10:45 A.M. **Coffee Break To View Exhibits**

11:05 A.M. **Role Prostoglandins Play In the Prevention of NSAID Induced Gastropathy**

Donald E. Wilson, M.D., Brooklyn

Professor and Chairman, Department of Medicine, State University of New York Health Science Center at Brooklyn

11:45 A.M. **A. Walter Suiter Lectureship Fund**

Non-Cardiac Chest Pain

Daniel Camera, M.D., Buffalo
Associate Professor of Medicine, State University of New York at Buffalo School of Medicine, Buffalo; Clinical Director of Gastroenterology, Buffalo General Hospital, Buffalo

12:20 P.M. **Questions and Answers**

12:30 P.M. **Lunch Break**

2:00 P.M. **Campylobacter Pylori Infection, Role in Peptic Disease**

William Marsh, M.D.

Associate Professor of Medicine, Medical University of South Carolina, College of Medicine, Charleston, South Carolina

2:40 P.M. **AIDS and the Gastrointestinal Tract**

Jan M. Novak, M.D.

Director of Gastroenterology, Erie County Medical Center, Buffalo

3:20 P.M. **Coffee Break to View the Exhibits**

3:40 P.M. **Viral Hepatitis: Diagnosis, Treatment and Prevention**

Robert Scheig, M.D.

Professor of Medicine and Biological Sciences, Head of the Department of Medicine, Buffalo General Hospital, Buffalo

4:25 P.M. **Questions and Answers**

Psychiatry
Joint Meeting With
New York State Psychiatric Association

9:30 A.M.–5:00 P.M.
NASSAU B

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 1 Hour, Category 1; AAFP, 5.25 Hours, Prescribed

RECENT ADVANCES IN PSYCHIATRY

Richard B. Drooz, M.D., New York City, *Moderator*

Associate Professor of Psychiatry (Retired), State University of New York Health Science Center at Brooklyn; Attending Psychiatrist (Retired), Kings County Hospital Center, Brooklyn

Objective: Physicians of all specialties attending this program will be provided a comprehensive view of how recent psychiatric advances impinge on their patient care.

9:30 A.M. **Welcome and Orientation**

George L. Ginsberg, M.D., New York City

President, New York State Psychiatric Association, Director of Psychiatric Services and Graduate Education, New York University Medical Center

and

Richard B. Drooz, M.D., New York City

9:45 A.M. **Resistant Depression: Diagnosis and Treatment**

Matthew V. Rudorfer, M.D., Bethesda, Maryland

Chief, Clinical Research Unit, National Institute of Mental Health, Bethesda, Maryland

10:15 A.M. **Coffee Break**

10:30 A.M. **The Psychotherapy of a Battered Woman**

Samuel C. Klagsbrun, M.D., New York

Executive Medical Director, Four Winds Hospitals, Katonah, Saratoga Springs and Chicago; Attending Psychiatrist, Presbyterian and St. Luke's Hospitals

11:30 A.M. **Psychiatric Emergencies Which May Result in Malpractice Cases**

Harold E. Berson, M.D., Brooklyn

Attending Psychiatrist, Maimonides Medical Center, Brooklyn; Attending Neuropsychiatrist, Interfaith Medical Center, Brooklyn

12:00 P.M. **Lunch**

Check for Messages at the Message Center

(continued)

1:45 P.M. **Managing the Dangerous Patient in the General Hospital**

HARVEY BLUESTONE, M.D., Bronx

Director, Department of Psychiatry, Bronx-Lebanon Hospital Center, Bronx, New York; Professor of Psychiatry, Albert Einstein College of Medicine

2:05 P.M. SHELDON TRAVIN, M.D., Bronx

Associate Director, Department of Psychiatry, Bronx-Lebanon Hospital Center; Associate Professor of Psychiatry, Albert Einstein College of Medicine

2:25 P.M. HUNG KON LEE, M.D., Bronx

Assistant Professor of Psychiatry, Albert Einstein College of Medicine; Chairman, Quality Assurance Committee, Bronx-Lebanon Hospital Center

2:45 P.M. **Coffee Break**

3:00 P.M. **Single Photon Emission Computed Tomography (SPECT) in Psychiatric Differential Diagnosis**

RALPH A. O'CONNELL, M.D., New York City

Clinical Director of Psychiatry, St. Vincent's Hospital and Medical Center, New York City; Professor of Clinical Psychiatry, New York Medical College

RONALD L. VAN HEERTUM, M.D., New York City

Chief of Nuclear Medicine, St. Vincent's Hospital and Medical Center, New York City; Associate Professor of Clinical Radiology, New York Medical College

3:30 P.M. **Toward a Cellular Neurobiology of Schizophrenia: Cell Culture of Mesolimbic Dopamine Neurons**

STEPHEN RAYPORT, M.D., Ph.D., New York City

Assistant Professor of Clinical Psychiatry, Neurobiology and Behavior, Anatomy and Cell Biology; Psychiatrist, New York State Psychiatric Institute, New York City

4:00 P.M. **Family Psychoeducation in the Aftercare Treatment of Schizophrenia**

STEPHEN A. COLE, M.D., New York City

Chief of Partial Hospitalization Program, St. Vincent's Hospital, New York City; Clinical Associate Professor of Psychiatry, New York University Medical Center, New York City

4:45 P.M. **Discussion**

Neurology
Joint Meeting With
Ophthalmology (Neuro-ophthalmology)

9:30 A.M.–5:00 P.M.
BEEKMAN PARLOR

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 6 Hours, Category 1; AAFP, 5.25 Hours Prescribed

NEURO-OPHTHALMOLOGIC ADVANCES IN VISUAL DISORDERS: VASCULAR DIAGNOSIS AND NEURO-IMAGING

JACQUELINE M. S. WINTERKORN, Ph.D., M.D., Manhasset, *Moderator*

Physician-in-Charge, Neuro-Ophthalmology; North Shore University Hospital, Manhasset; Neuro-Ophthalmology Attending, Manhattan Eye, Ear and Throat Hospital, New York City

Objective: Physicians attending this program will be updated on recent developments in Neuro-Ophthalmology and their application in practice.

9:30 A.M. **Introduction**

JACQUELINE M. S. WINTERKORN, Ph.D., M.D.

9:35 A.M. **Optic Nerve Dysfunction**

MYLES M. BEHRENS, M.D., New York City

Neuro-Ophthalmologist, Harkness Eye Institute, Columbia Presbyterian Medical Center, New York City

10:05 A.M. **Diagnosis of Vascular Disorders in Neuro-Ophthalmic Patients**

JAY MOHR, M.D., New York City

Neurologic Institute, Columbia Presbyterian Medical Center, New York City

11:05 A.M. **Coffee Break to View Exhibits**

11:20 A.M. **Neuro-Imaging of the Visual System**

SADEK HILAL, M.D., New York City

Department of Neuroradiology, Neurological Institute, Columbia Presbyterian Medical Center, New York City

12:20 P.M. **Questions and Answers**

12:30 P.M. **Lunch**

2:00 P.M. **Diagnosis and Treatment of Pseudotumor Cerebri**

JAMES J. CORBETT, M.D., Iowa

Professor of Ophthalmology/Neurology, University of Iowa

2:40 P.M. **Advances in Diagnosis and Treatment of Glaucoma**

CARL CAMRAS, M.D., New York City

Ophthalmology Department, Mt. Sinai Medical Center, New York City

3:20 P.M. **Coffee Break To View Exhibits**

(continued)

3:35 P.M. **Surgical Treatment of Optic Nerve Disorders**

ROBERT C. SERGOTT, M.D., Pennsylvania

Wills Eye Hospital, Pennsylvania

4:15 P.M. **Understanding Automated Visual Fields**

STEVEN A. NEWMAN, M.D., Virginia

Assistant Professor of Ophthalmology and Neurological Surgery, University of Virginia Health Sciences

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Dermatology
Joint Meeting With
New York State Society of Dermatology
The American Society of Plastic and Reconstructive Surgery

9:30 A.M.–12:30 P.M.
NASSAU A

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 2.75 Hours, Prescribed

ADVANCES IN CUTANEOUS SURGERY

LOWELL A. GOLDSMITH, M.D., Rochester, *Moderator*

James H. Sterner Professor of Dermatology; Chairman, Department of Dermatology, University of Rochester

ALAN MATARASSO, M.D., New York City, *Moderator*

Attending Surgeon, Manhattan Eye, Ear & Throat Hospital; Faculty, Department of Plastic Surgery, Albert Einstein Hospital

Objective: Objective: integration of advances in cutaneous surgery from both dermatologists and plastic surgeons—to increase dialogue among these two groups, and to inform physicians of other disciplines of these advances.

9:30 A.M. **Management of Congenital and Dysplastic Nevi**

ALFRED KOPF, M.D., New York City

Clinical Professor, Department of Dermatology, New York University Medical Center, New York

10:00 A.M. **Biologic Requirements and Approaches to Skin Replacement in Severe Burn Injury**

CHARLES CUONO, M.D., New Haven, Connecticut

Associate Professor of Surgery, Yale University School of Medicine; Director, Yale Skin Bank, New Haven

10:30 A.M. **Coffee Break**

10:45 A.M. **Overview of Chemical Peels**

EUGENE BODIAN, M.D., Great Neck

Clinical Professor of Medicine (Dermatology), Cornell Medical Center; Chief, Dermatology, North Shore University Hospital Medical Center, Great Neck, New York

11:10 A.M. **Dermabrasions and Chemical Peels**

ARTHUR SHIP, M.D., New York City

Associate Clinical Professor, Dept. of Plastic and Reconstructive Surgery, Albert Einstein College of Yeshiva University; Attending Surgeon, Department of Surgery, Division of Plastic Surgery, Montefiore Hospital College of Medicine, Bronx

11:40 A.M. **Management of Recurrent Skin Cancers**

MICHAEL ALBOM, M.D., New York City

Associate Clinical Professor, Dept. of Dermatology, Skin Cancer Unit, New York University Medical Center

12:10 P.M. **Closure of Skin Cancer Defects**

PAUL WEISS, M.D., New York City

Associate Clinical Professor, Department of Plastic and Reconstructive Surgery, Albert Einstein College of Yeshiva University; Attending Surgeon, Department of Surgery, Division of Plastic Surgery, Montefiore Hospital, Bronx

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Dermatology
Joint Meeting With
American Academy of Dermatology Advisory Council and New York State Society of Dermatology

2:30 P.M.–5:00 P.M.
NASSAU A

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 2.7 Hours, Prescribed

EFFECTS OF STATE LEGISLATION AND REGULATION ON THE PRACTICE OF DERMATOLOGY

RICHARD L. CORNELISON, M.D., Oklahoma, *Moderator*

Chairman, Advisory Council, American Academy of Dermatology

(continued)

Objective: The program will provide practicing dermatologists and primary care physicians an opportunity to consider how state laws and directives from state agencies affect the current and future practice of dermatology.

2:30 P.M. **An Overview of State Issues of Concern to the Specialty of Dermatology**

RICHARD L. CORNELISON, M.D., Oklahoma

2:45 P.M. **A Dermatologist in the State Legislature**

The Honorable HAROLD L. COLBURN, JR., M.D.

Assemblyman, New Jersey State Legislature; Practicing Dermatologist, New Jersey

3:00 P.M. **A Dermatologist as a State Regulator**

EDWARD W. LUKA, M.D., New Jersey

Past Chairman, New Jersey Board of Medical Examiners; Practicing Dermatologist

3:15 P.M. **Working with the New York State Medical Society; Process and Some Issues**

GERARD L. CONWAY, ESQ., Albany

Director, Division of Governmental Affairs, Medical Society of the State of New York

3:30 P.M. **Coffee Break to View Exhibits**

3:45 P.M. **Legislation from the New York Perspective: Process and Issues**

JOEL S. KORANSKY, M.D.

Practicing Dermatologist, Saratoga Springs; Chairman, Committee on Public Affairs, New York State Society of Dermatology

4:00 P.M. **Status of Current Issues and Regional Trends**

IRWIN M. FREEDBERG, M.D., New York City

Chairman, Department of Dermatology, New York University Medical Center; Chairman, Council on Government Liaison, American Academy of Dermatology

4:15 P.M. **Questions and Discussion**

Substance Abuse

9:30 A.M.–12:30 P.M.
SUTTON NORTH

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1;
AAFP, 2.75 Hours, Prescribed

COCAINE USE IN THE REAL WORLD

ANNE GELLER, M.D., New York, *Moderator*

Director of Smithers Center, St. Luke's Roosevelt Hospital; Assistant Professor of Clinical Social Medicine at Columbia College of Physicians and Surgeons, New York City

Objective: Physicians attending this program will develop an understanding of the way in which cocaine addiction may present in medical settings; treatment applications will be discussed.

9:30 A.M. **Introductory Remarks**

ANNE GELLER, M.D., New York City, *Moderator*

9:35 A.M. **Who Uses Cocaine and How it Works**

ROBERT B. MILLMAN, M.D., New York City

SAUL B. STEINBERG, M.D., Professor of Psychiatry and Public Health, Cornell University Medical College, New York

10:10 A.M. **Cocaine and Sex, the Intimate Connection**

ARNOLD M. WASHTON, Ph.D., New York City

Executive Director of the Washton Institute, New York

10:45 A.M. **Coffee Break To View Exhibits**

11:00 A.M. **An Integrated Medical Model of Addiction**

JAMES FINE, M.D., Brooklyn

Director of Psychiatric Services and Program Services, Addictive Disease Hospital, State University of New York at Brooklyn

11:30 A.M. **Cocaine in the Emergency Room**

GREGORY HUSK, M.D., New York City

Director of Emergency Room, Roosevelt Hospital, New York City

12:00 P.M. **Panel Discussion with Audience Participation**

General Surgery
Joint Meeting With
New York State Society of Surgeons

9:30 A.M.–12:30 P.M.
SUTTON SOUTH

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 2.75 Hours, Prescribed

(continued)

ADVANCES IN GENERAL SURGERY

LESLIE WISE, M.D., F.A.C.S., New Hyde Park, *Moderator*

Chairman, Department of Surgery, Long Island Jewish Medical Center, New Hyde Park, New York; Professor of Surgery, SUNY, Stony Brook, New York

Objective: This program is presented for surgeons and primary care physicians to acquaint them with newer developments in general surgery and their applications to patient care.

9:30 A.M. **Introduction**

LESLIE WISE, M.D., F.A.C.S., New Hyde Park

9:40 A.M. **The New Economics of Surgical Care**

ERIC MUNOZ, M.D., F.A.C.S., New Hyde Park

Head, Research Division, Department of Surgery, Long Island Jewish Medical Center, New Hyde Park, New York

10:10 A.M. **Gall Bladder Lithotripsy—A New Cost Effective Option**

HOUSTON JOHNSON, M.D., F.A.C.S., New Hyde Park

Department of Surgery, Long Island Jewish Medical Center, New Hyde Park, New York; Full-time Surgical Oncologist; Assistant Professor of Surgery, State University of New York, Stony Brook

10:40 A.M. **Coffee Break to View Exhibits**

11:00 A.M. **Peripheral Vascular Surgery, Percutaneous and Laser Techniques**

JON COHEN, M.D., F.A.C.S., New Hyde Park

Department of Surgery, Long Island Jewish Medical Center, New Hyde Park, New York; Peripheral Vascular Surgeon, Department of Surgery, Long Island Jewish Medical Center, New Hyde Park, New York

11:25 A.M. **AIDS and the Surgeon**

IRVING MARGOLIS, M.D., F.A.C.S., Jamaica

Director of Surgery, Queens Hospital Center Affiliation of the Long Island Jewish Medical Center, Jamaica; Professor of Clinical Surgery, State University of New York at Stony Brook

11:50 A.M. **New Technologies in Pediatric Plastic Surgery**

BARRY DOUGLAS, M.D., New Hyde Park

Staff Surgeon, Division of Plastic Surgery, Department of Surgery, Long Island Jewish Medical Center, New Hyde Park, New York

12:15 P.M. **Discussion**

Obstetrics and Gynecology/Urology
Joint Meeting With
American College of Obstetricians and Gynecologists, District II
New York State Urological Society

9:30 A.M.–12:30 P.M.
MURRAY HILL A

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 2.75 Hours, Prescribed

CASE MANAGEMENT AND CLINICAL DILEMMAS ASSOCIATED WITH OBSTETRICS/GYNECOLOGY AND UROLOGY

JOHN T. PARENTE, M.D., *Moderator*, New York City

Director, Department of Obstetrics and Gynecology, Bronx-Lebanon Hospital Center, Bronx

MAURICE GONDER, M.D., *Moderator*, Stony Brook

Chairman, Department of Urology, State University of New York at Stony Brook

MARTIN STONE, M.D., *Moderator*, Stony Brook

Professor Emeritus, Obstetrics and Gynecology, State University of New York at Stony Brook

Objective: Directed toward obstetricians/gynecologists, urologists and primary care physicians who often are faced with critical decisions in the management of the patient with gynecological/urological problems.

9:30 A.M. **Introduction**

JOHN T. PARENTE, M.D., Bronx, *Moderator*

9:35 A.M. **Ureteral Injuries in Pelvic Surgery and Management**

WILLIAM MANN, M.D., Stony Brook

Chief of Gynecologic Oncology, State University of New York at Stony Brook

10:15 A.M. **Tumors of Pregnancy**

ROBERT WACHESKA, M.D., Stony Brook

Attending, Department of Urology, University Hospital, Stony Brook

(continued)

10:45 A.M. **Coffee Break to View Exhibits**

11:00 A.M. **Ureteral Stinting**

MAURICE GONDER, M.D., Stony Brook

Chairman, Department of Urology, State University of New York at Stony Brook

11:30 A.M. **Vesico-Vaginal Fistulae**

LEON TANCER, M.D., Brooklyn

Director, Department of Obstetrics and Gynecology, Maimonides Medical Center, Brooklyn, New York

12:00 P.M. **Questions and Answers**

Otolaryngology
Joint Meeting With
Neurosurgery and Plastic Surgery

2:00 P.M.–5:00 P.M.
SUTTON SOUTH

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 2.75 Hours, Prescribed

BASE OF SKULL SURGERY, MULTIDISCIPLINARY APPROACH

STEVEN M. PARNES, M.D., Albany, *Moderator*

Professor and Head, Division of Otolaryngology, Albany Medical Center, Albany, New York

Objective: This program, although highly technical for a specialized audience, should be of interest to primary care physicians as well. Specialists will learn new techniques and generalists will learn of the newer therapeutic possibilities which will be of assistance in properly referring their patients for treatment.

2:10 P.M. **Otolaryngologic Aspects of Combined Cranial Facial Resection**

ANDREW BLITZER, M.D., New York City

Professor of Otolaryngology, Vice Chairman, Columbia University College of Physicians & Surgeons, Department of Otolaryngology, Columbia-Presbyterian Medical Center, New York

2:50 A.M. **Neurosurgical Aspects of Combined Cranial-Facial Resection**

KALMON POST, M.D., New York City

Professor and Vice Chairman, Department of Neurosurgery, Columbia University College of Physicians and Surgeons, New York

3:30 P.M. **Coffee Break**

3:40 P.M. **Middle Cranial Fossa Surgery**

YOSEF P. KRESPI, M.D., Brooklyn

Professor and Chairman, Department of Otolaryngology, Long Island College Hospital/SUNY Health Science Center at Brooklyn

4:20 P.M. **Craniofacial Reconstruction**

GREGORY LATRENTA, M.D., New York City

Assistant Professor of Surgery, Cornell Medical School; Director, Craniofacial, Cleft Lip/Palate Division, New York Hospital, New York

Pediatrics/Forensic Medicine
Joint Meeting With
American Academy of Pediatrics—District II

2:00 P.M.–5:00 P.M.
SUTTON NORTH

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 2.75 Hours, Prescribed

CLINICAL DILEMMAS IN PEDIATRICS

MARVIN L. BLUMBERG, M.D., Jamaica, *Moderator*

Chairman, Department of Pediatrics, The Jamaica Hospital; Assoc. Professor of Clinical Pediatrics, School of Medicine, SUNY, Stony Brook, NY

Objective: Physicians often encounter pediatric problems that are not the usual diseases and require experience, special testing and intricate management. There are fatal cases where diagnoses depend on autopsy findings. Physicians attending this program will be made aware of these problems and how they can be managed.

(continued)

(SATURDAY continued)

2:00 P.M. **Diagnosis and Management of Infant Apnea**

JAMES G. LIONE, M.D., Flushing

Chairman, Department of Pediatrics, Flushing Hospital, Flushing, New York

2:30 P.M. **AIDS in Infants and Children**

LEONARD R. KRILOV, M.D., New Hyde Park

Schneider Children's Hospital, L.I.J. Medical Center, New Hyde Park, N.Y.; Attending, Division of Infectious Diseases, Schneider Children's Hospital

3:00 P.M. **Coffee Break to View Exhibits**

3:20 P.M. **Radiological Diagnosis of Child Abuse**

MELVIN H. BECKER, M.D., New York City

Attending in Radiology, New York University Hospital and Bellevue Hospital; Consultant in Roentgenology, Goldwater Memorial Hospital

3:50 P.M. **Clinical Interrelation between the Pediatrician and the Medical Examiner**

ELLIOT M. GROSS, M.D., New York City

Former Chief Medical Examiner of New York City; Visiting Clinician, Professor of Pathology, Indiana University School of Medicine, Northwest Center for Medical Education, Indiana

4:20 P.M. **Questions and Answers**

ATTENTION WOMEN PHYSICIANS AND MEDICAL STUDENTS

Please join us for dinner on Friday evening at The Italian Pavilion, a fine Italian restaurant, after the CME program.

MSSNY and the Women's Medical Societies of New York State are pleased to host this second annual event to bring women in medicine together for an informal evening of socializing and networking.

The entire CME weekend, and Friday's program in particular, includes presentations on topics that should interest you, regardless of your specialty. Topics such as The Psychotherapy of a Battered Woman; The Doctor As A Defendant (a mock trial); Clinical Dilemmas Associated With Ob-Gyn and Urology; Clinical Dilemmas in Pediatrics (including infant apnea and AIDS in infants and children); and Cocaine Use in the Real World.

Don't let your educational experience end with the lectures.

The Italian Pavilion is one short block away from the New York Hilton and we plan to gather there immediately after the 2–5pm session on Osteoporosis presented by the Women's Medical Societies and the Orthopaedic Surgery Society.

THE ITALIAN PAVILION
24 West 55th Street
New York City

5:00–6:00 pm	Cocktails (cash bar)
6:00 pm	Dinner

To reserve your place for dinner, please send a check in the amount of $40.00, payable to MSSNY, to:

Division of Membership Services
Medical Society of the State of New York
420 Lakeville Road
Lake Success, NY 11042

Questions? Please call MSSNY at (516) 488-6100 or (800) 523-4405, Ext. 236.

Complete and return this form to the Medical Society address as indicated below.

Advance Registration Form

1989 Annual CME Assembly, September 15-16
The New York Hilton, New York City

Name____
PLEASE PRINT LAST FIRST

Address____

City/State/Zip___________________________________

Medical Specialty________________________________

REGISTRATION FEE *Please check categories*

☐ Active Member*	$25.00	☐ Nurse	☐ Lab Technician
☐ Non-Member Physician*	$50.00	☐ Physician Assist.	☐ PT/OT
☐ Allied Profession	$10.00	☐ Dentist	☐ Other______ *Specify*

NO FEE *Please check category*

- ☐ MSSNY Life Member
- ☐ Resident/Intern
- ☐ Medical Students
- ☐ Medical Assist.
- ☐ Speaker
- ☐ Spouse/Family
- ☐ News Media/Medical Writer

REMITTANCE ENCLOSED $__________

* of any state medical society

Please make check payable to:
Medical Society of the State of New York, and return this form with remittance to: Division of Meeting Services/MSSNY, 420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042.

Important: Your ID Badge will be held at the MSSNY Registration Desk located in the Exhibit Hall, on the second floor of the New York Hilton.

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25 mg. Hydrochlorothiazide/50 mg. Triamterene/SKF

Before prescribing, see complete prescribing information in SK&F CO. literature or PDR. The following is a brief summary.

Indications and Usage: *Hypertension or edema in patients who develop hypokalemia on hydrochlorothiazide alone, in patients who require a thiazide diuretic and in whom the development of hypokalemia cannot be risked*

This fixed combination drug is not indicated for the initial therapy of edema or hypertension except in individuals in whom the development of hypokalemia cannot be risked.

'Dyazide' may be used alone or as an adjunct to other antihypertensive drugs, dosage adjustments may be necessary

Contraindications *Concomitant use with other potassium-sparing agents such as spironolactone or amiloride, potassium supplements (except in presence of severe hypokalemia), anuria, acute and chronic renal insufficiencies or significant renal impairment, hypersensitivity to drug or other sulfonamide-derived drugs, preexisting elevated serum potassium concentration*

Warnings *Abnormal elevation of serum potassium levels (greater than or equal to 5 5 mEq/liter) can occur with all potassium-conserving diuretic combinations, including 'Dyazide' Hyperkalemia is more likely to occur in patients with renal impairment, diabetes (even without evidence of renal impairment), elderly or severely ill patients. Since uncorrected hyperkalemia may be fatal, serum potassium levels must be monitored at frequent intervals especially in patients first receiving 'Dyazide', when dosages are changed or with any illness that may influence renal function*

If hyperkalemia is suspected, obtain an ECG and monitor serum potassium If hyperkalemia develops, discontinue 'Dyazide' and initiate thiazide therapy if needed Persistent hyperkalemia may require dialysis Monitor serum electrolytes frequently in patients with mild renal dysfunction and in diabetic patients In patients who may develop respiratory or metabolic acidosis, monitor serum electrolytes and acid/base balance frequently

Precautions *The bioavailability of the hydrochlorothiazide and triamterene components of 'Dyazide' is about 50% of the maximum obtainable with oral therapy Theoretically, a patient transferred from therapy with hydrochlorothiazide with or without triamterene might show an increase in blood pressure, fluid retention, or change in serum potassium Extensive clinical experience with 'Dyazide', however, suggests that these conditions have not been commonly observed in clinical practice (See CLINICAL PHARMACOLOGY) Use thiazides cautiously in patients with impaired hepatic function. They can precipitate coma in patients with severe liver disease, potassium depletion induced by the thiazide may be important in this connection, administer 'Dyazide' cautiously and be alert for such early signs of impending coma as confusion, drowsiness and tremor; if mental confusion increases, discontinue 'Dyazide' for a few days, attention must be given to other factors that may precipitate hepatic coma, such as blood in the gastrointestinal tract or preexisting potassium depletion If patients develop hypokalemia, which is uncommon with 'Dyazide', increase potassium intake (i e , with supplements or potassium-rich foods). If repeat determinations show serum potassium concentrations below 3 0 mEq/L, discontinue 'Dyazide' and initiate potassium chloride supplementation. Institute corrective measures cautiously and monitor serum potassium concentrations frequently, especially in patients receiving digitalis or those with a history of cardiac arrhythmias Diuretics may aggravate existing electrolyte imbalances, especially at high dosages or in patients on salt-restricted diets Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids) Chloride replacement may be required in the treatment of metabolic acidosis. If dilutional hyponatremia develops, restrict water intake In actual salt depletion, initiate sodium chloride replacement Use 'Dyazide' cautiously in patients with a history of renal stone formation*

If hyperkalemia develops when treating for hypokalemia, take corrective measures Also discontinue 'Dyazide' and, if appropriate, substitute a thiazide diuretic until potassium levels return to normal Do periodic BUN and serum creatinine determinations, especially in the elderly and in patients with suspected or confirmed renal insufficiency Serum PBI levels may decrease without signs of thyroid disturbance Discontinue thiazides before conducting parathyroid function tests

Angiotensin-converting enzyme (ACE) inhibitors can elevate serum potassium, use with caution with 'Dyazide' Concurrent use with chlorpropamide may increase the risk of severe hyponatremia A few occurrences of acute renal failure have been reported in patients on 'Dyazide' when treated with indomethacin. Therefore, caution is advised in administering nonsteroidal anti-inflammatory agents with 'Dyazide' Diuretics reduce renal clearance of lithium and increase the risk of lithium toxicity Clinically insignificant reductions in arterial responsiveness to norepinephrine have been reported Thiazides have also been shown to increase the paralyzing effect of nondepolarizing muscle relaxants such as tubocurarine, therefore use cautiously in patients undergoing surgery Monitor electrolytes in patients taking amphotericin B, corticosteroids or corticotropin concomitantly Thiazides may potentiate the action of other antihypertensive drugs The effects of oral anticoagulants may be decreased when used concurrently with hydrochlorothiazide, dosage adjustments may be needed 'Dyazide' may raise the level of blood uric acid, dosage adjustments of antigout medication may be needed to control hyperuricemia and gout The following agents given with triamterene may promote serum potassium accumulation and possibly result in hyperkalemia, especially in patients with renal insufficiency blood from blood bank (may contain up to 30 mEq of potassium per liter of plasma or up to 65 mEq of potassium per liter of whole blood when stored for more than 10 days), low salt milk (may contain up to 60 mEq of potassium per liter), potassium-containing medications (such as parenteral penicillin G potassium), and salt substitutes (most contain substantial amounts of potassium) Exchange resins, such as sodium polystyrene sulfonate, whether administered orally or rectally, reduce serum potassium concentrations by sodium replacement of the potassium, fluid retention may occur in some patients because of the increased sodium intake Chronic or overuse of laxatives may reduce serum potassium concentrations by promoting excessive potassium loss from the intestinal tract, laxatives may interfere with the potassium-retaining effects of triamterene The effectiveness of methenamine may be decreased when used concurrently with hydrochlorothiazide because of alkalinization of the urine 'Dyazide' will interfere with the fluorescent measurement of quinidine

There are no adequate and well-controlled studies in pregnant women This drug should be used during pregnancy only if clearly needed Thiazides and triamterene cross the placental barrier and appear in cord blood The use of thiazides in pregnancy requires weighing the anticipated benefit against possible hazards, including fetal or neonatal jaundice, pancreatitis, thrombocytopenia, and possibly other adverse reactions which have occurred in the adult Thiazides appear, and triamterene may appear, in breast milk If use of the drug is essential, the patient should stop nursing Safety and effectiveness in children have not been established

Adverse Reactions *The serious adverse effects associated with 'Dyazide' have commonly occurred in less than 0.1% of patients treated with this product. Anaphylaxis, rash, urticaria, photosensitivity, cardiac arrhythmias, postural hypotension, diabetes mellitus, hyperkalemia, hyperglycemia, glycosuria, hyperuricemia, hypokalemia, hyponatremia, acidosis, hypochloremia, jaundice and/or liver enzyme abnormalities, pancreatitis, nausea and vomiting, diarrhea, constipation, abdominal pain, acute renal failure, interstitial nephritis, renal stones composed primarily of triamterene, elevated BUN and serum creatinine, abnormal urinary sediment, leukopenia, thrombocytopenia and purpura. megaloblastic anemia, muscle cramps, weakness, fatigue, dizziness, headache, dry mouth, impotence, sialadenitis Thiazides alone have been shown to cause the following additional adverse reactions paresthesias, vertigo, xanthopsia, transient blurred vision, allergic pneumonitis, pulmonary edema, respiratory distress, necrotizing vasculitis, exacerbation of lupus, aplastic anemia, agranulocytosis, hemolytic anemia. In neonates and infants, thrombocytopenia and pancreatitis—rarely, in newborns whose mothers have received thiazides during pregnancy*

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Qualified?

The American Board of Quality Assurance and Utilization Review Physicians (ABQAURP) will be holding a Certification Examination/Lecture Course on October 11-12, 1989 in New York City. ABQAURP identifies its diplomates as experts in the area of QA/UR. Certification leads increased opportunities for career advancement within the field. For further information please contact: ABQAURP - Murdock Professional Center, 1777 Tamiami Trail, Box 12, Dept. 8, Port Charlotte FL 33948.

Get Certified!

(continued from p 7A)

HAWAII

Aug 5–13. **Ninth Annual Comprehensive Fine Needle Biopsy Course.** Westin Hotel, Kauai. Contact: Extended Programs in Medical Education, University of California, Room U-569, San Francisco, CA 94143-0742.

ILLINOIS

Aug 22–29. **33rd Tutorial on Clinical Cytology.** 80 Cat I Credits. Knickerbocker Hotel, Chicago. Contact: 33rd Tutorial on Clinical Cytology, University of Chicago, 5841 S Maryland Ave, HM#449, Chicago, IL 60637. Tel: (312) 702-6569.

MASSACHUSETTS

Aug 4–6. **The Cape Cod Conference on Pediatrics.** The Tara Hyannis Hotel, Hyannis, Cape Cod. Contact: Dr Elliot F. Ellis, PO Box 5720, Jacksonville, FL 33247. Tel: (904) 390-3676.

NORTH CAROLINA

Aug 3–6. **Pediatric Update.** The Grove Park Inn, Asheville. Contact: Debbie Shealy, South Carolina Chapter AAP, PO Box 11188, Columbia, SC 29211. Tel: (803) 798-6207.

WYOMING

Aug 24–26. **Office Gynecology.** 16 Cat I Credits. Grand Teton Lodge, Jackson Hole. Contact: The American College of Obstetricians and Gynecologists, 409 12th St, SW, Washington, DC 120024-2188. Tel: (800) 533-8811.

AROUND THE WORLD

BERMUDA

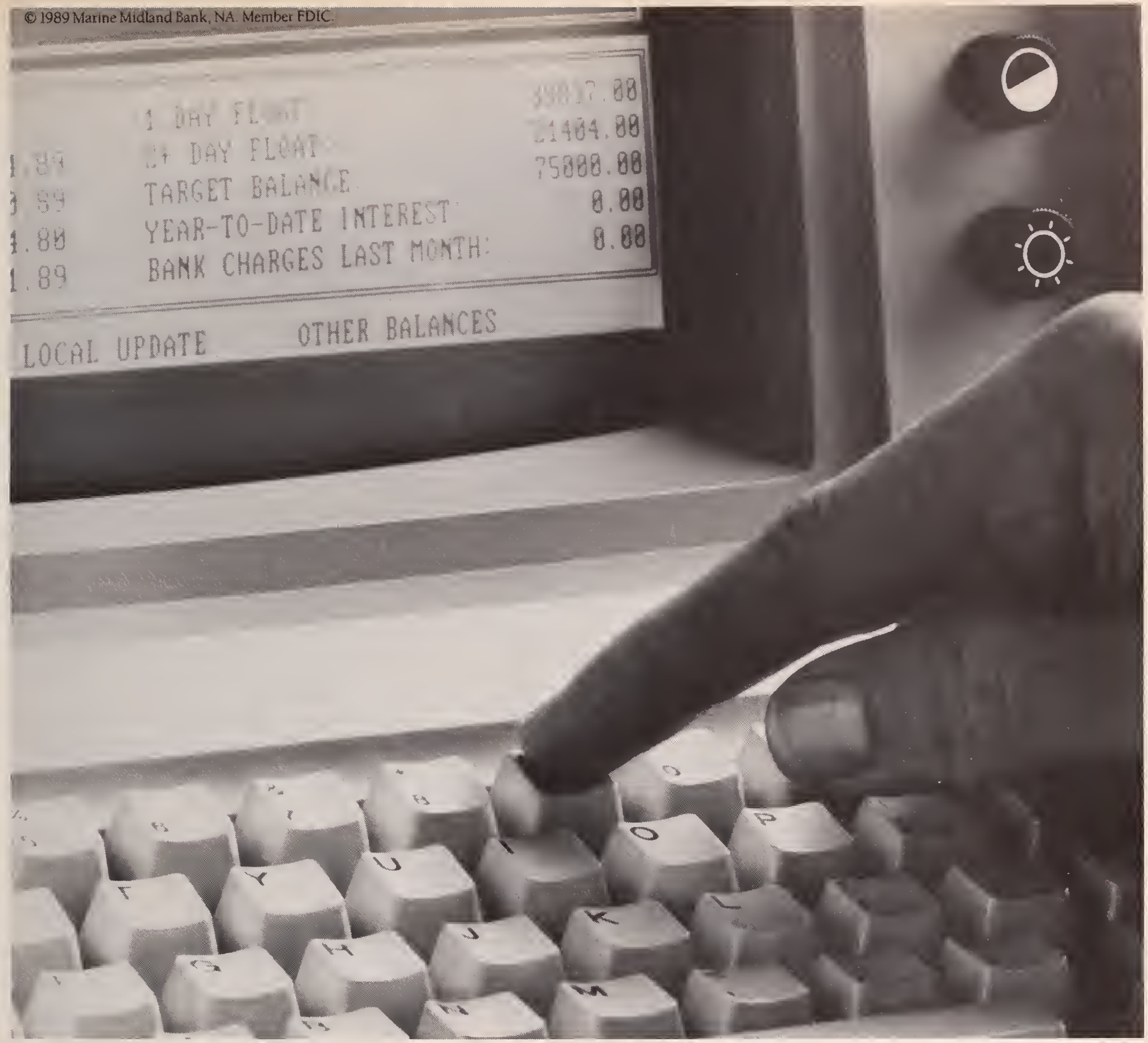
Aug 2–6. **Second Annual Meeting of the Southern Association for Oncology.** Hamilton. Contact: Education Department, Southern Medical Association, 35 Lakeshore Dr, Birmingham, AL 35219-0088. Tel: (205) 945-1840.

Aug 10–12. **Controversies in Gynecologic Surgery.** 16 Cat I Credits. Sonesta Beach Hotel, Southampton. Contact: The American College of Obstetricians and Gynecologists, 409 12th St, SW, Washington, DC 120024-2188. Tel: (800) 533-8811.

CANADA

Aug 13–16. **Nutrition in Health—The Scientific Challenge for the 21st Century.** Montreal Convention Centre, Montreal. Contact: The Columbia/Kenness Team, 1010 Ste Cathereine St W, Suite 645, Montreal, Quebec H3B 1G7. Tel: (514) 874-1833. □

Why me, Mommy?
St. Jude Children's Research Hospital
505 N. Parkway, Memphis, TN 38105



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Index to Advertisers

Beth Israel Medical Center 1A

Classified Advertising 8A, 32A, 34A

Diagnosticdata, Inc. 8A

Eli Lilly and Company .... 2nd Cover

Marine Midland Bank 31A

Maryland Bank, N.A. 3A

Palisades Pharmaceuticals 7A

Roche Laboratories .... 3rd & 4th Cover

SK&F Company 29A, 30A

Wyeth-Ayerst Laboratories 4A, 5A, 6A

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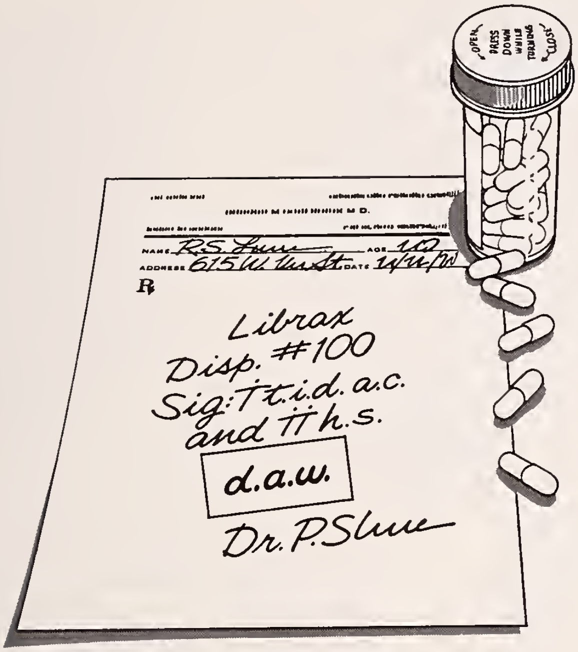

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Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium bromide.

Please consult complete prescribing information, a summary of which follows:

* **Indications:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows: "Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.
Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

> *Usage in Pregnancy:* **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur.

Withdrawal symptoms of the barbiturate type have occurred after discontinuation of benzodiazepines (see Drug Abuse and Dependence).

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established. Inform patients to consult physician before increasing dose or abruptly discontinuing this drug.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

Drug Abuse and Dependence: Withdrawal symptoms similar to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of chlordiazepoxide; more severe seen after excessive doses over extended periods; milder after taking continuously at therapeutic levels for several months. After extended therapy, avoid abrupt discontinuation and taper dosage. Carefully supervise addiction-prone individuals because of predisposition to habituation and dependence.

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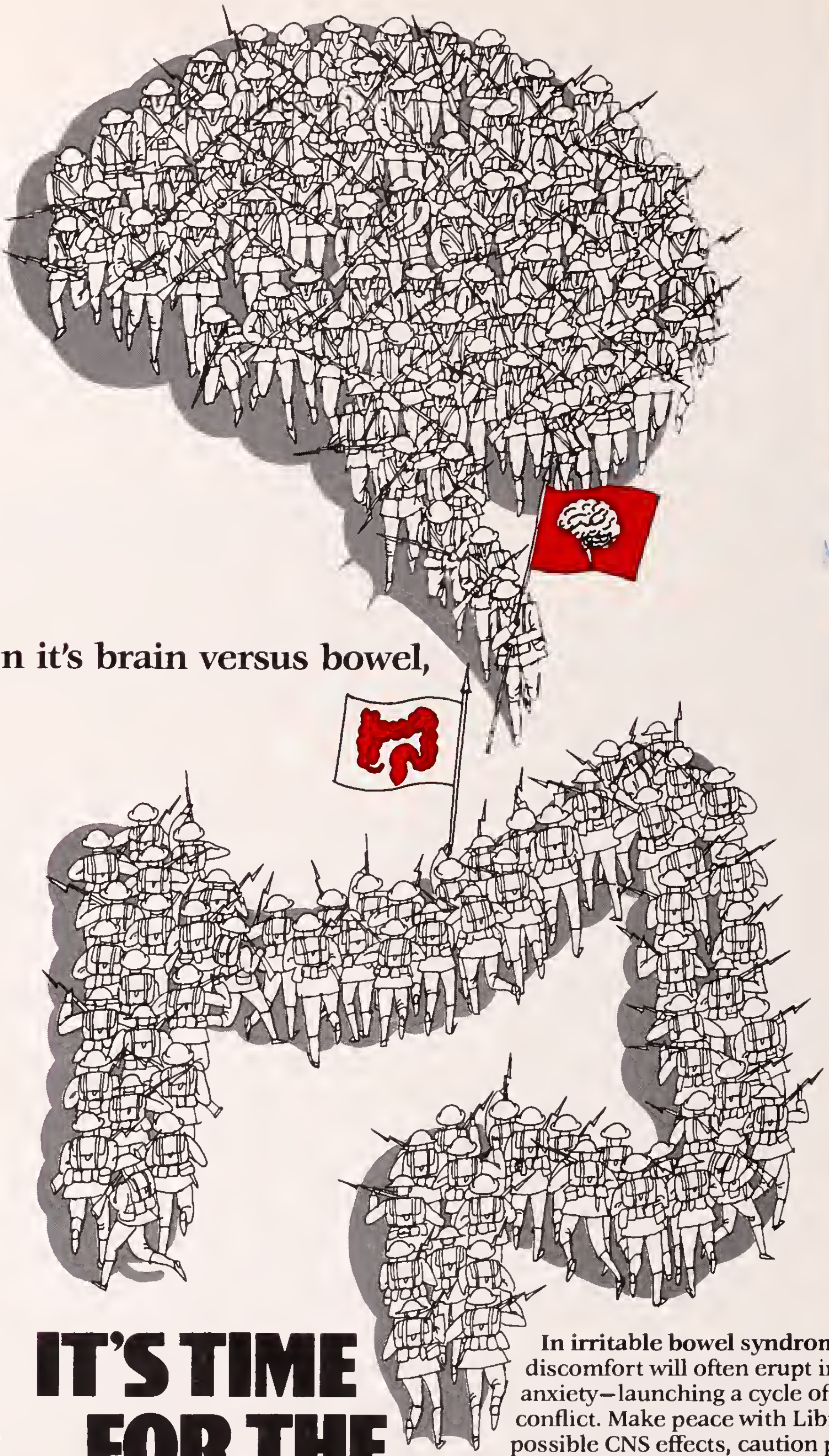

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Please see summary of prescribing information on adjacent page.

NEW YORK STATE JOURNAL OF MEDICINE

AUGUST 1989 • Volume 89, Number 8

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Contents

COMMENTARIES

Continuing competence in the medical profession 443
HENRY A. FERNANDEZ, JD

The impact of technology on the clinical decision making of house staff 444
DAVID B. SEIFER, MD, JOHN BERNARD HENRY, MD

RESEARCH PAPERS

Emergency room visit time: Changes over a 16-year period 446
SUSAN ABRAMOWITZ, PhD; SHARON A. JOY, MBA; ROGER W. YURT, MD

Health, income, and activities of retired physicians 450
M. LAURENS ROWE, MD

SPECIAL ARTICLES

House staff stress 454
STEVEN J. WALERSTEIN, MD; FRED ROSNER, MD; ELEANOR Z. WALLACE, MD

The impact of the new code regulations on postgraduate medical education in New York State 457
STEVEN C. REINER, MD

POSITION PAPER

The education of physicians and the health of New York: Associated Medical Schools of New York position paper on the mission and responsibilities of medical schools and academic health centers in New York State 462

HISTORY

A history of the Public Health Association of New York City 467
FLORENCE KAVALER, MD, MPH, BENJAMIN WAINFELD, MD

Vesical calculus, lithotrity, and Napoleon III 472
JAIN I. LIN, MD

CASE REPORTS

Temporal arteritis and intestinal perforation: Report of three cases 476
BRUCE PATSNER, MD; SYLVAIN PITZELE, MD; WILLIAM J. MANN, MD; EVA CHALAS, MD

Pulmonary oncocytoma 477
MARITESS G. DE JESUS, MD; TUNG P. POON, MD; KUN-YOUNG CHUNG, MD

Common duct stone with associated *Clonorchis sinensis* infection 480
THOMAS JIMENEZ, MD; ANTHONY DiBENEDETTO, MD

Testicular malignancy associated with massive inguinal adenopathy and life threatening hemorrhage 482
STUART M. LICHTMAN, MD; ALLEN D. SEFTEL, MD; MYRON ARLEN, MD; DANIEL BUDMAN, MD; MYRON FREUND, MD; MYRON SUSIN, MD; DONALD FAGELMAN, MD

LETTERS TO THE EDITOR

Himself, Mrs Plucker, and the rites of spring 484
ROBERT J. SILICH, MD

Abuse of the elderly 484
WAYNE A. MYERS, MD

Physician recredentialing 484
DAVID R. REICH, MD

The future of medicine: A resident's perspective 484
ANDREW I. ELKOWITZ, MD

ESSAY REVIEW

Body and soul: Doctors tell all 487
NAOMI R. BLUESTONE, MD

LEADS FROM EPIDEMIOLOGY NOTES 490
BOOK REVIEWS 494
NEWS BRIEFS 498
OBITUARIES 500
MEDICAL MEETINGS AND LECTURES 6A

Annual CME Assembly, September 15–16, 1989, New York Hilton, New York City
(See July issue for Preliminary Program)

ARMY RESERVE MEDICAL PROFILE NO.9

Dr. Holwick outside of hospital where she practices as a civilian traumatologist.

Dr. Holwick in operating room at Letterman Army Medical Center.

JANN L. HOLWICK, M.D.

General and Trauma Surgeon.
Captain, U.S. Army Reserve.

EDUCATION University of Southern California, B.S.; University of California School of Medicine.

RESIDENCY Harbor General Hospital—UCLA Medical Center.

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Brief Summary

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Indications and Usage: Axid is indicated for up to eight weeks for the treatment of active duodenal ulcer. In most patients, the ulcer will heal within four weeks.

Axid is indicated for maintenance therapy for duodenal ulcer patients at a reduced dosage of 150 mg h.s. after healing of an active duodenal ulcer. The consequences of continuous therapy with Axid for longer than one year are not known.

Contraindication: Axid is contraindicated in patients with known hypersensitivity to the drug and should be used with caution in patients with hypersensitivity to other H_2-receptor antagonists.

Precautions: *General* – 1. Symptomatic response to nizatidine therapy does not preclude the presence of gastric malignancy.

2. Because nizatidine is excreted primarily by the kidney, dosage should be reduced in patients with moderate to severe renal insufficiency

3. Pharmacokinetic studies in patients with hepatorenal syndrome have not been done. Part of the dose of nizatidine is metabolized in the liver. In patients with normal renal function and uncomplicated hepatic dysfunction, the disposition of nizatidine is similar to that in normal subjects.

Laboratory Tests – False-positive tests for urobilinogen with Multistix® may occur during therapy with nizatidine.

Drug Interactions – No interactions have been observed between Axid and theophylline, chlordiazepoxide, lorazepam, lidocaine, phenytoin, and warfarin. Axid does not inhibit the cytochrome P-450-linked drug-metabolizing enzyme system; therefore, drug interactions mediated by inhibition of hepatic metabolism are not expected to occur. In patients given very high doses (3,900 mg) of aspirin daily, increases in serum salicylate levels were seen when nizatidine, 150 mg b.i.d., was administered concurrently.

Carcinogenesis, Mutagenesis, Impairment of Fertility – A two-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a two-year study in mice, there was no evidence of a carcinogenic effect in male mice; although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups. The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid.

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, mouse lymphoma assay, chromosome aberration tests, and a micronucleus test.

In a two-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy – Teratogenic Effects – Pregnancy Category C – Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in one fetus and at 50 mg/kg it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in one fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers – Studies conducted in lactating women have shown that <0.1% of the administered oral dose of nizatidine is secreted in human milk in proportion to plasma concentrations. Caution should be exercised when administering nizatidine to a nursing mother.

Pediatric Use – Safety and effectiveness in children have not been established.

Use in Elderly Patients – Ulcer healing rates in elderly patients are similar to those in younger age groups. The incidence rates of adverse events and laboratory test abnormalities are also similar to those seen in other age groups. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Clinical trials of nizatidine included almost 5,000 patients given nizatidine in studies of varying durations. Domestic placebo-controlled trials included over 1,900 patients given nizatidine and over 1,300 given placebo. Among reported adverse events in the domestic placebo-controlled trials, sweating (1% vs 0.2%), urticaria (0.5% vs < 0.01%), and somnolence (2.4% vs 1.3%) were significantly more common in the nizatidine group. A variety of less common events was also reported; it was not possible to determine whether these were caused by nizatidine.

Hepatic – Hepatocellular injury, evidenced by elevated liver enzyme tests (SGOT [AST], SGPT [ALT], or alkaline phosphatase), occurred in some patients and was possibly or probably related to nizatidine. In some cases, there was marked elevation of SGOT, SGPT enzymes (greater than 500 IU/L) and, in a single instance, SGPT was greater than 2,000 IU/L. The overall rate of occurrences of elevated liver enzymes and elevations to three times the upper limit of normal, however, did not significantly differ from the rate of liver enzyme abnormalities in placebo-treated patients. All abnormalities were reversible after discontinuation of Axid.

Cardiovascular – In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in two individuals administered Axid and in three untreated subjects.

CNS – Rare cases of reversible mental confusion have been reported.

Endocrine – Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to Axid. Impotence and decreased libido were reported with equal frequency by patients who received Axid and by those given placebo. Rare reports of gynecomastia occurred.

Hematologic – Fatal thrombocytopenia was reported in a patient who was treated with Axid and another H_2-receptor antagonist. On previous occasions, this patient had experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported.

Integumental – Sweating and urticaria were reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported

Hypersensitivity – As with other H_2-receptor antagonists, rare cases of anaphylaxis following administration of nizatidine have been reported. Because cross-sensitivity in this class of compounds has been observed, H_2-receptor antagonists should not be administered to individuals with a history of previous hypersensitivity to these agents. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

Other – Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine administration have been reported.

Overdosage: Overdoses of Axid have been reported rarely. The following is provided to serve as a guide should such an overdose be encountered.

Signs and Symptoms – There is little clinical experience with overdosage of Axid in humans. Test animals that received large doses of nizatidine have exhibited cholinergic-type effects, including lacrimation, salivation, emesis, miosis, and diarrhea. Single oral doses of 800 mg/kg in dogs and of 1,200 mg/kg in monkeys were not lethal. Intravenous median lethal doses in the rat and mouse were 301 mg/kg and 232 mg/kg respectively.

Treatment – To obtain up-to-date information about the treatment of overdose, a good resource is your certified regional Poison Control Center. Telephone numbers of certified poison control centers are listed in the *Physicians' Desk Reference (PDR)*. In managing overdosage, consider the possibility of multiple drug overdoses, interaction among drugs, and unusual drug kinetics in your patient.

If overdosage occurs, use of activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. Renal dialysis for four to six hours increased plasma clearance.

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Additional information available to the profession on request.

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[1] Data on file, Lilly Research Laboratories.

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Warnings: *Angioedema*: Angioedema of the face, extremities, lips, tongue, glottis, and/or larynx has been reported in patients treated with ACE inhibitors, including VASOTEC. In such cases, VASOTEC should be promptly discontinued and the patient carefully observed until the swelling disappears. In instances where swelling has been confined to the face and lips, the condition has generally resolved without treatment, although antihistamines have been useful in relieving symptoms. Angioedema associated with laryngeal edema may be fatal. **Where there is involvement of the tongue, glottis, or larynx likely to cause airway obstruction, appropriate therapy, e.g., subcutaneous epinephrine solution 1:1000 (0.3 mL to 0.5 mL), should be promptly administered.** (See ADVERSE REACTIONS.)

Hypotension: Excessive hypotension is rare in uncomplicated hypertensive patients treated with VASOTEC alone. Heart failure patients given VASOTEC commonly have some reduction in blood pressure, especially with the first dose, but discontinuation of therapy for continuing symptomatic hypotension usually is not necessary when dosing instructions are followed, caution should be observed when initiating therapy. (See DOSAGE AND ADMINISTRATION.) Patients at risk for excessive hypotension, sometimes associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death, include those with the following conditions or characteristics: heart failure, hyponatremia, high-dose diuretic therapy, recent intensive diuresis or increase in diuretic dose, renal dialysis, or severe volume and/or salt depletion of any etiology. It may be advisable to eliminate the diuretic (except in heart failure patients), reduce the diuretic dose, or increase salt intake cautiously before initiating therapy with VASOTEC in patients at risk for excessive hypotension who are able to tolerate such adjustments. (See PRECAUTIONS, *Drug Interactions* and ADVERSE REACTIONS.) In patients at risk for excessive hypotension, therapy should be started under very close medical supervision and such patients should be followed closely for the first two weeks of treatment and whenever the dose of enalapril and/or diuretic is increased. Similar considerations may apply to patients with ischemic heart disease or cardiovascular disease in whom an excessive fall in blood pressure could result in a myocardial infarction or cerebrovascular accident.

If excessive hypotension occurs, the patient should be placed in supine position and, if necessary, receive an intravenous infusion of normal saline. A transient hypotensive response is not a contraindication to further doses of VASOTEC, which usually can be given without difficulty once the blood pressure has stabilized. If symptomatic hypotension develops, a dose reduction or discontinuation of VASOTEC or concomitant diuretic may be necessary.

Neutropenia/Agranulocytosis: Another ACE inhibitor, captopril, has been shown to cause agranulocytosis and bone marrow depression, rarely in uncomplicated patients but more frequently in patients with renal impairment, especially if they also have a collagen vascular disease. Available data from clinical trials of enalapril are insufficient to show that enalapril does not cause agranulocytosis at similar rates. Foreign marketing experience has revealed several cases of neutropenia or agranulocytosis in which a causal relationship to enalapril cannot be excluded. Periodic monitoring of white blood cell counts in patients with collagen vascular disease and renal disease should be considered.

Precautions: *General: Impaired Renal Function:* As a consequence of inhibiting the renin-angiotensin-aldosterone system, changes in renal function may be anticipated in susceptible individuals. In patients with severe heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with ACE inhibitors, including VASOTEC, may be associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death.

In clinical studies in hypertensive patients with unilateral or bilateral renal artery stenosis, increases in blood urea nitrogen and serum creatinine were observed in 20% of patients. These increases were almost always reversible upon discontinuation of enalapril and/or diuretic therapy. In such patients, renal function should be monitored during the first few weeks of therapy.

Some patients with hypertension or heart failure with no apparent preexisting renal vascular disease have developed increases in blood urea and serum creatinine, usually minor and transient, especially when VASOTEC has been given concomitantly with a diuretic. This is more likely to occur in patients with preexisting renal impairment. Dosage reduction and/or discontinuation of the diuretic and/or VASOTEC may be required.

Evaluation of patients with hypertension or heart failure should always include assessment of renal function. (See DOSAGE AND ADMINISTRATION.)

Hyperkalemia: Elevated serum potassium (> 5.7 mEq/L) was observed in approximately 1% of hypertensive patients in clinical trials. In most cases these were isolated values which resolved despite continued therapy. Hyperkalemia was a cause of discontinuation of therapy in 0.28% of hypertensive patients. In clinical trials in heart failure, hyperkalemia was observed in 3.8% of patients, but was not a cause for discontinuation.

Risk factors for the development of hyperkalemia include renal insufficiency, diabetes mellitus, and the concomitant use of potassium-sparing diuretics, potassium supplements, and/or potassium-containing salt substitutes, which should be used cautiously, if at all, with VASOTEC. (See *Drug Interactions*.)

Surgery/Anesthesia: In patients undergoing major surgery or during anesthesia with agents that produce hypotension, enalapril may block angiotensin II formation secondary to compensatory renin release. If hypotension occurs and is considered to be due to this mechanism, it can be corrected by volume expansion.

Information for Patients:

Angioedema. Angioedema, including laryngeal edema, may occur especially following the first dose of enalapril. Patients should be so advised and told to report immediately any signs or symptoms suggesting angioedema (swelling of face, extremities, eyes, lips, tongue, difficulty in swallowing or breathing) and to take no more drug until they have consulted with the prescribing physician.

Hypotension: Patients should be cautioned to report lightheadedness especially during the first few days of therapy. If actual syncope occurs, the patients should be told to discontinue the drug until they have consulted with the prescribing physician.

All patients should be cautioned that excessive perspiration and dehydration may lead to an excessive fall in blood pressure because of reduction in fluid volume. Other causes of volume depletion such as vomiting or diarrhea may also lead to a fall in blood pressure, patients should be advised to consult with the physician.

Hyperkalemia: Patients should be told not to use salt substitutes containing potassium without consulting their physician.

Neutropenia: Patients should be told to report promptly any indication of infection (e.g., sore throat, fever) which may be a sign of neutropenia.

NOTE: As with many other drugs, certain advice to patients being treated with enalapril is warranted. This information is intended to aid in the safe and effective use of this medication. It is not a disclosure of all possible adverse or intended effects.

Drug Interactions

Hypotension: Patients on Diuretic Therapy: Patients on diuretics and especially those in whom diuretic therapy was recently instituted may occasionally experience an excessive reduction of blood pressure after initiation of therapy with enalapril. The possibility of hypotensive effects with enalapril can be minimized by either discontinuing the diuretic or increasing the salt intake prior to initiation of treatment with enalapril. If it is necessary to continue the diuretic, provide close medical supervision after the initial dose for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and DOSAGE AND ADMINISTRATION.)

Agents Causing Renin Release: The antihypertensive effect of VASOTEC is augmented by antihypertensive agents that cause renin release (e.g., diuretics).

Other Cardiovascular Agents: VASOTEC has been used concomitantly with beta-adrenergic-blocking agents, methyldopa, nitrates, calcium-blocking agents, hydralazine, prazosin, and digoxin without evidence of clinically significant adverse interactions.

Agents Increasing Serum Potassium: VASOTEC attenuates potassium loss caused by thiazide-type diuretics. Potassium-sparing diuretics (e.g., spironolactone, triamterene, or amiloride), potassium supplements, or potassium-containing salt substitutes may lead to significant increases in serum potassium. Therefore, if concomitant use of these agents is indicated because of demonstrated hypokalemia, they should be used with caution and with frequent monitoring of serum potassium. Potassium-sparing agents should generally not be used in patients with heart failure receiving VASOTEC.

Lithium. A few cases of lithium toxicity have been reported in patients receiving concomitant VASOTEC and lithium and were reversible upon discontinuation of both drugs. Although a causal relationship has not been established, it is recommended that caution be exercised when lithium is used concomitantly with VASOTEC and serum lithium levels should be monitored frequently.

Pregnancy—Category C. There was no fetotoxicity or teratogenicity in rats treated with up to 200 mg/kg/day of enalapril (333 times the maximum human dose). Fetotoxicity, expressed as a decrease in average fetal weight, occurred in rats given 1200 mg/kg/day of enalapril but did not occur when these animals were supplemented with saline. Enalapril was not teratogenic in rabbits. However, maternal and fetal toxicity occurred in some rabbits at doses of 1 mg/kg/day or more. Saline supplementation prevented the maternal and fetal toxicity seen at doses of 3 and 10 mg/kg/day, but not at 30 mg/kg/day (50 times the maximum human dose).

Radioactivity was found to cross the placenta following administration of labeled enalapril to pregnant hamsters.

There are no adequate and well-controlled studies of enalapril in pregnant women. However, data are available that show enalapril crosses the human placenta. Because the risk of fetal toxicity with the use of ACE inhibitors has not been clearly defined, VASOTEC® (Enalapril Maleate, MSD) should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Postmarketing experience with all ACE inhibitors thus far suggests the following with regard to pregnancy outcome. Inadvertent exposure limited to the first trimester of pregnancy has not been reported to affect fetal outcome adversely. Fetal exposure during the second and third trimesters of pregnancy has been associated with fetal and neonatal morbidity and mortality.

When ACE inhibitors are used during the later stages of pregnancy, there have been reports of hypotension and decreased renal perfusion in the newborn. Oligohydramnios in the mother has also been reported, presumably representing decreased renal function in the fetus. Infants exposed *in utero* to ACE inhibitors should be closely observed for hypotension, oliguria, and hyperkalemia. If oliguria occurs, attention should be directed toward support of blood pressure and renal perfusion with the administration of fluids and pressors as appropriate. Problems associated with prematurity such as patent ductus arteriosus have occurred in association with maternal use of ACE inhibitors, but it is not clear whether they are related to ACE inhibition, maternal hypertension, or the underlying prematurity.

Nursing Mothers. Milk in lactating rats contains radioactivity following administration of ^{14}C enalapril maleate. It is not known whether this drug is secreted in human milk. Because many drugs are secreted in human milk, caution should be exercised when VASOTEC is given to a nursing mother.

Pediatric Use: Safety and effectiveness in children have not been established.

Adverse Reactions: VASOTEC has been evaluated for safety in more than 10,000 patients, including over 1000 patients treated for one year or more. VASOTEC has been found to be generally well tolerated in controlled clinical trials involving 2987 patients.

HYPERTENSION: The most frequent clinical adverse experiences in controlled trials were: headache (5.2%), dizziness (4.3%), and fatigue (3%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in controlled clinical trials were: diarrhea (1.4%), nausea (1.4%), rash (1.4%), cough (1.3%), orthostatic effects (1.2%), and asthenia (1.1%).

HEART FAILURE: The most frequent clinical adverse experiences in both controlled and uncontrolled trials were: dizziness (7.9%), hypotension (6.7%), orthostatic effects (2.2%), syncope (2.2%), cough (2.2%), chest pain (2.1%), and diarrhea (2.1%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in both controlled and uncontrolled clinical trials were: fatigue (1.8%), headache (1.8%), abdominal pain (1.6%), asthenia (1.6%), orthostatic hypotension (1.6%), vertigo (1.6%), angina pectoris (1.5%), nausea (1.3%), vomiting (1.3%), bronchitis (1.3%), dyspnea (1.3%), urinary tract infection (1.3%), rash (1.3%), and myocardial infarction (1.2%).

Other serious clinical adverse experiences occurring since the drug was marketed or adverse experiences occurring in 0.5% to 1% of patients with hypertension or heart failure in clinical trials in order of decreasing severity within each category:

Cardiovascular: Cardiac arrest, myocardial infarction or cerebrovascular accident, possibly secondary to excessive hypotension in high-risk patients (see WARNINGS, *Hypotension*), cardiac arrest, pulmonary embolism and infarction; rhythm disturbances, atrial fibrillation, palpitation.

Digestive: Ileus, pancreatitis, hepatitis or cholestatic jaundice, melena, anorexia, dyspepsia, constipation, glossitis.

Nervous/Psychiatric. Depression, confusion, ataxia, somnolence, insomnia, nervousness, paresthesia.

Urogenital. Renal failure, oliguria, renal dysfunction (see PRECAUTIONS and DOSAGE AND ADMINISTRATION).

Respiratory: Bronchospasm, rhinorrhea, asthma, upper respiratory infection.

Skin. Herpes zoster, pruritus, alopecia, flushing, photosensitivity.

Other. Vasculitis, muscle cramps, hyperhidrosis, impotence, blurred vision, taste alteration, tinnitus.

A symptom complex has been reported which may include fever, myalgia, and arthralgia, an elevated erythrocyte sedimentation rate may be present. Rash or other dermatologic manifestations may occur. These symptoms have disappeared after discontinuation of therapy.

Angioedema: Angioedema has been reported in patients receiving VASOTEC (0.2%). Angioedema associated with laryngeal edema may be fatal. If angioedema of the face, extremities, lips, tongue, glottis, and/or larynx occurs, treatment with VASOTEC should be discontinued and appropriate therapy instituted immediately. (See WARNINGS.)

Hypotension: In the hypertensive patients, hypotension occurred in 0.9% and syncope occurred in 0.5% of patients following the initial dose or during extended therapy. Hypotension or syncope was a cause for discontinuation of therapy in 0.1% of hypertensive patients. In heart failure patients, hypotension occurred in 6.7% and syncope occurred in 2.2% of patients. Hypotension or syncope was a cause for discontinuation of therapy in 1.9% of patients with heart failure. (See WARNINGS.)

Clinical Laboratory Test Findings.

Serum Electrolytes. Hyperkalemia (see PRECAUTIONS), hyponatremia.

Creatinine, Blood Urea Nitrogen. In controlled clinical trials, minor increases in blood urea nitrogen and serum creatinine, reversible upon discontinuation of therapy, were observed in about 0.2% of patients with essential hypertension treated with VASOTEC alone. Increases are more likely to occur in patients receiving concomitant diuretics or in patients with renal artery stenosis. (See PRECAUTIONS.) In patients with heart failure who were also receiving diuretics with or without digitalis, increases in blood urea nitrogen or serum creatinine, usually reversible upon discontinuation of VASOTEC and/or other concomitant diuretic therapy, were observed in about 11% of patients. Increases in blood urea nitrogen or creatinine were a cause for discontinuation in 1.2% of patients.

Hemoglobin and Hematocrit: Small decreases in hemoglobin and hematocrit (mean decreases of approximately 0.3 g % and 1.0 vol %, respectively) occur frequently in either hypertension or heart failure patients treated with VASOTEC but are rarely of clinical importance unless another cause of anemia coexists. In clinical trials, less than 0.1% of patients discontinued therapy due to anemia.

Other (Causal Relationship Unknown): In marketing experience, rare cases of neutropenia, thrombocytopenia, and bone marrow depression have been reported.

Liver Function Tests. Elevations of liver enzymes and/or serum bilirubin have occurred.

Dosage and Administration: *Hypertension*: In patients who are currently being treated with a diuretic, symptomatic hypotension occasionally may occur following the initial dose of VASOTEC. The diuretic should, if possible, be discontinued for two to three days before beginning therapy with VASOTEC to reduce the likelihood of hypotension. (See WARNINGS.) If the patient's blood pressure is not controlled with VASOTEC alone, diuretic therapy may be resumed.

If the diuretic cannot be discontinued, an initial dose of 2.5 mg should be used under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, *Drug Interactions*.)

The recommended initial dose in patients not on diuretics is 5 mg once a day. Dosage should be adjusted according to blood pressure response. The usual dosage range is 10 to 40 mg per day administered in a single dose or in two divided doses. In some patients treated once daily, the antihypertensive effect may diminish toward the end of the dosing interval. In such patients, an increase in dosage or twice-daily administration should be considered. If blood pressure is not controlled with VASOTEC alone, a diuretic may be added.

Concomitant administration of VASOTEC with potassium supplements, potassium salt substitutes, or potassium-sparing diuretics may lead to increases of serum potassium (see PRECAUTIONS).

Dosage Adjustment in Hypertensive Patients with Renal Impairment. The usual dose of enalapril is recommended for patients with a creatinine clearance >30 mL/min (serum creatinine of up to approximately 3 mg/dL). For patients with creatinine clearance ≤30 mL/min (serum creatinine ≥3 mg/dL), the first dose is 2.5 mg once daily. The dosage may be titrated upward until blood pressure is controlled or to a maximum of 40 mg daily.

Heart Failure. VASOTEC is indicated as adjunctive therapy with diuretics and digitalis. The recommended starting dose is 2.5 mg once or twice daily. After the initial dose of VASOTEC, the patient should be observed under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, *Drug Interactions*.) If possible, the dose of the diuretic should be reduced, which may diminish the likelihood of hypotension. The appearance of hypotension after the initial dose of VASOTEC does not preclude subsequent careful dose titration with the drug, following effective management of the hypotension. The usual therapeutic dosing range for the treatment of heart failure is 5 to 20 mg daily given in two divided doses. The maximum daily dose is 40 mg. Once-daily dosing has been effective in a controlled study, but nearly all patients in this study were given 40 mg, the maximum recommended daily dose, and there has been much more experience with twice-daily dosing. In addition, in a placebo-controlled study which demonstrated reduced mortality in patients with severe heart failure (NYHA Class IV), patients were treated with 2.5 to 40 mg per day of VASOTEC, almost always administered in two divided doses. (See CLINICAL PHARMACOLOGY, *Pharmacodynamics and Clinical Effects*.) Dosage may be adjusted depending upon clinical or hemodynamic response. (See WARNINGS.)

Dosage Adjustment in Heart Failure Patients with Renal Impairment or Hyponatremia. In heart failure patients with hyponatremia (serum sodium <130 mEq/L) or with serum creatinine >1.6 mg/dL, therapy should be initiated at 2.5 mg daily under close medical supervision. (See DOSAGE AND ADMINISTRATION, *Heart Failure*, WARNINGS, and PRECAUTIONS, *Drug Interactions*.) The dose may be increased to 2.5 mg b.i.d., then 5 mg b.i.d. and higher as needed, usually at intervals of four days or more, if at the time of dosage adjustment there is not excessive hypotension or significant deterioration of renal function. The maximum daily dose is 40 mg.

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References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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SEPTEMBER 1989

AROUND THE STATE

BRONX

Sept 27. **The Medicolegal Ramifications of Police Stress.** Lehman College. Contact: Mark L. Taff, MD, President, New York Society of Forensic Sciences, 130 Gold Pl, Malverne, NY 11564. Tel: (516) 887-4691.

COOPERSTOWN

Sept 13. **Update on Nutrition.** 3 Cat I Credits. Basset Hall Conference Center. Contact: Charlotte L. Hoag, Medical Education Coordinator, Office of Medical Education, The Mary Imogene Bassett Hospital, One Atwell Rd, Cooperstown, NY 13326-1394. Tel: (607) 547-3926.

MANHATTAN

Sept 15–17. **Introduction to Clinical Hypnosis Workshop.** Mt Sinai Hospital. Contact: The Society for Clinical and Experimental Hypnosis, 128-A Kings Park Dr, Liverpool, NY 13090. Tel: (315) 652-7299.

Sept 20. **Ninth Annual Cosmetology Seminar.** Beth Israel Medical Center. Contact: Beverley R. Baptiste, Medical Education Office, Beth Israel Medical Center, First Ave at 16th St, New York, NY 10003. Tel: (212) 420-2000.

Sept 21–23. **Multidisciplinary Perspectives on Menopause.** Sheraton Centre Hotel. Contact: Conference Director, The New York Academy of Sciences, 2 E 63rd St, New York, NY 10021. Tel: (212) 838-0230.

(*continued on p 15A*)

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Medical Society of the State of New York

ANNUAL CME ASSEMBLY

CASE MANAGEMENT & CLINICAL CARE DILEMMAS

September 15–16, 1989

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The theme of the CME sessions will be "Case Management and Clinical Care Dilemmas." The chosen subjects are varied and topical, e.g., the battered woman syndrome (Psychiatry)—child abuse (Pediatrics/Forensic Medicine)—cocaine (Substance Abuse)—osteoporosis (American Medical Women's Assoc./Orthopaedic Surgery)—malpractice (Mock Trial, Medical Liability Mutual Insurance Co.) are but a few. . . . Our programs are designed to be interesting and informative.

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AWARDS TO BE GIVEN AT THE CME ASSEMBLY

Suiter Lectureship presented during the Internal Medicine Program on Saturday, 9/16. Suiter Lectureship is given for a non-surgical subject. The recipient of this award will be announced.

Albert F. R. Andresen Memorial Lecture presented during the Gastroenterology/Colon and Rectal Surgery Program on Friday, 9/15. The Albert F. R. Andresen Memorial Lecture is for a lecture on Gastroenterology. Adrian J. Greenstein, M.D., Professor of Surgery, Mount Sinai School of Medicine, NYC will receive this award.

Lucien Howe Prize presented during the Neurology/Ophthalmology Program on Saturday, 9/16. Lucien Howe Prize is given for the best original contribution to our knowledge of some branch of surgery, preferably ophthalmology. Myles Behrens, M.D. of Columbia University, NYC will be the recipient of this award.

1989 ANNUAL CME ASSEMBLY

Medical Society of the State of New York
The New York Hilton, New York City
September 15–16, 1989

CASE MANAGEMENT AND CLINICAL CARE DILEMMAS

FRIDAY, SEPTEMBER 15

8:30 a.m.–9:30 a.m.	Medical Technology, *Nassau A*
9:30 a.m.–12:30 p.m.	Gastroenterology/Colon & Rectal Surgery, *Sutton Center*
	Computers in Medicine, *Nassau A*
	Occupational & Environmental Health/Family Practice, *Murray Hill B*
	Physical Medicine & Rehabilitation/Neurology, *Murray Hill A*
	Medical Liability Mutual Insurance Company, *Sutton South*
2:00 p.m.–5:00 p.m.	Chest Diseases/Radiology, *Sutton Center*
	Cardiovascular Diseases, *Sutton South*
	Women's Medical Societies of New York State/Orthopaedic Surgery Society, *Murray Hill A*
	Neurology/Ophthalmology, *Nassau A*
	Neurosurgery, *Murray Hill B*
	Emergency Medicine/Critical Care, *Nassau B*

SATURDAY, SEPTEMBER 16

8:30 a.m.–9:30 a.m.	Medical Technology, *Murray Hill B*
9:30 a.m.–12:30 p.m.	Substance Abuse, *Sutton North*
	Internal Medicine (All Day), *Sutton Center*
	Neurology/Ophthalmology (All Day), *Beekman Parlor*
	Psychiatry (All Day), *Nassau B*
	Obstetrics & Gynecology/Urology, *Murray Hill A*
	General Surgery, *Sutton South*
	Dermatology/Plastic & Reconstructive Surgery (All Day), *Nassau A*
2:00 p.m.–5:00 p.m.	Internal Medicine (All Day), *Sutton Center*
	Neurology/Ophthalmology (All Day), *Beekman Parlor*
	Psychiatry (All Day), *Nassau B*
	Pediatrics/Forensic Medicine, *Sutton North*
	Otolaryngology/Neurosurgery/Plastic Surgery, *Sutton South*
	Dermatology/Plastic & Reconstructive Surgery (All Day), *Nassau A*

CME CREDITS: The Medical Society of the State of New York (MSSNY) is accredited by the Accreditation Council for Continuing Medical Education to sponsor CME for physicians. MSSNY designates all CME activities in this CME Assembly as meeting the criteria for Category 1 of the AMA, PRA. The credits are enumerated for each session. Those programs approved for credit by the American Academy of Family Physicians (AAFP) are so designated.

□

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1989 Annual CME Assembly, September 15-16
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NEW YORK STATE
JOURNAL OF MEDICINE

August 1989 • Volume 89, Number 8

COMMENTARIES

Continuing competence in the medical profession

The continued competence of professionals, especially physicians, has been of paramount importance to the New York State Board of Regents, the New York State Education Department, and the Boards for the Professions. A variety of concerns have been expressed about the competence of licensed physicians. Several reports have suggested that the public needs an extra measure of certainty that physicians are competent, although there is no existing research or verifiable data to demonstrate this need.

The only quantifiable data regarding the extent of physician "incompetence" (however it may be defined) is derived from the New York State Department of Health's records of actions taken against physicians. According to information provided by the Office of Professional Medical Conduct, disciplinary actions were taken against 958 physicians in New York State for several categories of misconduct during the period 1983–1988. These included impairment; sexual abuse, negligence, incompetence, or fraudulent practice; improper drug dispensing; Medicaid, Medicare, or insurance fraud; and "other." Of the 958 cases, only 13.6% (130) involved negligence, incompetence, or fraudulent practice. The Office of Professional Medical Conduct did not segregate the number of cases according to charges of negligence, incompetence, or fraudulent practice but it appears that the number of cases involving incompetence are probably in the 4–6% range.

We do not know, however, if charges of incompetence involve a single act or if charged physicians exhibit an identifiable pattern of incompetent acts. In many cases our perceptions of the pervasiveness of incompetence are based on cases sensationalized in the media or our exposure to exceptional cases.

The malpractice insurance crisis has been identified as further evidence that the competency of physicians is in question. In the only study of its kind, the Minnesota Department of Commerce examined virtually 100% of all malpractice claims filed between 1982 and 1987 in Minnesota, North Dakota, and South Dakota. Between 1982 and 1987 physicians experienced medical malpractice insurance premium increases of approximately 300%. Yet the report concluded that throughout the five-year period, the number of claims, the severity of the injuries claimed, and the actual payment in response to the claims *had not* materially increased.[1]

In 1986 Governor Mario Cuomo of New York announced his intention of requiring physicians in New York State to have their credentials periodically recertified in order to continue their practice of medicine. He appointed a committee (the New York State Advisory Committee on Physician Recredentialing) in the spring of 1987 to prepare recommendations, establish criteria, and propose a system for recredentialing physicians. In January 1988 the committee issued a draft report.[2]

The report concluded that the goal of recredentialing is the prevention of medical errors. Recredentialing, as proposed, is not designed to determine a physician's knowledge of all aspects of medicine, but rather to determine whether his or her current practice meets appropriate standards. The report stressed that recredentialing for appropriate standards of medical practice is not synonymous with certification of medical competence. It also stated that medical competence cannot be precisely defined and that there are no tests of continuing competence which have been validated.

The report distinguished between physicians who have hospital staff privileges and physicians whose practice is largely or exclusively limited to the office or some other ambulatory facility and who rarely care for patients in a hospital. It proposed three methods for recredentialing all physicians in patient care as follows: specialty board certification (or recertification) within a nine-year period of recredentialing; cognitive and clinical problem solving tests (SPEX examination, CBX Simulation Model, and a practice oriented examination) within the same period; or either hospital staff peer review for physicians with significant inpatient care activities, or an evaluation of office records for office based physicians. Two options were made available to physicians who hold administrative positions, are employed by government or business, or are retired. The options allow them to elect to be placed on an inactive list or to be recredentialed through certification or recertification by a specialty board or by passing a cognitive and problem solving examination.

The report outlined three results of the recredentialing process: approval of credentials and renewal of the medical license, issuance of time limited licenses due to limited deficiencies, and referral to the Office of Professional Medical Conduct in the event that material deficiencies, delinquencies, inadequate knowledge, or misconduct are disclosed.

The committee's recredentialing proposals may include requirements restricting the practice of physicians that are inconsistent with sections 6501, 6502, and 6503 of the Education Law.[3] These sections provide that a license is permanent and entitles the licensee to practice the profession. Registration requirements do not include any qualitative requirements. Any attempt to restrict the practice of licensees and add recredentialing requirements by rule or regulation may be found to exceed the scope permitted for regulation.

The Hospital Association of New York State has concluded that legislation is necessary to enact these requirements.[4] Dr Alfred Gellhorn, co-chairman of the committee, also noted during a recent national planning conference on the identification and treatment of physicians with clinical deficiencies, that the legislature may have to approve the committee's proposed program before it may be implemented.

Ideally, a method proposed for measuring the dimensions of physician competence (eg, reexamination, record audit, specialty recertification) should be simultaneously valid, reliable, and cost effective. In a study of such assessment mechanisms in Michigan, the Council of the Michigan State Medical Society concluded that no mechanism or combination of mechanisms met the criteria for meaningful measurement.[5] The least costly approaches had the disadvantages of little or no validity. The more complex approaches, such as computer simulations and multimedia approaches used by specialty boards for recertification, had more validity and reliability but are very costly and time consuming.

Although there are many methods proposed at this point, the best direction in which to proceed is not yet clear. However, in selecting a method to promote physician competence, the following points should be considered:

The primary goal should be the protection of the public. The public interest requires that a continuing competence program contain a combination of incentives and sanctions whose primary purpose is to protect the public without restricting access to medical care, without unjustifiably increasing costs, and without imposing unnecessary constraints on professional judgment.

Continuing competence should be definable and measurable. Most definitions of physician competence include at least three dimensions: factual knowledge and understanding relating to the physician's functions; abilities such as communication skills, technical skills, self-discipline, work habits, professional attitudes; and clinical judgment for appropriately applying knowledge, synthesizing, and testing hypotheses.

The rights of both physicians and patients should be protected.

Resource constraints should be recognized. Total costs would include administrative costs, costs paid by the practitioners, costs of the development of educational and remedial programs, costs to consumers in the form of increased prices or reduced access to medical care, and costs associated with defensive medical practice.

Continuing competence programs should be accountable. New regulations that are impossible to enforce, or that cannot be related to specific outcome measures, do not contribute to the public interest.

Continuing competence programs should be credible and acceptable to physicians, consumers, employers, third party payers, and regulatory agencies.

HENRY A. FERNANDEZ, JD
Deputy Commissioner for the Professions
The State Education Department
The University of the State of New York
Albany, NY 12230

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1. Hatch MA: *Medical Malpractice Claim Study 1982–1987.* St Paul, Minn, Department of Commerce, State of Minnesota, 1988 (unpublished).
2. *Report of the New York State Advisory Committee on Physician Recredentialing. Phase One: General Principles, Proposed Process, Recommendation.* Albany, NY, New York State Department of Health, 1988.
3. *Education Law, McKinney's Consolidated Laws of New York,* Book 16. St Paul, Minn, West Publishing Co, 1985, pp 247–252.
4. Position statement of the Hospital Association of New York State on the Report of the New York State Advisory Committee on Physician Recredentialing. *NY State J Med* 1989; 89:223–225.
5. Paper on Physician Competency. Council of the Michigan State Medical Society, East Lansing, MI, Michigan State Medical Society, 1982.

The impact of technology on the clinical decision making of house staff

In July 1989 new regulations governing graduate medical education were implemented in New York State.[1] How these requirements will affect the impact of technology on clinical decision making is an issue that should be addressed. The final result may be an exacerbation of the already described "cascade effect"[2] of ordering tests in patient care.

Clinicians are trained to be effective "problem solvers" who can make patient management decisions with compassion. As medicine progresses and technology becomes more sophisticated, medical decision making becomes more complex. With this complexity, physicians-in-training have become increasingly tempted to place over-reliance on and on occasion to be misled by information derived from technology.[3-5] Two hypothetical examples illustrate this impression.

Consider the 50-year-old smoker who has recently undergone a cholecystectomy and who becomes dyspneic, tachypneic, and complains of chest pain. An electrocardiogram is unremarkable. Arterial blood gas study shows a PaO_2 of 70 mm Hg. The ventilation perfusion scan is read as "low probability for pulmonary embolism." The chest pain and dyspnea subside, and the patient is eventually discharged home without medication. Several days later he dies. An autopsy shows evidence of deep venous thrombosis and multiple pulmonary emboli. More confidence in the clinical findings might have changed the outcome.

Another example is that of a young woman who has been trying for years to become pregnant and who arrives in the emergency room with a history of being two weeks late for her menses. A beta-human chorionic gonadotropin (HCG) test is positive and pelvic ultrasound reveals a possible gestational sac without a fetal pole being identified. The patient complains of some vaginal spotting and intermittent cramping pain. After a lengthy discussion with the patient, the physicians decide to hospitalize her for observation and serial serum beta-HCGs. Several days later she undergoes laparoscopy because the "doubling time" of her beta-HCG is not appropriate for an intrauterine pregnancy. At laparoscopy, she is noted to have a hemoperitoneum and a ruptured ectopic pregnancy. A salpingectomy is performed. Perhaps if laparoscopy had been performed at the time of admission, based on clinical judgment, an unruptured ectopic pregnancy could have been detected and conservative surgery preserving the remainder of the fallopian tube could have taken place.

In each example cited the outcomes were less than optimal. These scenarios, although uncommon, do occur in busy clinical services across the country. Physicians-in-training may be directing an undue amount of attention to results of tests and imaging techniques without realizing their limitations. Patients may not be receiving optimal care if residents are distracted by the results of tests and are tempted to ignore what the patient is saying as well as what is demonstrated by physical examination. A "Pavlovian" response to results of technology may result in further promoting the "cascade" of ordering additional tests.

The training of residents and the development of technology occur simultaneously in academic health centers. There is an obvious need carefully to integrate both without causing impairment of the clinical decision making abilities of future clinicians. Nor should technology be "applied uncritically without evidence of effectiveness with disastrous effects on health resources."[5]

There are no simple solutions. However, it is important to emphasize the role of technology as serving a consultative function only. Information provided by technology needs to be viewed by residents as offering one single component of the total data base that will be used in inductive reasoning for problem solving. It is critical to understand the limitations as well as the predictive values of these tests in the clinical context.

As a result of the Bell Commission report, new section 405 regulations of the New York State Hospital Code were implemented in July 1989. The Bell Commission report and these regulations limit working hours and increase supervision for residents. How will this change in graduate medical education affect the issues we have raised? Will rested, alert minds be more critical in their approach to problem solving, or will residents, hampered by a lack of continuity of care, have less of an opportunity to evaluate test results in their proper clinical context? It is important to examine carefully the results of this "natural experiment" that began in July 1989. Will patients receive better care from more rested and better supervised house officers or receive less care due to over-reliance on technology?

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Emergency room visit time: Changes over a 16-year period

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ABSTRACT. A one-week time and motion study was conducted at a large urban hospital replicating a study performed in 1969 in the same hospital, allowing a longitudinal study of emergency room utilization to be reported. Despite a 36% increase in the number of emergency room patients over the 16-year period, the findings from both studies replicate many of the patterns previously reported. Over time, both length of stay and wait times to see physicians have increased, although the values are still well within the ranges found in the literature. As competition increases, hospitals and their emergency room managers will have to determine what constitutes acceptable visit and waiting times in order to compete effectively for emergency room "clientele." Studies such as this one serve as a foundation for hospitals to implement policy changes that will improve health care delivery, and, in fact, this institution has already implemented changes based on this study.

(NY State J Med 1989; 89:446-449)

When Goss et al[1] reported their study of utilization of a large urban hospital emergency room (ER) in 1971, only one study had been reported investigating the length of patient visits to emergency rooms and the amount of time spent waiting to see a physician. Since then, research on emergency room utilization has burgeoned. Weekly, daily, and hourly cycles have been observed.[2-4] Moreover, other studies have replicated the finding of Goss et al of a positive relationship between ER census and patient wait time to see a physician.[5-11]

To date, however, a longitudinal study of ER utilization has yet to be reported. A recent study of time spent by patients in a hospital emergency room allows a replication of the earlier examination in the same facility. The results provide not only an indication about how time spent in the ER has changed in almost 16 years, but also a baseline predating the implementation of prospective payment and competition from what is now an increasing number of free-standing emergency and urgent care practices.[12-14] In addition, the results have provided guidelines for modifying emergency room operations.

METHODS

Located in New York City, The New York Hospital is a voluntary, acute care, university teaching hospital with 1,100 beds. At the time of this study in 1985, it handled 47,223 emergency room visits annually, 142,434 clinic visits, and 165,000 visits to members of the faculty practice plan. Since the report by Goss et al,[1] the emergency room has been consolidated from three to two sections: the adult ER and the pediatric acute care unit (PACU). The PACU is open from 8:00 AM to 11:30 PM daily; when it is closed, infants and children are seen in the adult ER. In addition to providing pediatric care when the PACU is closed, the adult ER is the site for treatment of all trauma patients regardless of age. At the time of the report by Goss et al,[1] this institution did not maintain an ambulance service; however, this emergency room now receives patients through its own ambulance service as well as through the New York City Emergency Medical Services 911 System.

The present survey focused on both the adult and pediatric emergency rooms. As in 1969, the adult ER continues to be staffed by two different departments: medicine and surgery. The physician staffing pattern of the adult ER has not changed dramatically over the past 16 years. Goss et al[1] reported that there was at least one surgical resident and one medical resident on duty at all times in the adult ER. In addition, one pediatric resident was assigned to the adult ER during nights, evenings, and weekends when the pediatric ER was closed. In the present study, there were between two and three medical residents assigned to the adult ER at all times. One surgical resident was on duty at all times, and a surgical assistant was present from 8:00 AM to 5:00 PM weekdays. As in 1969, a pediatric resident is on duty in the adult ER during evenings and weekends when the PACU is closed. In both studies, the departments of medicine and surgery had attendings on call. The pediatric ER is staffed by a pediatric attending, an intern, and a medical student from 8:00 AM to 5:00 PM. From 5:00 PM to 11:30 PM, the pediatric attending is on call with one resident available in the PACU.

In both studies, physicians representing various subspecialties were on call at all times. Goss et al[1] reported that nursing coverage varied according to shift and day; however, in the recent survey, four nurses were assigned to the ER from 11:00 PM to 11:00 AM, and seven nurses were on duty from 11:00 AM to 11:00 PM. As in 1969, x-ray and laboratory facilities were continuously available.

At the time of the original study in 1969, a systems approach to emergency medicine was beginning to take shape. Patients arriving at the emergency room were seen initially by a registrar and then by a nurse who determined priorities regarding the patient's need for immediate care. The patient was then placed in a treatment room and seen by a physician. During the past two decades, a standard approach to emergency medicine has evolved. In the present study, patients entering the emergency room are seen initially by a triage nurse, who assesses patient

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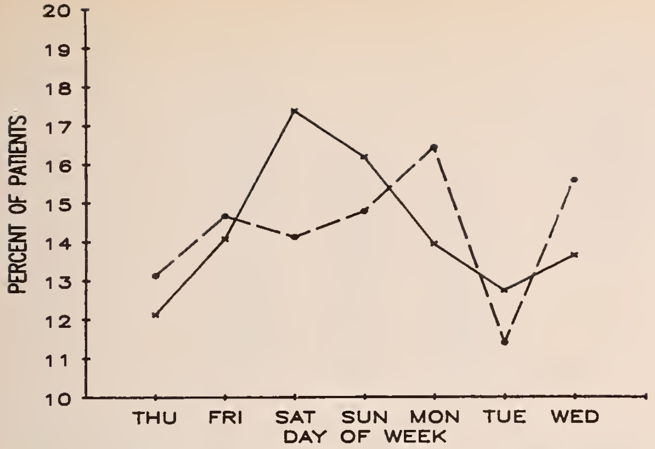

FIGURE 1. Patient utilization of the emergency room by day of week in 1969 (solid line) and 1985 (broken line).

acuity and triages the patient to medicine or surgery. Patient acuity can vary from "A" (needs to be seen right away) to "B" (needs to be seen within ten minutes) to "C" (needs to be seen within 30 minutes). After triage, patients are registered by clerical staff. When registration is completed, nurses pick up the charts and place patients in one of 22 treatment rooms, where the nurses take a brief history and physical assessment. The charts are then given to either the medical or surgical resident.

Data were collected for a one-week period in May, from 8:00 AM Friday the 17th to 5:00 PM Friday the 24th. Time clocks were stationed at the ER entrance and at the triage, physician's, and clerk's desks. A special form was stamped with the times of entry, triage, registration, physical examination, x-rays, and consults, and the time of final dispositions and exit from ER. A nurse with no other duties was assigned to the study to insure that complete and accurate data were recorded.

RESULTS

During the period of study, there were 1,130 visits to the ER. Ninety-three patients were triaged directly to one of the hospital's clinics. The remaining 1,037 patients were registered in the ER and followed through their visit. Of the 1,037 registered patients, 191 were admitted to the hospital. Data collected were sufficient to analyze 86% to 93% of the registered patients.

Utilization. Goss et al[1] reported that the average number of patients served in the ER per day in 1969 was 95, with an average of 12 patients admitted. Currently, the average daily load has increased to 129, with an average of 24 patients admitted. Patterns of utilization appear to have changed from 1969 to 1985: Volume in 1969 peaked on Saturday and during the evening shift, but on Monday and during the day and evening shifts in 1985 (Figs 1, 2).

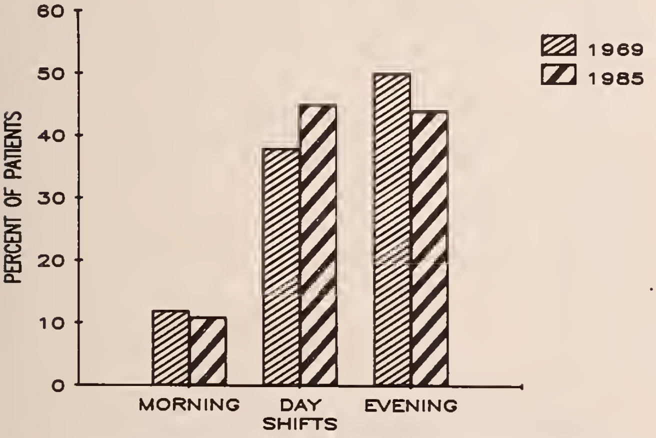

FIGURE 2. Patient utilization of the emergency room by shifts (morning—12:01 AM to 7:59 AM; day—8:00 AM to 3:59 PM; evening—4:00 PM to 11:59 PM) in 1969 and 1985.

TABLE I. Length of Stay in an Emergency Room, 1969 and 1985

Length of Stay (minutes)	1969 No. Patients	1969 % Patients	1985 No. Patients	1985 % Patients
0–29	46	8	43	4.5
30–59	137	24	139	14.4
60–119	177	31	319	33.1
120–179	109	19	216	22.4
180 +	102	18	248	25.7

Total Visit Time. In 1969, the median total visit time during the week under study was 93 minutes, while in 1985 the median total visit time was 115 minutes, almost a 25% increase. For both time periods, however, there is considerable variation among patients. The increased length of a visit in 1985 is reflected in the distribution. In 1969, 32% of the ER visits were completed in an hour or less, while in 1985, only 19% were completed in the same time span. Conversely, almost half of the ER visits in 1985 lasted two hours or longer, compared to only 37% in 1969 (Table I).

Figure 3 compares the median total visit time for patients over the seven-day time period. Patients experienced longer median total visit times in 1985 on four days as compared to longer median total visit times in 1969 on only two days.

Wait Time to See a Physician. In 1969, the median waiting time to see a physician in the week under study was 27 minutes; in 1985, the median waiting time was 37 minutes, almost a 40% increase. In both investigations, there was less variation in wait time than in visit time. In 1969, 83% of the ER patients saw physicians in less than an hour; in 1985, the figure was 72%. In both cases, very few waited to see a physician two or more hours (3% and 8%, respectively) (Table II).

Figure 4 presents the median waiting time to see a physician experienced by patients each day of the week for the two time periods under study. In 1969, median wait times to see a physician exceeded 30 minutes on only two days (Saturday and Wednesday). By 1985, there was only one day of the week when median wait times did not exceed 30 minutes. In 1969, wait time to see a physician increased from the morning to the daytime to the evening shifts. This finding was replicated in 1985 (Fig 5).

A comparison of waiting times by days of the week indicates that the relationship between ER census and wait time found in 1969 was not replicated in 1985 (Fig 6). There was a significant ($p < 0.05$) correlation ($r = 0.73$) between number of patients per shift and median waiting time to see a physician in 1969; however, this correlation was only 0.20 for 1985.

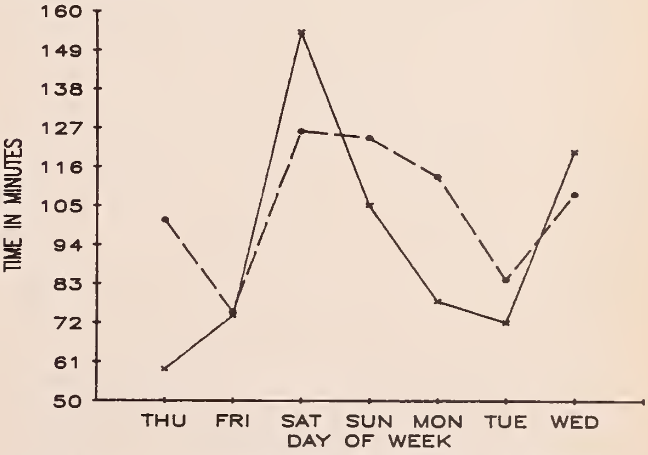

FIGURE 3. Median total visit time by day of week in 1969 (solid line) and 1985 (broken line).

TABLE II. Wait Time to See a Physician in an Emergency Room: 1969 and 1985

Length of Stay (minutes)	1969 No. Patients	1969 % Patients	1985 No. Patients	1985 % Patients
0–29	297	58	355	40
30–59	127	25	284	32
60–119	70	14	189	21
120 +	17	3	68	8

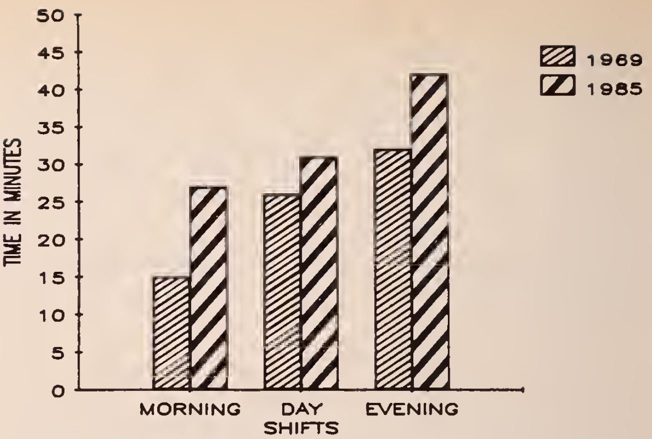

FIGURE 5. Median waiting time to see a physician by shifts (morning—12:01 AM to 7:59 AM; day—8:00 AM to 3:59 PM; evening—4:00 PM to 11:59 PM) in 1969 and 1985.

DISCUSSION

Surveys of total visit time and time spent waiting to see a physician were conducted in the emergency room of a voluntary, university teaching hospital during a week in both April 1969 and May 1985. Data were obtained similarly through the use of time clocks, which automatically recorded the time for each patient when punched on a special form at appropriate points in the visit. Time clock records were sufficiently complete to permit analysis of more than 75% of the 668 patients who came to the ER in 1969 and between 86% and 93% of the patients in 1985.

In the 16 years since the first study was conducted, much has changed in this hospital's emergency room. The number of emergency rooms has been consolidated from three to two; the number of patients has increased 36%, and the institution has become a receiving facility for New York City's Emergency Medical Services 911 System. Physician staffing in the ER has been augmented with an additional medical resident as well as one surgical assistant during peak hours. The laboratory system has been computerized and nursing coverage expanded better to meet the peak demand.

According to Bertram,[11] patient census in most emergency rooms is highest over the weekend and during the evening shift, with peaks around the noontime and dinner time hours. This appeared to be true in 1969; however, this was only partially replicated in 1985. Emergency room utilization in 1985 peaked during the day and evening shifts, but census was highest on Monday and Wednesday (Figs 1, 2).

Reported visit times range from 78 minutes to 133 minutes.[11] The results from both studies fall within this range, although it is clear that over time, visit time at this hospital has increased. In the 1985 study, the percentage of patients in the ER for more than three hours (26%) is significantly ($p < 0.00001$) greater than that reported by Goss et al[1] (18%) (chi square = 161.4556).

The reported ranges in the literature for wait time to see a physician are 27 to 47 minutes (Bertram[11]), putting the Goss study at the low end of the spectrum and the replication study at the midpoint. It is clear that over time, both visit and wait times have increased, although they are still well within the ranges reported in the literature.

The relationship between ER census and wait time to see a physician had been, until now, a well-replicated finding. Other data from the 1985 time and motion study indicate that wait time is only one of several physician-related variables that affect patient visit time (S. Abramowitz and R. W. Yurt, unpublished data). Waiting for consults, for patient disposition decisions to be made, and for laboratory results appear to be equally important determinants of an ER length of stay. However, recent work also indicates that increases in physician staffing can mitigate the effect of peak ER usage, suggesting that emergency room managers have some degree of flexibility in decreasing the amount of time patients wait to see a physician.

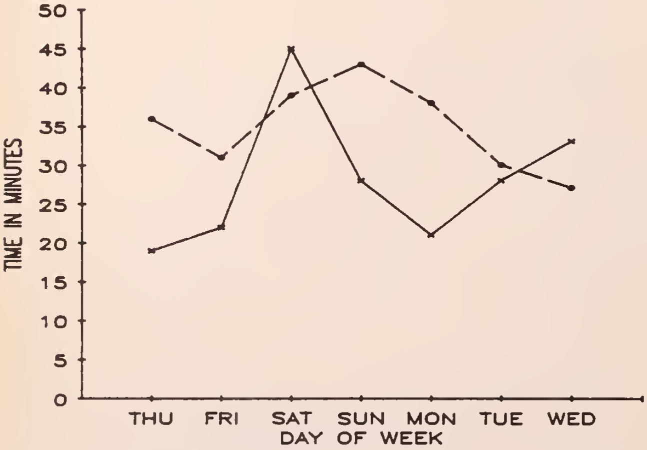

FIGURE 4. Median waiting time to see a physician by day of week in 1969 (solid line) and 1985 (broken line).

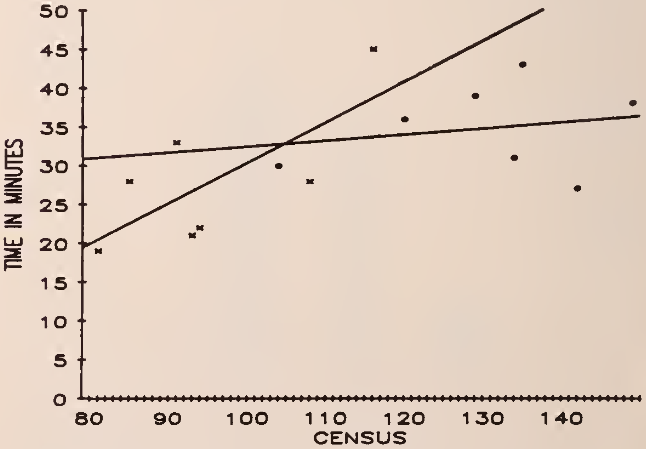

FIGURE 6. Median waiting time to see a physician as compared to emergency room census in 1969 (X) and 1985 (O).

While the visit and wait time increased at this hospital in the last 16 years, their ratio has remained constant. Wait time to see a physician, in both studies, constituted only a third of the median total visit time. In this study, the median time with the physician was 12 minutes, the median time for ancillary tests was 25 minutes, and the median time between final disposition and exit from the ER was 5 minutes (S. Abramowitz and R. W. Yurt, unpublished data). These figures, together with a median wait time of 37 minutes, account for 79 out of 115 minutes, or 67% of the median total visit time.

Most of the research reported on the length of a visit and wait time to see a physician predates the era of increasing competition for patients and pressures for cost containment. As competition increases, hospitals and their emergency room managers will have to determine what constitutes acceptable visit and waiting times in order to be able to compete effectively for emergency room "clientele." In order to accomplish this, hospitals will need to reevaluate their systems approach to emergency medicine. A recent study by Saunders[15] showed that high acuity patients received an efficient level of care in the emergency department, while low acuity patients received less efficient care. Saunders reported an inverse relationship between total wait time to see a physician and level of patient acuity. It is important to note that 87.8% of the patients in Saunders' study were reported to be low acuity. In general, however, emergency services have directed their efforts toward critically ill patients, yet the ER is often heavily used by a population seeking primary care.

One solution is to set up a separate track for nonacute patient care. Recently, many institutions have opted for this solution. A recent report by Meilin et al[16] showed that an urgent care center within a teaching hospital setting does result in shorter average turnaround times for nonacute patients.

Hospital emergency departments must identify patient flow bottlenecks on an ongoing basis in order to remain competitive. A computerized patient tracking system from triage to discharge would allow timely updates on problem areas such as waiting for ancillary services or consults. Management can then make decisions on staffing, space utilization, and overall resource allocation so as to address these problems as they arise. Powills and Matson[17] reported that emergency departments can, in fact, make money by instituting changes such as competitive pricing, patient courtesy programs for employees, and faster turnaround times. Studies such as the one presented here serve as a foundation for hospitals to implement policy changes that will improve health care delivery, and in fact this institution has already implemented changes based on this study.

Based on the data gathered in these two prospective studies, specific steps have been taken to improve ER utilization and decrease waiting time. Since a large proportion of the population consisted of "C" category patients, an urgent care center has been opened adjacent to the main ER. During the period between 8:00 AM and 11:00 PM, selected "C" patient are now triaged to this area. Furthermore, ER physicians have been added so that at least one full-time attending ER physician is present at all times in the emergency room. These changes appear to have improved efficiency, shortened waiting time, and led to support for ongoing studies similar to the one presented to verify the findings and implement additional changes.

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Health, income, and activities of retired physicians

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ABSTRACT. Driven to retire mainly by changes in the health care delivery system, physicians nevertheless find it difficult to reduce their professional activities as they grow older. Retired physicians in a seven-county region of upstate New York were surveyed to discover how they were faring in retirement and to elicit information of possible value to doctors planning to retire.

Good health and adequate income were the prime factors in successful retirement, along with active involvement in postretirement activities, especially nonmedical ones. Three quarters of respondents found annual incomes between $25,000 and $50,000 to be comfortable. All agreed that private investments must make up at least a third of this amount, supplementing Social Security, pensions, individual retirement accounts, and Keogh plan distributions.

There was diversity of opinion on remaining active in medical affairs after retirement. A large majority of those who found retirement better than anticipated advised a clean break with medicine. Most of their colleagues who rated retirement about as expected or not as good as anticipated counseled continued involvement in professional activities.

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Many physicians in the 1980s, affected by the radical economic and political changes in the health care delivery system, are electing to retire from the practice of medicine. Their predecessors were able to cut back gradually on their professional activities as they grew older and maintain a comfortable and rewarding level of practice well into their sunset years. Today's physicians can seldom afford to taper off and still meet the expenses of escalating malpractice insurance rates and the high office overhead necessary to cope with the mounting tide of paper work.

More physicians work in incorporated clinical groups, in industry, or in governmental agencies where there are often attractive inducements to retire. Many hospitals routinely deny operating room privileges to physicians after age 70. The net effect has been a significant increase in the number of retired physicians during the decade of the 1980s.

In 1980, the Directory of the Seventh District Branch of the Medical Society of the State of New York, which lists the members of the seven county medical societies in the Rochester, New York, area and of the Rochester Academy of Medicine, showed 98 retired physicians among its 1,577 members. The Directory for 1988 lists 169 retirees among 1,627 members. This represents an increase in the proportion of retired physicians in the membership from 6% in 1980 to 10% eight years later.

Many doctors feel they are being pushed into a retirement that they do not really want and for which they are frequently ill prepared and poorly suited. Their dedication to their life pursuit has, in many cases, caused them to sacrifice hobbies and interpersonal relationships with family and friends.[1] More than most retiring Americans, they suffer a severe role crisis, equating the loss of practice with a loss of status.[2]

How are these doctors faring in retirement? Can anything be learned from their experience that might be useful to their contemporaries or to their younger colleagues who may be contemplating retirement?

The Rochester Academy of Medicine, which serves a medical education function for physicians of Rochester and the surrounding seven-county area, has for some time sponsored special programs for senior physicians. It was decided it might be useful to survey the retired physicians in the area with the hope that they could provide helpful information, recommendations of value to future retirees, and suggestions with regard to further Academy activities on their behalf.

METHODS

A questionnaire was drafted to elicit information and solicit comments and advice from the roster of retired physicians. In addition to demographic data, the survey asked for facts on health, income, postretirement activities, and reasons for retirement, and for opinions on financial planning and the ingredients of successful retirement.

The questionnaire was mailed with an appropriate cover letter explaining the objectives of the survey to each of the 169 retired physicians. A stamped, preaddressed return envelope was included in the packet. Respondents were assured of anonymity and asked not to use their names or street addresses on the survey forms or on the return envelopes. Completed questionnaires were returned by 142 (84%) of the 169 retired physicians, and these returns comprise the data base for the study.

RESULTS

The average age of the retirees was 75 years, with a range from 52 to 96 years. Their average age at the time of retirement was 68, and they had been retired for an average of seven years, with a range from one year to 34 years. Fifty-six (39%) had been retired for less than five years.

Residence. Eighty percent continued to live in the local area. The remaining 20% had moved out of state after retiring, most of them to areas with warmer climates. More than half went to Florida. Of those remaining in the local area, 14% had changed their residence since retirement, most moving to apartments or condominiums. Two thirds of all respondents continued to live in their preretirement residences. Of those who continued to live in the local area, 30% went

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south in the winter for periods ranging from three weeks to six months.

Nearly 70% lived in single homes, while 25% lived in apartments or condominiums. Only 5% of the respondents lived in designated retirement communities or health-related facilities.

Family. At the time of the survey, 89% of the retirees were married, 9% were widows or widowers, and only 1% were divorced. The retirees had an average of three children, with a range from none to nine. They fell far short of reproducing their profession in numbers of offspring. Among the 425 children of the 142 respondents, only 32 were physicians. Over half (55%) of the retirees had children living within an hour's drive.

Health. Nearly two thirds (63%) rated their health as good, 28% as fair, and only 9% as poor. Thirty-eight percent reported some limitation of postretirement activities because of health problems. An additional 9%, although not restricted by their own health problems, reported that their activities had to be modified because of the health problems of their spouses.

There were interesting correlations between age and health rating. Of the relatively few (12) retirees below the age of 65, only 42% reported themselves to be in good health, a reflection of the fact that a significant number in this age group were forced to retire because of impaired health. Past the age of 65, and up until age 80, good health was reported by nearly three quarters (72%) of the respondents. Over the age of 80, less than half (47%) reported good health.

In response to the request to list diagnoses for their most bothersome health problems, 21% indicated they had no significant health problems. Seventy-nine percent (112), however, listed a total of 222 diagnoses. Table I presents the most common diagnoses listed by respondents. A separate column shows the incidence of these conditions in the general population, age 65 and over, as calculated by the National Center for Health Statistics.

Nearly half of the respondents (48%) had been hospitalized on one or more occasions for these problems since retirement. Twenty-eight percent had had one or more operations after they retired.

Twenty-four percent denied regular use of any medications. Seventy-six percent listed a total of 244 medications that they took on a regular basis. Most commonly used were cardiovascular drugs (156). Among the cardiovascular drugs, the most frequently listed were diuretics (30), beta blockers (28), and calcium channel blockers (25). The next most often used category of drugs was antiarthritic preparations (39). The single drug most commonly listed was aspirin (47), used both as a thrombus inhibitor and as an antiarthritic agent.

Income. Retired physicians were asked to state their joint annual retirement incomes in one of four brackets, and 138 (97%) complied. The breakdown is shown in Table II. They were then asked to indicate, by approximate percentages, the sources of their income, using the information from page one of the 1987 Internal Revenue Service Form 1040 as a guide. Usable data were provided by 115 (81%) of the respondents, and the averages for income sources are shown in Table III.

TABLE I. Diagnoses of Responding Retired Physicians Compared with Those of the General Population, Age 65 and Over*

Diagnoses	Retired Physicians (N = 112) Number	Retired Physicians (N = 112) Percent	General Population 65+ Percent
Heart conditions	42	37	30
Coronary artery disease	27	24	—
Other heart disease	15	13	—
Hypertension	25	22	39
Arthritis (all types)	30	27	47
Genito-urinary diseases	19	17	7
Benign prostatic hypertrophy	13	12	—
Orthopedic conditions	18	16	16
Visual impairment	14	13	9
Hearing impairment	7	6	32
Diabetes	6	5	8
Depression	6	5	—

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* National Center for Health Statistics, 1988. Persons with selected chronic conditions, 1983.

TABLE II. Reported Income of Retired Physicians (N = 138)

Income Bracket	Number	Percent
Less than $25,000	8	6
Between $25,000 and $50,000	63	45
Between $50,000 and $75,000	41	30
More than $75,000	26	19
Total	138	100

Finally, the retirees were asked their opinion as to the adequacy of their incomes to support their expected life styles in retirement. They were given four possible answers: (1) Not quite enough; (2) Adequate, but just barely; (3) Comfortable; or (4) More than we/I really need.

The perception of adequacy of income did not always conform to the actual levels of annual income. Incomes of less than $25,000 were regarded as not enough or just barely adequate by all respondents in this income bracket. Among the 46% who had annual incomes between $25,000 and $50,000, more than three quarters (76%) felt this was comfortable or more than was really needed. Twenty-four percent of this group, however, felt the sum was not quite enough or was barely adequate. Incomes of $50,000 and over were deemed comfortable or more than needed by 97% of those in the top two brackets.

Financial advice was offered by 25% of the respondents. There was general agreement that savings for retirement should be started early (one respondent said not later than age 40) and be regular, with the money being invested in safe and conservative instruments. The use of IRA and Keogh plans was recommended, and several suggested that a full- or part-time job in government or industry be considered for the pension benefits. All agreed, however, that Social Security and pension benefits would not be enough and urged private investments in addition.

Two mentioned that, while inflation was a concern, the financial needs in retirement tended to be less than during active professional life and might offset the effects of inflation. It was also pointed out that, with advancing years and diminishing activity, less income was required. Several suggested considering part-time work in government or as a consultant after retirement to augment income, but agreed that such jobs were difficult to find, especially in view of the problem of malpractice coverage. A few thought it would be a good idea for the Academy to sponsor seminars on financial planning for retirement, but doubted that busy practitioners, who would profit most from such information, would attend them in significant numbers.

Activities. Respondents were asked to rate the importance of each of five medical activities and 14 nonmedical activities in their retirement lives by using a one-to-three scale: 1, indicating somewhat important; 2, moderately important; and 3, very important. Space was provided for listing and rating other activities in both categories. The overall importance of the various postretirement activities, medical and nonmedical, was evaluated by adding the total

TABLE III. Sources of Income of Retired Physicians (N = 115)

Social Security	24%
Pensions, IRA distributions, annuities	26%
Corporate savings and investment plans	6%
Private investments	36%
Rents, royalties, etc	2%
Postretirement earnings	1%
Other sources	5%
Total	100%

score given by all of the respondents to each activity according to the numerical rating scale. Activities in both medical and nonmedical categories are listed in Table IV and are ranked according to the importance ratings assigned to them by the respondents.

Among other medical activities, teaching and research were most frequently listed. One respondent was instrumental in establishing a visiting nurse service in the small rural community to which he retired. Another organized and set up a hospice for the care of the terminally ill.

Within certain of the nonmedical activities listed, respondents were asked to state the specific activities they found most rewarding. Golf was the most favored sports activity, with tennis and swimming the next most popular. In the music category, listening to classical music was listed over playing musical instruments in a ratio of two to one. Woodworking was, by far, the most specified crafts activity. The most popular arts activities were painting and sculpture.

A wide range of other nonmedical activities was listed, ranging from archeology to wine-making, from bird watching, cooking, and computers to model building and tree farming. One hearty septagenarian listed sex and gave it an importance rating of 3.

Reasons for Retiring. Respondents were asked to rate the importance of each of seven provided reasons for retiring, and of any other reasons which they might list, on the same one-to-three scale used for activities ratings. Each of the reasons for retiring was evaluated for overall significance by the same method used for activities rating, that is, by simple addition of the rating points to arrive at a total score for each reason. Results are shown in Table V.

Several respondents added comments that are aptly summarized by the statement of Handler[3]: "The practice of medicine, previously a revered and satisfying profession, is rapidly becoming a trade in which concerns of quality are supplanted by a bottom-line economic mentality. Contributing to physician malaise is the rapidity with which these changes are occurring."

Most common among the other reasons for retiring were age-related mandatory retirement rules in some industries and governmental agencies, and the denial of operating room privileges to surgeons at age 70 by most hospitals. Next most commonly cited were inducements to retire and favorable pension arrangements set up by many industries. Several wrote that they were physically unable to work the long hours and carry the patient load necessary to meet overhead expenses.

TABLE IV. Activities of Retired Physicians (N = 141)

	Cumulative Score*
Medical activities	
Reading medical literature	222
Attending meetings, rounds, lectures, etc	164
Volunteer work in medical activities	106
Consultation or other fee-for-service work	88
Writing on medical topics	80
All other medical activities	33
Nonmedical activities	
Reading nonmedical literature	273
Visiting family	229
Travel	210
Gardening and yard work	175
Television	172
Sports participation	160
Photography	89
Music	86
Taking courses in nonmedical subjects	84
Volunteer work in nonmedical activities	74
Playing cards, chess, checkers, etc	71
Crafts	51
Arts	42
Writing on nonmedical subjects	26
All other nonmedical activities	57

* Highest possible score, assuming all respondents rated the importance of an activity as 3 (very important), would be 423. Lowest possible score would be 0.

TABLE V. Reasons for Retiring Given by Physicians (N = 141)

Reason	Cumulative Score*
High cost of malpractice insurance	156
Aggravations of third party medicine	154
Impaired health	131
Diminishing satisfaction with work	127
High office overhead related to paper work	110
Wish to undertake other activities	102
Fear of loss of competence	85
All other reasons for retiring	105

* Highest possible score, assuming all respondents rated the importance of a reason as 3 (very important), would be 423. Lowest possible score would be 0.

Retirement: Expectation versus Reality. Finally, respondents were asked to evaluate their overall satisfaction with their retirement lives compared with their pre-retirement expectations. Exactly half said that retirement was about what they had expected. More than a third, though (39%), found it better than they had anticipated. Only 11% stated that their retirements were not as good as they had hoped.

Although the replies may reflect as much the subjective mind sets and attitudes of the respondents as the objective realities of their retirement lives, the total responding population was divided into three groups according to their assessments of satisfaction versus expectation. These groups were then separately evaluated for factors that might prove significant in determining the level of satisfaction with retirement. These values are shown in Table VI. The group of 55 respondents who find retirement better than expected are designated RET+, the 71 respondents whose retirement is about as expected are titled RET= and the small group of 16 who are disappointed in retirement are labeled RET−. For purposes of comparison, the values for the total population are shown in a fourth column.

DISCUSSION

The findings in Table VI clearly point to good health as an important factor in satisfactory retirement. A second prime factor is an adequate annual income, or one perceived to be adequate. Other factors that seem to have some significance, such as the average importance scores for activities, notably nonmedical ones, are influenced by the primary factors of health and income, more significantly by health status. Age does not seem to be an important factor, although those whose retirement was better than expected were, on the average, a year younger, retired a little earlier, and had been retired a few months less than the survey population as a whole.

Special observations and advice concerning retirement were added at the end of the questionnaire by 54 respondents (38%). Unlike the general consensus expressed regarding financial planning for retirement, comments on successful retirement showed considerable variation and, in some areas, complete disagreement. On the crucial question of whether to retire at all, for instance, several respondents advocated retiring at the earliest possible age, not later than 65, while health was good and retirement could be fully enjoyed. A roughly equal number reflected the opinions expressed to Grauer and Campbell[1] in their survey of 108 Canadian physicians, age 65 and over, and advised holding off retirement as long as possible, cutting back the work load when necessary, or seeking part-time work where available.

TABLE VI. Comparison of Selected Factors Among Retired Physicians Grouped According to Their Evaluation of Satisfaction with Retirement

Factors	RET+ (N = 55)	RET= (N = 71)	RET− (N = 16)	Total Pop (N = 142)
Average age at time of survey (yr)	73.5	75.4	74.5	74.5
Years retired	6.2	6.9	6.1	6.6
Age at retirement (yr)	67.3	68.6	67.0	67.9
Health rating				
Good	82%	59%	19%	63%
Fair	16%	36%	31%	28%
Poor	2%	5%	50%	9%
Restricted by health problems	20%	43%	81%	38%
Income brackets				
Less than $25,000	2%	4%	29%	6%
$25,000 to $50,000	47%	42%	57%	46%
$50,000 to $75,000	34%	32%	0%	30%
More than $75,000	17%	21%	14%	19%
Perceived adequacy of income				
Not quite enough or barely adequate	6%	15%	82%	20%
Comfortable or more than needed	94%	85%	18%	80%
Activities (average total scores)				
All nonmedical activities	15.6	11.7	6.1	12.5
All medical activities	5.3	4.9	3.9	4.9

This diversity of opinion was reflected in the general tone of advice with regard to preserving a foothold in medicine. One group (20) strongly advised maintaining old hospital contacts, attending medical meetings, keeping up with the medical literature, and generally staying abreast of medical affairs. A second group of about equal size (21) believed this was a mistake. They felt that keeping up with medical developments was nearly impossible, and that all physicians over 75 suffered a loss of competence that was apparent to colleagues, although not always to themselves. In brief, the second group counseled a clean break with medicine, a new life with new interests and new friends. They were clearly echoing the advice of Sharon,[4] that "a person not only must adjust from the previously structured environment to the presently unstructured environment, but must create a new, personally comfortable life-style."

In this area of contention, it is of interest that among those who found retirement better than expected, advice advocating a separation from medicine prevailed by a ratio of four to one. Those whose retirement was about as expected, on the other hand, advised about three to one in favor of remaining involved in medicine. Among the respondents whose retirement was not as good as had been expected and who wrote comments on this subject, all advised staying current in medicine.

Several pointed to the loss of respect and dignity of the retired physician and suggested that retired physicians had to be prepared for this eventuality. Several authors have stressed the same point.[1,2,5] Silver,[6] in a highly personal statement, regrets that he will not be regularly entering "the sanctuary" to be greeted with the deference accorded his rank and station.

Many of those who felt continued active participation in medicine was desirable suggested that the Academy sponsor a placement bureau to bring the retired physician anxious to do part-time work, on either a volunteer or a fee-for-service basis, into contact with those willing to provide such work. All mentioned the need to protect such physicians from malpractice claims. None was very specific about how this might be accomplished.

The broadest area of agreement among all who wrote special advice involved the importance of cultivating hobbies and other potential postretirement activities early in professional life. Several pointed out that a broad range of such activities was essential, and should include some that could be pursued even in poor health. Many of those who found sports participation moderately or very important in their retirement lives advised achieving competence in one or two individual sports, such as golf or tennis, well before retirement.

Lynch[5] wrote colorfully on this point, stating,

> [The retiree] is aware that the days seem longer, and since he is out of the mainstream of activities, his psychological boat is adrift at first without rudder or power. If he is not careful, the boat will land in the backwaters in stagnant pools and he will deteriorate. If he has planned something after retirement, he finally gets his boat started towards at least the sidelines of the mainstream.

Several respondents suggested that the Academy sponsor seminars on retirement planning with lectures by retirement counselors, such as are frequently utilized in business and industry, and dialogues between retired physicians and those planning for retirement. Such seminars are now in the planning stage.

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House staff stress

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Writing in *JAMA* in 1981, Norman Cousins[1] described internship as ". . . hazing at best and systematic desensitization at worst . . ." Readers of *JAMA* responded fiercely, claiming the year was ". . . one of the most rewarding experiences that I have ever been through" or ". . . the absolute highlight of my entire medical career."[2] Despite the strenuous, however nostalgic, defense of the entrenched house staff training system, it has become apparent that the gap between the needs of trainees and their educational programs is widening. There is growing evidence of adverse psychological effects of residency training.

SLEEP DEPRIVATION

If asked to form a mental image of the typical intern, many of us would picture a beleaguered young physician, dressed in scrubs, hair askew, with bags under his/her eyes secondary to a sleepless night on call. Sleep deprivation is perhaps the most consistent and potentially harmful form of house staff abuse. In an age in which residents are expected to master the art and science of Swan-Ganz catheter insertions, cardiac telemetry, arterial lines, and selective, nonselective, partial agonist, and lipophilic beta blocker drugs, the 32-hour work shift pushes even the best young house officers to their limits. Studies on the effects of sleep deprivation on house officers do not instill confidence in the current training system.

Friedman and colleagues[3] studied the effect of sleep loss on interns at the Columbia Presbyterian Medical Center in New York. An electrocardiograph interpretation test was administered to rested interns (having slept for 11 hours) and fatigued interns (having slept for less than two hours); the fatigued interns scored significantly worse. A simultaneously administered mood scale indicated rested interns felt more elation, social affection, egotism, and vigor, but less fatigue and sadness than sleep deprived interns. A later report[4] revealed that difficulty in thinking, depression, irritability, depersonalization, inappropriate affect, and recent memory deficits among interns were common, if not universal, after a taxing on-call tour.

Wilkinson et al[5] sent questionnaires to 5,600 house officers in the United Kingdom in 1973 asking, among other questions, "Do you think that your hours of duty are so long as to impair your ability to work with adequate efficiency?" Nearly 40% of the respondents replied either "always" or "often." Hart et al[6] assessed speed of information processing, decision making, recent memory, and mood in sleep deprived and rested interns at the Medical College of Virginia. The sleep deprived interns had less retention of information, longer response latencies, and greater mood disturbances, including hostility, than the rested house officers.

Would passengers feel comfortable if the pilot of their jumbo-jet aircraft had been sleepless for 24 hours? Would patients want their dentist to perform root canal work on them in a sleep deprived state? Various degrees of sleep deprivation continue to be considered acceptable or desirable at some major institutions of medical education. Askan and Raham[7] point to four justifications for the current system: Sleep depriving night call is a valid learning experience; individual idiosyncrasies, not night hours and sleep deprivation, make night call a negative experience; night call does not cause permanent distortion of personal and professional sensitivities; and quality of care is not compromised by sleep deprived physicians.

As medical scientists who are committed to double blind, randomized, controlled studies, physicians are, at best, inconsistent in perpetuating a training system without documenting whether the negative effects of sleep deprivation on our trainees are real, important to education, or adversely affecting patient care.

STRESS AND PSYCHOLOGICAL DIFFICULTIES

During medical residency training, stress is both unavoidable and perhaps even desirable. An intern presented with his or her first patient in diabetic ketoacidosis is certainly stressed but, in a properly supervised environment, the experience should promote adaptive behavior, learning, and professional growth. However, if the patient workload remains highly demanding in an ongoing fashion and is accompanied by a large amount of nonphysician service responsibility ("scut"), such as pressures to keep up to date with expanding paperwork, and the need to turn over patients rapidly to meet length of stay criteria, even the most well-organized house officer's ability to manage

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is eroded. The first functions to be abandoned are the educational components of the training program. Lectures, visits to the library, and teaching during morning rounds are sacrificed to the non-negotiable demands of patient care. Self-doubt about career choice is likely in such a setting.

Various types of maladaptive behavior may ensue, such as callous attitude development, poor role modeling, and depression. When the overworked resident is finally able to leave the hospital after a tour of duty, it may be to face financial and social pressures, sometimes of an overwhelming degree. Small wonder that those concerned with internal medicine training have examined these problems in some detail. Valko and Clayton[8] interviewed 53 first-year residents at Barnes and St Louis Children's hospitals in 1972. Using standardized criteria, they found the incidence of intern depression was 30%, most cases (69%) developing within the first two months of the year on clinical services that made rounds seven days per week. Four interns had suicidal ideation, three had a suicidal plan. Six interns developed new marital difficulties. Fatigue, decreased libido, and irritability were common symptoms among the depressed group of house staff, some of whom also had a prior history of depression and/or a family history of depression or suicide.

In 1981 Small[9] described a "house officer stress syndrome," characterized by the four universal features of episodic cognitive impairment, chronic anger, pervasive cynicism, and family discord. This syndrome may also be accompanied by three uncommon but serious features: depression, substance abuse, and suicidal ideation or actual suicide. McCue[10] cites data indicating that these problems continue into the postresidency period. Physicians have a suicide rate two to three times that of the general population, suffer from drug addiction 30 to 100 times as often, and are more likely to have had ten or more visits to a psychiatrist than nonphysicians.

Schwartz et al[11] surveyed residents at the University of Rochester Medical Center and discovered that increased stress was associated with emergency room rotations, frequent night call, and lack of sleep. Reuben[12] administered a self-reporting depression questionnaire monthly to 68 medical residents (27, 23, and 18, respectively, at the first, second, and third postgraduate year level) at the Rhode Island Hospital during the 1981–1982 academic year. First year residents had the highest rate of depression (28.7%), with rates falling in each successive year of training. Responses indicating depression were received from 24.7% of house officers on general medical wards, 31.5% in intensive care units, 26.2% in the emergency room, and 13.4% on elective rotations.

Gordon et al[13] prospectively analyzed mood changes among 35 interns at the University of California, Irvine/Long Beach Medical Program during the 1982–1983 academic year and found four factors that changed significantly during the course of the year. Anger/hostility and fatigue/inertia scores increased, while tension/anxiety and vigor/activity scores declined as the year progressed. Girard and colleagues[14] at the Oregon Health Sciences University prospectively studied interns' emotions and attitudes. Levels of excitement, nervousness, anxiety, and "being overwhelmed" decreased progressively while fatigue, depression, and feelings of defeat increased during the year, reaching a maximum at eight months. Ford and Wentz[15] at the Vanderbilt University School of Medicine discovered that the most stressful issues among their interns were inadequate sleep, inadequate time to pursue personal matters, and inadequate personal interest and support from attending physicians. Finally, in a national survey of internal medicine residency programs, Smith et al[16] tried to determine the extent of emotional impairment in medical house staff from 1979 to 1984. During this time period, 55.5% of training programs granted leaves of absence because of emotional impairment; 0.9% of all medical house staff required such a leave, with 10% of these eventually dropping out of medicine completely, 3% unsuccessfully attempting suicide, and 2% successfully committing suicide.

Two studies have specifically examined the influence of residency experience on family and spousal relationships. Landau et al[17] found that 40% of their respondents experienced serious difficulties and 72% of this group believed these problems were due to the residency program alone. Nelson and Henry[18] surveyed family practice residents at the University of Minnesota and reported that the most common concerns were lack of leisure time, spouse complaints, conflicting demands on study time, career ambivalence, decreasing sexual expression, and worries about parenting.

Overall, the reports in the literature do not present an optimistic outlook. House staff are at high risk for a variety of psychological problems, ranging from anxiety to drug abuse to suicide. Despite this reality, changes come slowly to medical education and residency training. The shock of the Libby Zion case in New York and the subsequent report of the New York State Advisory Committee on Emergency Services, Supervision and Resident Working Conditions have resulted in recommendations for corrective action to lessen house staff workload, improve house staff working conditions, and alleviate some of the factors that contribute to house staff stress.

THE LIBBY ZION CASE AND THE BELL COMMISSION

The death of a young woman at a major New York hospital, the subsequent publicity and grand jury report, and the recommendations of a special state advisory committee headed by Dr Bertram Bell stimulated new thinking and a new era in medical training in New York State. The details of the case are well chronicled.[19] The grand jury made five recommendations:

1. The New York State Department of Health should propagate regulations that mandate all level-one hospitals to staff their emergency rooms with physicians who have completed at least three years of postgraduate training and who are specifically trained to evaluate and care for patients on an emergency basis.
2. The state department of health should propagate regulations to ensure that interns and junior residents in level-one hospitals are supervised contemporaneously and in person by attending physicians or those members of the house staff that have completed at least a three-year postgraduate residency

program. These regulations should narrowly define the circumstances under which interns may practice medicine without direct supervision.

3. The state department of health should propagate regulations to limit consecutive working hours for interns and junior residents in teaching hospitals.
4. Legislation should be enacted to prescribe when a patient in a medical hospital may be physically restrained and to standardize the care and attention necessary for a patient in restraints.
5. The state department of health should conduct a study to determine the feasibility of requiring level-one hospitals to implement a computerized system to check for contraindicated combinations of drugs.

The grand jury was obviously concerned about inadequate supervision and instruction, sleep deprivation and overwork, and informational overload. In response to the grand jury report, the Bell Commission issued a series of recommendations to the New York State Department of Health, most of which were due to be fully implemented by July 1, 1989. The most controversial recommendation is the following:

> Individual residents who have direct patient care responsibilities in areas other than the emergency department shall have a scheduled work week which will not exceed an average of eighty hours per week over a four week period and should not be scheduled to work as a matter of course for more than twenty-four consecutive hours, with one twenty-four-hour period of non-working time per week . . . .

The Bell Commission's recommendation caused a great deal of controversy. Although some aspects of the old training system are clearly "abusive," it was built as a model of education, continuity of care, and graded responsibility. Concerns have been raised[20] that the new guidelines are too rigid and will subvert the educational process, leading to a civil service and private industry worker-like "shift" mentality, and to loss of continuity of care. The logistics of scheduling appear daunting: a statewide meeting of 25 chief residents was held in New York to discuss the issue and no consensus on the scheduling alternatives emerged.[21] There is cynicism that the estimated $200 million needed to put the commission's recommendations into effect will be made available.

STRESS REDUCTION TECHNIQUES

The cornerstone of stress reduction should be a reemphasis of the primacy of education in residency training. Too often hospital administrators view house staff as a source of cheap labor for phlebotomy, transport, clerical work, and the like. As recently stated, ". . . central to organization of training . . . is a need to confront finally where . . . meaningful patient care necessary in order to gain eventual competence as independent practitioners . . . ends and . . . burdensome non-medical chores . . . which contribute to fatigue and frustration begin."[22]

Also central to stress management is the reduction of workload, in terms of both hours and nonphysician chores. Specific support systems such as adequate attending supervision, computerization of laboratories, and the hiring of nonphysician care providers (physicians' assistants, nurse practitioners, etc) have been used successfully in different settings.

Specific stress reduction programs have been reported in the literature. As early as 1978, Siegel and Donnelly[23] described a support group for pediatric interns in which the interns committed themselves to ten weekly 90-minute discussion sessions led by two attending physicians. Topics discussed included stress, long hours of work and sleep deprivation, career choices, anger, and helplessness. Overall, the experience was felt to be positive as judged by written evaluations, but the authors emphasized that such groups are only one component in a house staff support system and that ". . . nothing could be more supportive than decreasing the number of hours worked each week." Since that time, reports of discussion groups,[24,25] stress reduction programs,[26-28] and intervention techniques[29,30] have appeared. From a scientific point of view, it is difficult to comment on the effectiveness of the programs reported in the literature to date. There are no randomized trials and there is no standardized tool with which to measure efficacy. In general, authors have used questionnaires to analyze their anti-stress efforts. In spite of these shortcomings, there appears to be agreement on several points, and some lessons can be learned.

Participants in support groups are made aware that it is reasonable to expect camaraderie, empathy, and an opportunity to "ventilate." Problem solving and programatic changes in residencies are welcome byproducts of stress reduction efforts, but they should not be viewed as primary goals since such an approach may lead to disappointment and frustration. Second, the more severely a house officer is impaired, the less likely it is that he or she will participate in such a group. Therefore, program directors should not view discussion sessions as a mental health screening device. Finally, sessions should be scheduled at convenient times and house officers should be totally free to attend, unencumbered by a beeper, in order to maximize participation and continuity. At present, it appears reasonable to consider all stress reduction programs as equally valid and local resources should dictate local policy. Discussion groups, faculty advisor systems, "liver rounds," retreats, and athletic events are some of the most common devices used by training programs. However, these do not take the place of readily available faculty or a formal counseling program for the more severely impaired.

CONCLUSION

These are difficult times for medical education. Current house officers, caring for sicker patients with shorter hospital stays than those of a decade or generation ago, exposed to an explosion of information in medicine, and having at their fingertips technologies undreamed of 20 years ago, are facing hardship and uncertainty. Their training period demands much of them, physically and emotionally, and puts them at risk for psychological and emotional problems. Service (ie, caring for patients), and not education, tends to be the driving force in their lives. With society responding slowly, at best, to the epidemic of acquired immunodeficiency syndrome, residents are working bravely on the front lines, repressing fear of personal danger. The future looks uncertain, with more economic competition and lower incomes and more regulation and governmental control a likely scenario.

It is time for national organizations to analyze the current dilemma and offer proposed solutions, as the Association of Program Directors in Internal Medicine recently did.[31] Those individuals who are responsible for teaching and supervising residents are challenged to devise programs that, on the one hand, deliver excellent patient care and, on the other, provide an environment in which trainees can learn, grow, and prosper.

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The impact of the new code regulations on postgraduate medical education in New York State

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In July of this year, new health code regulations became effective which established working conditions and limited hours for interns and residents. A full explanation of the regulations is presented elsewhere.[1] Briefly summarized, these regulations place limits on working hours of house staff in all teaching programs in New York State. Emergency department shifts are limited to 12 hours at a maximum; house staff work schedules must not exceed 80 hours per week; and a minimum of eight hours of nonworking time must separate work periods. Hospitals must adopt and enforce policies prohibiting dual employment, where such employment exceeds the allotted number of hours. Supervision of house staff by attending physicians or "chief resident" level house staff is mandatory 24 hours a day, and attending physicians must be equally available on call within 20–30 minutes.

These regulations, enacted with surprising speed by the New York State Department of Health, have generated intense debate in the medical educator community with regard to their advisability, workability, and imputed benefits to house staff and patients, as well as the quality of care. The purpose of this article is to discuss the impact of this legislation on the quality and quantity of house staff education in the State of New York. I am certain that more issues and questions will be raised than can be answered.

Rare indeed would be the hospital administrator or house staff member who has not heard of Libby Zion. An excellent review of the case and its effects, seminal to this new legislation, is presented by Asch and Parker.[2] Acting in response to questions raised by the grand jury which

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investigated the Zion case, and issues of the quality of health care in New York City raised by Andrew Stein (New York City Council President), the New York State Commissioner of Health appointed Dr Bertrand M. Bell of the Albert Einstein College of Medicine to chair the Ad Hoc Advisory Committee on Emergency Services, which was composed of "nine distinguished New York physicians."[2] The results of the ad hoc committee hearings, which included representation from graduate medical education, are apparent in the new health code recommendations.

SLEEP DEPRIVATION AND THE QUALITY OF CARE

The "general public overwhelmingly disapproves of [36-hour shifts and 100-hour weeks] and expects residency reform."[3] Why are residency working hours so long? Theories offered by Asch and Parker[2] include "a long tradition maintained by inertia"; "a bow to medicine's historic foundations"; "a rite of passage that tests residents' worthiness"; an "essential of proper training for observation of disease over a period of time"; and lastly, "residents provide an elastic source of physician labor compatible with the economic realities of hospital administration."[2]

Despite the intense debate focused on resident working hours, it has proven extremely difficult to demonstrate a correlation between resident sleep deprivation and quality of care. Deaconson and colleagues[4] summarized ten studies of sleep-deprived house staff performance prior to their own. "Investigators concluded that physicians could compensate for the effect of sleep loss in the performance of both simple and complex psychomotor and cognitive function tests... Investigators have commented on the ability to compensate for the perceived fatigue effect of sleep deprivation."[4] There was little evidence that resident judgment and the quality of care were affected by sleep deprivation. Deaconson and O'Hair studied a cohort of surgical residents by examining them each morning on a daily basis with a battery of psychometric tests. These measured cognition, discernment, visual and auditory vigilance, and eye-hand coordination. Their results showed that "sleep deprivation did not affect overt cognitive or motor performance," and that the correlation between sleep and performance revealed... "only trivial effects due to sleep." They concluded, "the assumption that sleep deprivation associated with usual on call schedules impairs cognitive and motor performance of residents such that clinical care of patients may be compromised is not supported by our observations."[4]

Colford and McPhee,[5] after reviewing an extensive bibliography, concluded that "although it has been widely studied, very few studies have examined the effects of lack of sleep on residents or its impact on the quality of patient care." In 1971, Friedman et al,[6] in an often-cited study, demonstrated a statistically significant increase in electrocardiogram reading errors among sleep deprived interns. Leighton and Livingston[7] described an informal study of 12 sleep deprived house staff members. These residents appeared to have a decreased ability to perform simple mathematical calculations, an effect that could "have tragic consequences." Reznick and Folse,[8] in an extensive review of the literature, concluded that "the literature on sleep deprivation as it relates to the physician is neither rich nor conclusive." Their study involved 33 sleep deprived residents at Southern Illinois University. In studying factual recall, speed and accuracy in concentration, and speed and accuracy in manual dexterity, they found "no significant differences in performance in the sleep-deprived condition compared with performance in the non-sleep-deprived condition in any of the three domains tested."

PERSPECTIVES ON REFORM

Despite the above cited research, few medical educators would deny that reform in resident working conditions is due. Residents are subjected to severe stress, and constructive suggestions for reducing this have been made.[5,9] The ill effects of sleep deprivation and constant stress levels are eloquently voiced by Garcia[10]; exhaustion, slow-wittedness, impaired dexterity, frustration, resentfulness, and intense anger toward patients replace sympathy and compassion in the exhausted resident. Changes in resident hours are "long overdue, sorely needed, and will enhance patient care,"[11] according to the New York Committee of Interns and Residents. A house staff union in Boston is on record as being supportive of similar legislation now pending before the Massachusetts legislature.[12]

The view of the ad hoc committee is quite clear:

> The intent of these regulations is to improve quality assurance. They will also improve postgraduate medical education by permitting residents to have the time and psychic energy to be in a condition to learn medicine and to make the kind of commitment to patients which is the essence of doctoring. Physicians who are sleep-deprived and overworked cannot make a commitment to continuity of care. The new regulations... will allow for improved patient care and resident education.[13]

Imperato[14] writes, "The law of diminishing return should have long ago told us that beyond a certain point little or nothing is added to the knowledge and skills of house officers who are obtunded by a lack of sleep." McCall adds the concern that public belief in sleep deprivation resulting in poor patient care could result in increased liability from lawsuits "alleging that tired residents provided poor care."[3]

The question therefore is not whether resident working conditions and the educational process should be reformed but rather what form this reshaping should take. A major concern in the minds of medical educators is the means by which resident work hours and stress may be reduced, while maintaining or even improving the quality of resident education and experience. McCall[3] believes that the situation is so urgent that conservative calls for studying the problem before making changes are inappropriate and that "we cannot wait for studies to resolve all remaining questions, now is the time for action." After implementation, the regulations can be "fine tuned." The American College of Physicians (ACP), while agreeing that reform is due, stringently opposed the Bell ad hoc committee recommendations when testifying before that body. Strict limits on resident hours as proposed in New York State would be unworkable and paradoxically could worsen the fatigue factor. New York State regulations as written,

> would create major logistical and fiscal problems. . . and problems of fatigue at least equal [to current] night call schedules. . . it is unrealistic to expect interns to function any better after a work week of 5½ 16-hour days (88 hours total) than after a week that included 2 32 hour shifts interspersed with 2½ 9-hour days.[15]

Explicit rules for supervision of junior by senior residents with an on-call system is preferable to on-site attending physicians, at least for medical education purposes. The American College of Physicians reworked these ideas into a formal position paper.[16] With specific reference to the recent enactment of regulations in New York, the ACP took the position that "fragmented changes, hastily implemented, in the structure of medical residencies are likely to produce major disruptions in the enormously complex system of patient care and education that characterizes the modern medical teaching service."[16] It called for placing the issue of working conditions in a broader perspective of medical education. Any changes intended to improve resident working conditions "must take into account the complex realities governing teaching services."[16] In summary, the ACP thought that change should be systematic and coordinated with a balance between patient care and teaching needs. Perhaps changes in the medical care system itself are necessary in synchrony with changes in the educational system. For example, reduction in resident work time must be accomplished in tandem with an increase in medical care support services such as intravenous teams, technicians, and transport personnel. In calling for efforts to reduce preventable medical error on teaching services, the ACP believes that because of the limits of uncertainty intrinsic to medical practice, the mere presence of a supervising or attending physician would not reduce medical error to zero.[16] Other proposals included reasonable restriction on total duty hours with the avoidance of excess breaks in continuity of care or premature disengagement from responsibilities. A formal and fundamental evaluation and review of the resident-supervisor relationship is necessary in order to strike a critical balance between residents' independent judgment and input from senior experienced and technically accomplished supervisory physicians.

On a national level, other states, closely observing the debate in New York, have introduced similar legislation. In California, Pennsylvania, Hawaii, and Connecticut similar bills were defeated in the legislature. "Legislators said they were willing to give organized medicine a chance to clear up its own problems."[12] Legislation similar to that of New York had been introduced in Massachusetts, and a counter proposal that would give the state's academic medical centers wide authority to police themselves is also under scrutiny. Organized house staff in Massachusetts support the legislative proposal. However, non-unionized residents are of the opinion that restriction by the state's ironclad proposed rules would cause a deterioration in the educational process. Richard Egdahl, MD, director of Boston University Medical Center and Chairman of the Coordinating Committee on House Staff Training Issues in Massachusetts, believes that rigid legislation governing house staff working conditions will have a severely deleterious effect on medical education. His opinion is that in spite of Massachusetts' reputation as the "acme of academic medicine," surgical and other residents would desert Massachusetts for programs elsewhere if rigid regulations interfered with their education.[12]

IMPLEMENTING REFORM

Having established that some type of reform is due, the question remains as to who will do it and how it will be done. Foremost among the objections to the new regulations is the impracticality of implementing them as of July 1, 1989. Inherent in the Bell commission's report were provisions for assumption of "non-educational activities" by ancillary workers, such as messengers, transporters, phlebotomists, and intravenous teams. The Greater New York Hospital Association made a conservative estimate that it would take two to three hundred million dollars per year to pay for these new personnel. Logistically, thousands of physician's assistants, practical nurses, phlebotomists, etc, would be needed. Bell[13] concedes that

> addressing this need may be complicated in the beginning by manpower shortages; the manpower should be available if training programs are quickly developed. These occupations must also be upgraded, and the people recruited for these important positions paid appropriately.

Unfortunately, there is not the slightest hint given as to how this will be accomplished. Furthermore, these personnel may not be available at any price. Any hospital administrator or residency director who has attempted to recruit a nurse practitioner, physician's assistant, or phlebotomist is well aware of this, yet hospitals are mandatorily expected to cut house staff time prior to having backup personnel in place.

The most obvious solution to the simultaneous mandate to reduce working hours and provide patient care is to recruit additional house staff. But this is a time when applications to internal medicine and family practice residencies are dropping.[17] Richard Gorlin, chairman of the department of medicine at Mt Sinai Hospital in New York City, recently discussed this "ripple effect."[18] He stated that at Mt Sinai, perhaps 16–18 additional house staff members were needed for the internal medicine program. Although he thought that Mt Sinai's reputation would help in attracting these house staff members, he would be digging deep into the pool of applicants and perhaps coming up with less qualified candidates. Since everyone is dipping into the same pool, he concluded that the municipal and voluntary hospitals, with less attractive residency programs, might come up empty handed. Considering the decreased interest in primary care specialties and the slowing influx of foreign medical graduates, the point is well made.

Organized house staff initially welcomed the proposed changes. But the original opinion of medical students, interns, and residents may be changing. In informal discussions, some residents have voiced the opinion that "shift work" and mandatory time off leaves them uneasy about maintaining continuity of patient care and uninterrupted clinical experience at important moments. Interestingly, the American College of Physicians voices similar concerns:

> The College asserts that a strict limit on consecutive hours to be spent with the sick is not fully compatible with the fundamental

> goals of either the best patient care or optimal clinical education . . .patient care is inevitably more fragmented by rigid duty hour restrictions, because transfer of responsibility between residents will occur with increased frequency, thus increasing the opportunity for errors in information transfer. . . Moreover, the resident who has developed a primary relationship with the patient through first and most extensive contact is less likely to be involved in important decisions affecting patient care. The College [recommends that] consecutive hours residents spend with their sick patients must not be rigidly limited to an extent that produces undesirable consequences for patient care and resident education. Residents must not disengage themselves from care of their patients until proper management and continuity of care are assured.[16]

As voiced by Egdahl, those in the surgical subspecialties might feel this most dramatically. A chief surgical resident, now faced with increased night call to supervise his juniors, must now go home at 8 AM instead of scrubbing in the operating room. To document his operative case load, and achieve the numbers and variety of surgical cases mandated by the American Board of General Surgery, may now become difficult. It is entirely possible that the length of residency training will need to be increased, as the intensity and volume of experience decreases, in order to meet the requirements of the various specialties. Within the specialty of family medicine, this problem would be especially acute. There is a very strong mandate in the residency requirements for continuity of care. Residents forced to "sign off" their family practice center scheduled visits because of previous night call would be deficient in this continuity of care, and it is entirely possible that residencies in New York State would be challenged by both the American Board of Family Practice and the Residency Review Committee. The American College of Physicians voices essentially the same concern in its position paper and believes that changes in policy governing residency training must of necessity be coordinated among the many jurisdictions responsible for postgraduate medical education, including the certifying specialty and subspecialty boards, the Advisory Committee on Graduate Medical Education, and the "appropriate residency review committees."[16]

THE VIEWS OF MEDICAL EDUCATORS

I conducted a simple telephone survey of ten residency directors in family medicine, internal medicine, and general surgery in New York State. This was not meant to be a rigorous scientific study but rather a search for some of the opinions of established and experienced medical educators. Despite the anecdotal nature of this information, it offers food for thought about the possible impact of the legislation on the medical education process in New York.

Ten respondents were asked to indicate on a five-point scale the impact of the new resident scheduling process on the quality and quantity of the resident's learning process. Responses were classified as "strongly negative," "somewhat negative," "neutral," "somewhat positive," and "strongly positive." The five sample areas of resident education were: usual and customary cognitive skills (obtaining history, physical exam, treatment plan formulation); obtaining experience in psychomotor skills (surgical procedures, obstetrical delivery, invasive diagnostic procedures); the continuity of care experience (outcome of results of efforts made); the ability to be present in the family practice center (or other outpatient department) for regularly scheduled or unscheduled visits of assigned families; the ability to be present at regularly scheduled conferences (case presentation, grand rounds, etc). Respondents were then asked to comment freely on the reasons for their opinion.

Briefly summarized, the results were as follows. Seventy percent thought that the effect on usual and customary cognitive skills would be either neutral (50%) or somewhat positive (20%). The explanation usually given was that rested and relaxed residents could better perform these intellectual tasks. Thirty percent of the respondents thought there would be a negative impact on developing these skills.

Sixty percent thought that there was some or strong negative impact on developing psychomotor skills. An additional 30% thought the impact was neutral. Explanations of negative impact included the belief that residents would not be around when patients were available. Those responding in the neutral category thought that rested residents would perform better and indeed have time to read about procedures rather than "see one, do one, teach one."

Turning to the more serious questions of continuity of care, availability in the outpatient department, and availability for conferences, opinion was nearly universal that the new regulations would have a very substantial negative impact on these resident experiences. Ninety percent thought that the impact on continuity of care was somewhat or strongly negative. The development of a "shift mentality," and the requirement to abandon patient care promptly after "sign out," were cited as explanations. Residency directors, especially in family practice, thought the new regulations would conflict with the American Board of Family Practice mandated requirements for continuity of care provided by resident physicians to their assigned families. Ninety percent thought it would have a negative impact on this area. The surgeons' and internists' responses were essentially the same.

With regard to availability for regularly scheduled conferences, 100% of respondents thought that a strong or somewhat negative impact would ensue.

CONCLUSIONS

The state has so far failed to clarify whether or not attendance at conferences would be considered "clinical duties." It seems unlikely, however, that residents about to begin eight or 24-hour rest periods would wish to remain for conferences.

Were one to draw a curve plotting resident hours against resident fund of knowledge (or performance on board scores, or any other quality judgment), it would surely result in a normal distribution. Residents forced to work long and laborious hours would be victims of a deteriorating educational process. Falling short of a minimum number of clinical conferences and didactic experiences would probably result in a poorer educational process. By a similar line of reasoning, the American College of Physicians concludes that responsibility of residents for an excessively large number of patients obviously carries certain risks, including inattention to important medical detail and difficulty in devoting the requisite time to the various elements of humane care. Responsibility for too few patients provides inadequate learning opportunities and is poor for morale. Its recommendation would include training programs that provide appropriate relief for resident teams during and after excessively demanding nights on call, and specific guidelines for acceptable numbers of patients admitted per resident during continuous duty hours.

The proposed regulations for resident working hours in New York State may prove to be one of the greatest uncontrolled experiments in medical education. Should we seize the opportunity "to effect needed changes in house staff working conditions. . .and [develop] an evaluative process to study the effect of these changes on patient care and graduate medical education"[19]? Or should we resist these "rigid regulations not tailored to local circumstance [and] as likely to affect care adversely as to improve it"[20]?

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The education of physicians and the health of New York

Associated Medical Schools of New York position paper on the mission and responsibilities of medical schools and academic health centers in New York State

New York's health care system is confronting a new era characterized by intense concern for balancing cost against the need for adequate access to high quality care. In addition, the system has had to cope with the tragic explosion of acquired immunodeficiency syndrome (AIDS). The medical community is also striving to manage the dramatic expansion of research-based technology, while paying heed to widespread concern about humane values in personal care.

The issues facing the health care system in New York State are felt throughout the country. Here, however, the burdens have been particularly acute, in substantial part because of the disproportionate levels of poverty, homelessness, AIDS, and drug abuse which contrast with the advantages of unparalleled educational and patient care resources in the state. A major challenge, dealt with in this paper, is the need to formulate an effective and comprehensive model for the education of physicians, and to link that process to providing access to high quality health care throughout the state.

A variety of issues impact on our ability to educate physicians. In addition to those cited above are government regulation, support for research and technology, the need to strengthen primary and geriatric care, affirmative action, and, finally, the availability of needed resources.

Of immediate concern to academic medicine in New York is the failure of the state's agencies charged with public oversight and accountability to enhance rather than constrain public and private educational and health care institutions in their response to the state's needs. For example, the recent regulatory initiatives of the New York State Department of Health, such as revisions of section 405 of the State Hospital Code, are understandable in intent, but unworkable as mandated. Many of the new regulations require increased personnel, which is in critically short supply, and will result in significantly higher costs for institutions already in deep financial trouble. Moreover, faculty members are diverted from necessary research and teaching to deal with mounting bureaucratic demands. Clearly, the department is under enormous pressure to resolve a host of issues, particularly containment of cost and protection of patients. Nevertheless, the department's current response to these pressures is overly restrictive, and potentially impedes desired change.

There is an overriding need to refocus the state's regulatory approach. Many of the shortcomings of state regulation stem from an imbalance in state agency oversight and the lack of effective working relationships, particularly between the departments of health and education. If the quality of medical education is to be maintained, the state education department must reassert its statutory responsibility for the quality of medical education at all levels. The changes needed in the education of physicians require reaffirmation of the locus of responsibility for medical education plus participation by the department with the educators to develop rational and jointly accepted educational and patient care goals.

This paper outlines the mission of academic medicine in the state including recommendations of the Associated Medical Schools of New York (AMS) on ways to confront the problems and challenges in medical care. We will point out the differences in the perceptions of the state health department and AMS regarding solutions to these problems and call for a more inclusive, better balanced mechanism for providing constructive accountability to the people of New York and making possible an effective partnership between the public and private sectors.

THE MISSION OF NEW YORK'S MEDICAL SCHOOLS: EDUCATION, RESEARCH AND PATIENT CARE

Education and Service. The primary mission of medical schools, the professional education of physicians, must be seen as a beginning, not an end, in the process of educating physicians for a lifetime of medical practice. Physicians should leave medical school with a core body of scientific knowledge and skills which must, however, be paralleled by the inculcation of values and attitudes that promote caring for the sick and concern for the health care needs of society as a whole. Consequently, these values and attitudes should be grounded in and derivative of the principles and concepts from both the natural and the social sciences as well as the humanities.

This mission is unchanged by time, but the difficulty of meeting its rigorous standards is clearly increased by social and economic factors, such as the cost of a medical education, the overall cost of health care, and the erosion of resources, together with apathy toward promotion of the public's health in general. Assuring quality in the training and credentialing of physicians begins with the medical schools, and the New York medical schools accept and welcome this responsibility and the required heightened accountability.

The elements of our mission are clear. We must ensure that students leave medical school with the basic knowledge, appropriate attitudes, and fundamental skills to enable them to enter residency and to continue their education throughout their professional lives. We must provide those students with a faculty dedicated to scholarship and excellence and with a curriculum that integrates biomedical science with the behavioral, ethical, economic, and humanistic elements essential to the practice of medicine. We must work with our affiliated teaching hospitals to promote scholarship and to provide quality health care while protecting patients from risks attendant upon learning processes. The medical schools must fully embrace the responsibility for the educational content of residency training. Finally, we must ensure the maintenance of research of the highest quality possible since research is indispensable to the promotion of scholarship.

Part of the medical schools' responsiveness to community and statewide needs must include steps to increase the number of physicians trained to provide primary care, including specific preparation for care of the elderly. In these areas, the goals of the state health department have been unassailable, but we have serious concerns about the department's chosen route to those goals and its impact on the quality of medical education.

We do not argue the importance of strengthening primary care in New York, but we must urge caution in pursuing approaches that impose a rigid formula on graduate medical education and which pose a real danger to some outstanding programs in specialty training. Any efforts to change the balance between primary care training and specialty training must be developed with full participation of the medical schools and teaching hospitals. Mutually agreed on changes must be implemented gradually and with due regard for resources as well as educational goals in order to avoid any weakening of good programs.

The health department has steadily sought to encroach on the responsibility of both the board of regents and the medical schools themselves in assuring educational quality. In the past year, the department has attempted to use the revised section 405 of the State Hospital Code as a lever for advancing its own agenda for medical education. The aim of the code revisions, many of which grew out of the recommendations of the Bell committee, is the improvement of the quality of hospital care, but the implementation of the new regulations must be carefully monitored to determine their impact. At this point, it would be unwise to assume that better care will automatically result from the revised regulations. Indeed, the possibility exists that the new regulations could have a negative impact on quality.

The rigidity of the department in its proposed policy on primary care is also evident in its proposals on geriatric care. In both instances, the most effective option would be joint efforts by the hospital community, the state departments of health and education, the state legislature, and AMS to determine what incentives can be developed to attract more physicians into geriatric care and how disincentives such as excessive debt burdens can be alleviated.

The AMS has already taken steps in this direction. At its June 16, 1987, meeting, the AMS deans voted formally to express support for a statewide initiative to strengthen the training of physicians in the care of the elderly. To capitalize on state efforts thus far, AMS has begun working jointly with the New York State Resource Center for Geriatric Education on ways to deal with this issue.

To be effective, a statewide program must take into account the significant differences among the New York medical schools in terms of their approaches to geriatric education, basic organizational and administrative structure, and the amount of time needed to make basic changes in curriculum.

State support can be effective and invaluable. An example is the Ohio model for geriatric training in which grants are provided to those medical schools with a serious commitment to strengthening their program. Schools with such a commitment can obtain seed money to develop their programs which would eventually be continued with school funds as a permanent part of the curriculum. The Ohio plan is sensitive to institutional differences, does not attempt to prescribe a single methodology for the teaching of geriatrics, and recognizes that it is the outcome that is important.

In summary, in the areas of primary and geriatric care, AMS recommends a carefully coordinated approach involving academic medicine, the teaching hospitals, and all appropriate state agencies to determine how the state's needs can best be met without jeopardizing the quality of the educational process. AMS endorses the Ohio model in geriatric training and will continue to work with the state resource center, the state legislature and state agencies to develop a program for New York State.

Research. Research is an essential component in the mission of medical schools. Basic research is the fundamental responsibility of the medical school, whereas clinical research is a shared responsibility between the school and its affiliated teaching hospitals. The responsibility to support research must not be restricted to the basic sciences and traditional clinical sciences but must include issues related directly to health care delivery.

The future of biomedical research in New York is a high priority concern for AMS. Data compiled by the State Health Research Council as reported in the *New York Times* sound an alarm we must heed. The *Times* article of October 17, 1988, stated:

> In the last four years, New York has relinquished its long-time pre-eminence in biomedical research to vigorous competitors in California, Massachusetts and elsewhere . . . No longer is New York, with its internationally known medical centers, the country's premier recipient of Federal grants. Nor can the quality of New York's hospitals and laboratories alone provide enough drawing power for many postdoctoral fellows and other top researchers, who must also weigh doubts about the forbidding cost of living and perceptions of a deteriorating quality of life in New York City.

Statistics only tell part of the story. In 1984, New York was overtaken by California as the largest recipient of research funds from the National Institutes of Health. In 1978, New York researchers received $250 million in federal funds, compared to the $231 million of those funds received by their counterparts in California. By 1987, the disparity in those figures was stark: $658 million to California and $589 million to New York.

In the past decade, Massachusetts, with a much smaller population and far fewer research institutions, has approached New York's level of research dollars.

There is no single reason for this decline. Some attribute it to a lack of aggressiveness of faculty, but since faculty are typically rewarded for research productivity, this is a simplistic and inaccurate perception. More to the point is the *Times* comment that the state and New York City have lagged behind in efforts to attract the rapidly growing biotechnology business that has been an economic boom in other areas. In fact, the presence of world renowned research institutions and adequately funded projects is, in itself, a magnet for such business and goes hand in hand with the kind of economic development New York needs to remain competitive.

State government has a critical role to play in galvanizing public and private resources and must assume leadership on this critical question. The failure of New York to keep its lead in biomedical research should be seen both as a failure of meeting its full economic development potential and a failure to invest in the research infrastructure required to meet tomorrow's health needs.

HEALTH MANPOWER AND GRADUATE MEDICAL EDUCATION

On November 30, 1988, the state Council on Graduate Medical Education approved a report and recommendations regarding graduate medical education in New York. For the most part, the recommendations represent a thoughtful proposal for state policymakers and a basis for new approaches in the state's investment in graduate medical education.

The AMS continues to adhere to the assertion expressed in its response to the original January 1986 report of the state Graduate Medical Education Commission, that graduate medical education must be regarded as part of the education continuum and that, although residents clearly provide much of the hospital-based physician manpower in the state, their primary mission is to learn medicine. AMS also continues to support the commission's position that the educational components of graduate medical education are primarily the responsibility of the medical school, in partnership with their affiliated hospitals.

The council calls for efforts to increase the number of primary care physicians, to increase the minority representation in house staffs, and to organize affiliations between schools and hospitals.

The AMS positions on primary care and minority representation are presented elsewhere in this paper.

The council's proposal for the development of consortia warrants careful scrutiny by hospitals and medical schools. In several areas in the state, such an arrangement has clear potential for strengthening the quality of graduate medical education. In New York City, however, the feasibility of consortia is unclear. The AMS supports demonstration projects to test the effectiveness of consortia but opposes any attempt to mandate such an arrangement statewide.

One overriding issue not prominently addressed in the council report is the impact of AIDS and poverty on graduate medical education and on health manpower in general. In a position statement to the council on June 2, 1988, the AMS underscored the impact of AIDS and stated that this problem should be a major priority for the council. We are reemphasizing that recommendation in light of indications that the problem continues to have profound impact on every aspect of clinical training and medical practice, particularly in New York City, and it still is not receiving the attention from state health policymakers that it warrants.

The combined pressures of AIDS and poverty, again most visible in New York City, have resulted in critical shortages of health manpower of all types and, as discussed later in this paper, have created enormous financial problems for hospitals. This is a bona fide state emergency and the AMS is ready to participate fully in discussions leading to specific proposals to deal with it.

We also recognize that the timing of this crisis could hardly be worse, given the mammoth budget deficit which the state is facing. Painful decisions must be made and cannot be postponed.

MINORITIES IN MEDICINE

The AMS strongly supports increased statewide efforts to increase both minority enrollment in medical school and the number of minorities in graduate medical education. For many years, nationwide and statewide data have produced a discouraging picture for minorities in medicine. Nearly a quarter of the state's population is composed of underrepresented minority groups, including black, mainland Puerto Rican, Mexican American, and Native American, but these groups made up only about 8.5% of the entering class of the 14 New York medical schools in 1987–1988.

Five years ago, the AMS made minority enrollment its highest priority agenda item and determined to attack the problem as close to the root as possible. Thus, with funding from the state education department's STEP program, and from several private foundations, the AMS has developed the most extensive precollege academic enrichment program for minorities in the country, serving some 346 junior and senior high school students in 1988–1989. The establishment of the AMS Office of Minority Affairs will permit a statewide, multifaceted program leading to a long range, permanent solution to this problem.

With a grant from the health department, the AMS has begun a recruitment program among minority medical students from New York who are enrolled at out-of-state schools. The objective is to encourage them to return to New York State through application for residency positions in New York hospitals. This program, however, will not meet its objectives until the total pool of minority medical students is greatly expanded. As an illustration, the entire pool of minority students from New York enrolled in their third year at out-of-state medical schools is only 119.

Our recent accomplishments are a base on which to build, not a step on which to rest. These programs represent a rational but long term approach aimed primarily at increasing the numbers of minorities qualified to pursue medical education at all levels. The AMS strongly recommends that the state expand support of efforts such as these.

While we agree with the broad state goals, we believe that addressing this issue by setting quotas or tying capitation to specific targets, however, is counterproductive and does nothing to strike at one of the major underlying problems: the lack of a sufficient number of qualified minority applicants. AMS will continue to encourage what has been a highly productive partnership between the public and private sectors. In this specific area, New York has become a pace-setter for the nation, and we should continue that momentum.

COST

Academic health centers are being forced to cope with changes in reimbursement methodologies at both the federal and state level. These changes have invariably added costs and/or reduced revenues, thus depriving academic health centers of the resources required to fulfill their mission. The present conundrum facing medical education and academic health centers represents a regulatory "reality gap," where health care expectations rise while resources fall. There is, indeed, a need for more effective cost control in health care; however, the burdens being thrust upon the health care system are increasing, not decreasing.

The impact of poverty on health care constitutes a crisis in New York, particularly in our major urban centers and in outlying rural areas. The most alarming example is New York City, where the health care consequences of poverty are simply overwhelming the present capacity of our health care system. The recent statistics showing a high infant mortality, an increase in tuberculosis, and the spread of drug addiction and its relationship to AIDS, are stark and irrefutable. The cycle of poverty to limited health care access to increased disease prevalence is accelerating.

Care cannot be provided without adequate financing. A comparative analysis reveals that New York is 50th in the country in terms of the rate of growth of hospital reimbursement and with respect to operating margins of hospitals.

With the changes in the mechanism of reimbursement, losses among the New York metropolitan area's 70 voluntary hospitals could amount to more than $100 million this year. The *New York Times* reported the statewide loss estimate for hospitals at $200 million for this year following $70 million last year. These losses are about to be compounded by a change in Medicare reimbursement. On November 1, 1988, the Department of Health and Human Services began a Medicare reimbursement policy that will disproportionately affect large metropolitan teaching hospitals that serve the inner city poor. This problem is compounded in that lengths of stay at such hospitals are often much longer than average because of case complexity and lack of long term care facilities.

According to the Greater New York Hospital Association, the losses will amount to $100 million for New York City's voluntary, not-for-profit hospitals, while hospitals statewide will lose $167 million or 28.5% of the $585 million they would have garnered prior to the change. These losses simply cannot be sustained if high quality care is to be maintained.

One prime reason for these losses is the current use of diagnosis related groups (DRGs) for in-patient care: This methodology penalizes a hospital if a patient's stay extends beyond a prescribed limit for a specified illness or the intensity of care is greater than anticipated.

Why has New York not realized the cost savings predicted by the switch to DRGs? One reason is poverty and its concentration in New York City. It is not possible to discharge people recovering from serious illness to unstable home situations, or, in a growing number of cases, to no home at all.

If providing care to all those in need is a state priority, reimbursement levels must be appropriately adjusted. The state must also take the lead in facilitating the development of alternative health care sites. It is a grave mistake for the health department to limit the number of these alternative facilities through the certificate of need (CON) process, when these alternative sites are essential to securing the advantages of a prospective payment system. Therefore, the AMS supports the Health Care Financing Council's review of the entire Article 28 CON process. The value of regionalization and the ultimate cost of the CON review process itself should be explored, in the hopes of rationalizing this process.

The assessment process, as currently administered by the Health Department, must also cease being merely punitive and accusatory. Mark Twain observed: "To do good is noble. To tell others to do good is also noble and a lot less troublesome." As an example, how can the health department in good conscience loudly excoriate an Interfaith Hospital or Lincoln Hospital for inadequate coverage and yet do little about the chronic shortage in nursing? Moreover, what impact do critical press releases have other than to further discourage nurses from working in these institutions?

ACCOUNTABILITY AND STATE POLICY DEVELOPMENT

Society must have confidence in its physicians and the integrity of the medical profession. The public must be assured of the quality as well as the relevance of the physician's education. Since, as indicated above, the mission of the medical schools encompasses both education and, through its clinical faculty, patient care, responsibilities and accountability become inevitably interwoven. The medical schools are accountable for the standards they set for medical education, and since faculty, students, and residents are involved in hospital-based patient care, both the schools and the hospital share in the responsibility for maintaining standards of care.

Similarly, state oversight in educational and practice goals must be shared primarily between the departments of health and education. This could be done through a new entity with representation from all of these constituencies and directly accountable to the legislature and governor. It would be similar in some respect to the state Council on Graduate Medical Education, but, unlike the council, it would not function under the auspices of a single department, and its scope would go beyond graduate medical education concerns. For example, it might be charged with conducting a thorough review of some of the pressing health issues touched upon in this paper, such as the crisis in nursing and allied health manpower, the need to strengthen primary care, particularly for geriatric patients, and the need to revise the reimbursement mechanism and the CON process.

Clearly, a single entity could not be expected to develop policy recommendations for all these issues, but would select the most urgent concerns for its review. Duplication of the work of existing bodies, such as the AIDS Advisory Council, would be avoided. Neither would this proposed body be involved in the accreditation process. It would direct its efforts to long term policy development in health. The advantage of such a body would lie in its ability to speed the exchange of ideas among the various health constituencies in the state while enhancing the likelihood of beneficial resolution and facilitated implementation.

Our theme in this paper is balance, permitting representatives of all facets of the medical care community to forge a true partnership between the public and private sectors that will permit a concerted statewide effort to find permanent solutions to these problems. The AMS is prepared to work with state government and the private sector to bring this partnership about.

FROM THE LIBRARY

THE ANNIVERSARY ADDRESS

THE OBLIGATIONS OF THE PHYSICIAN AND THE LAYMAN TO EACH OTHER

. . . There is a general tendency affecting all grades of people toward that which will create a sensation, and the advancement of science having disturbed very materially our old creeds, and looking around for others, we have been disposed to accept those which seem newer and which possess a larger amount of the marvellous. This is evidenced by the success of the travelling doctor, who goes from place to place, heralded by posters and trumpets, and who cries the virtues of his wares and his miraculous cures from the housetop. The old-time doctor is passing away, and in his place we have the up-to-date medical man who, having imbibed his share of the spirit of the times, is losing in many respects more than he gains. In many families there is no longer the old family physician; neither do there exist those ties which should exist between the medical man and his patient; and just who is to blame for this I am not sure. On the part of the physician I have been led to suppose that his desire to grow rich rapidly has led him into many pitfalls, and has often stranded him and lost him in the driftwood of the unplaced and unknown. Business ways and business means of being brought into the profession, and many a man's yearning for notoriety, are being gratified through the daily papers. The public are indebted to the medical profession, not only for the care and attention which they receive from them, but also in the security in which they are living from the ravages of contagious diseases. There is scarcely a town or village in this State that does not owe its protection in this respect to the physician. It is to him they turn when cholera is at their doors, when diphtheria is in their houses, and when contagion is abroad in the land; but this is all forgotten when the fruits of his labors are reaped. It is his work which has reduced the death-rates in our largest cities to the lowest point in the history of the land. It is his work which has built up the munificent fortunes of life-insurance institutions. It is his work which constitutes the true charity of the world over. The most laborious work is none too hard for him, and the best years of his life are often given in his efforts to assist humanity, and his reward lies in the empty glory of a discoverer. . . .

The medical man is expected to protect the public from the ravages of disease when called upon as a citizen, and it is his duty as a citizen to respond to these calls without any compensation, if need be; yet when they come to the public through their constituted legal societies they have to fight every step against obstacles placed in their pathway by the very public who so willingly accepts so much from them. There has not been a single safeguard enacted for the protection of the public by this State Medical Society but what it has had to go into your halls of legislation and fight for it for dear life. We ask you to give us the authority to say who shall practise medicine in the State of New York!. . . .

. . . Now, since this is the legally instituted and recognized medical society of the State of New York, and the body in which inheres the major responsibility for whatever action may be taken affecting medical affairs, it follows that all legislation relating to the practice of medicine, either as between physicians or between the physician and the community, should originate in this Society, and be under the control and administration of this Society. In that view, and with a hope that I have convinced you of the honesty of our purpose that we are working for the good of the community and the State at large, let me beg of you to go out from this hall determined to use all the influences at your command for the furtherance of honest medical charity and the destruction of quackery in all its phases.

SENECA D. POWELL, MD

(*Trans Med Soc State NY* 1898; 43–48)

A history of the Public Health Association of New York City

FLORENCE KAVALER, MD, MPH, BENJAMIN WAINFELD, MD

Voluntary health agencies are an important facet of the American health scene and function as nongovernmental, privately funded organizations with large community constituencies. These agencies include professional associations such as the American Medical Association and the Medical Society of the State of New York, disease-oriented societies (American Heart Association, American Cancer Society), and service organizations (Visiting Nurse Association, American Red Cross), and provide various levels of activities including community education, research support, direct health care services or indirect financing, community planning and policy development, etc. Within this classification, the Public Health Association of New York City (PHANYC) emerges with a distinguished history and a strong voice promoting the health of the public through community education, leadership, and policy interaction with governmental and private sectors of the health care system.

FOUNDING

Almost 100 people gathered at the New York City Department of Health for the first organizing meeting of the Public Health Association of New York City on Monday, May 18, 1936, at 8:30 P.M.[1] Dr George T. Palmer, Deputy Commissioner of Health of New York City, the presiding officer, recounted the original proposal which emerged from a District Health Officer's Committee of the New York City Department of Health, consisting of Dr Arthur I. Blau, Dr Leopold Rohr, Dr Louis A. Friedman, and the department's Director of the Bureau of District Health Administration, Dr Margaret Witter Barnard. This had led to a meeting of 40 health department employees who were members of the American Public Health Association (APHA) (which then had 5,000 members), and an invitation to all APHA members in New York City (about 400), to plan for a local organization.

The meeting was held in the new ten-story headquarters building of the Department of Health at 125 Worth Street, one of those completed by the Federal Public Works Administration during the Great Depression. These were the days of President Franklin D. Roosevelt's "New Deal" and New York City's Mayor Fiorello LaGuardia, characterized as "a reform mayor with a genuine interest in public health and welfare." [2] Dr John L. Rice, who was then commissioner of health, had launched the languishing decentralization plan of community-based health districts which had eight new districts in place by 1935 (and nine additional opened by 1939).

Response to the concept of a local voluntary public health professional organization was extraordinary, and the proposed constitution and by-laws were adopted and an executive board and officers were elected. It was agreed that affiliation with the APHA was to be requested, and it was noted that Reginald M. Atwater, MD, DrPH, the then executive secretary of the APHA, was present at the meeting.

PHANYC's founding statement of purpose was simply:

> To aid in the promotion and protection of public health and public safety, to provide scientific advancement of members, and to extend and develop the public health movement in New York City.[3]

In addition to the charter membership of those present at the meeting, "all APHA members residing or having their place of business in New York City at the time of the meeting" were given an opportunity to become enrolled as charter members. The list of 283 names, compiled by September 4, 1936, was submitted to APHA,[4] and by November 1936, a request for a representative to the APHA Governing Council was made. Shortly thereafter, Dr Margaret Witter Barnard, Director of the New York City District Health Administration, became its first delegate.[5]

It was in response to the Annual Report of 1937, and a request for support by APHA, that Dr Atwater wrote: "I find that I can squeeze out $100 for the purposes of the PHANYC" and "with it goes best wishes for the success of your venture in 1938." [6]

The first Executive Board meeting (Table I) was held on June 10, 1936, at a luncheon meeting hosted by Donald B. Armstrong, MD, at the Metropolitan Life Insurance Company (Fig 1). Dr Armstrong, the first PHANYC president, who was also the third vice-president at Metropolitan, chaired the meeting at which time "the question of dues was discussed. It was the consensus that, for the

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Dr Kavaler is Professor, and Dr Wainfeld is Clinical Professor, Department of Preventive Medicine and Community Health, State University of New York Health Science Center at Brooklyn, Brooklyn, New York.

Address correspondence to Dr Kavaler, Department of Preventive Medicine and Community Health, Box 43, State University of New York Health Science Center at Brooklyn, 450 Clarkson Ave, Brooklyn, NY 11203.

TABLE I. Initial Officers and Board of Directors, 1936–1937

President	Donald B. Armstrong, MD Third Vice-President, Metropolitan Life Insurance Company
First Vice-President	John L. Rice, MD Commissioner of Health, New York City
Second Vice-President	Hazel Corbin, RN, General Director, Maternity Center
Secretary-Treasurer	Frank Kiernan, Director, New York Tuberculosis and Health Association
One-year term	
Mary E. Chayer, RN	Public Health Nursing, Teachers College, Columbia University
Bernard S. Coleman	Executive Secretary, New York Tuberculosis and Health Association
Thomas J. Duffield	Director, Bureau of Records, New York City Department of Health
Ralph S. Muckenfuss, MD	Director, Bureau of Laboratories, New York City Department of Health
Clarence L. Scammon, MD	Staff, Commonwealth Fund
Alec N. Thomson, MD	Urologist, Medical Director of the American Social Hygiene Association
Two-year term	
Arthur I. Blau, MD	District Health Officer, New York City Department of Health
E. H. Lewinski-Corwin, PhD	New York Academy of Medicine
Haven Emerson, MD	Professor of Public Health, Delamar Institute of Public Health of College of Physicians and Surgeons of Columbia University
George T. Palmer, PhD	Deputy Commissioner of Health, New York City Department of Health
Professor Thorndike Saville	Dean, College of Engineering, New York University
Alfred E. Shipley, MD	Deputy Commissioner, New York City Department of Hospitals

present, no action should be taken on this matter." [7] It was agreed that a general scientific meeting be held shortly, and Dr George T. Palmer, the deputy commissioner of health of New York City, was appointed to chair a program committee (Fig 2).

PHANYC addressed its first policy issue at the executive board meeting on March 4, 1937, and supported the funding proposal of Dr Sigismund S. Goldwater, commissioner of hospitals, to the Board of Estimate of New York City for 1,700 additional hospital beds for tuberculosis patients.[8]

THE WAR YEARS

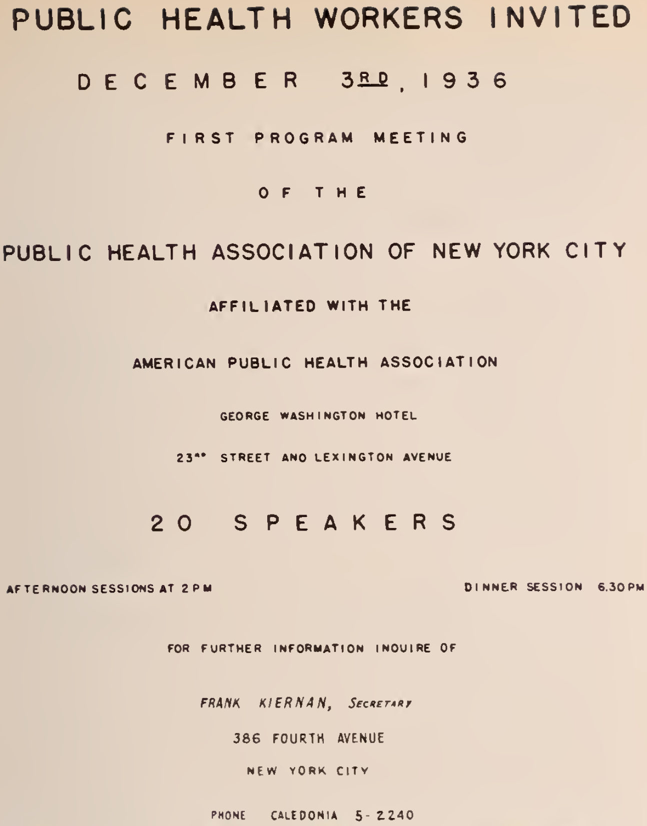
By 1937, PHANYC had swelled to 475 members and steadily grew until 1940 when membership peaked at 750. A dues policy of $1.00 per year was adopted in 1940, although members in the armed services had their dues deferred "for the duration." Throughout the 1940s, membership hovered at 650–750.

Communication among a growing membership practicing in various health settings became paramount in maintaining the spirit, emphasis, and activities of PHANYC. Thus the *Bulletin of the Public Health Association of New York*, Volume I, Number 1, was published October 1944 (Fig 3). PHANYC President Dr Leverett D. Bristol, the American Telephone & Telegraph Company's health director, proclaimed that, "This newborn Bulletin of our local Association should be both an inspiration and an instrument for our future achievements as members of the public health profession of this great world center." [9] That first issue concentrated on the announcement of the forthcoming "Second War-time Convention of the American Public Health Association," held in New York City at the Hotel Pennsylvania, the program for which featured "Public Health as an International Problem," "Today's Global Frontiers in Public Health—South America, China, Great Britain, United States," "Tuberculosis in the Army," and "Health Problems under Military Government."

FIGURE 1. Donald B. Armstrong, MD, Third Vice-President, Metropolitan Life Insurance Company (photo courtesy of Metropolitan Life Insurance Company archives).

The Tenth Annual Meeting at the Hotel Martinique was announced in the April 1946 *Bulletin*[10] (Fig 4) and was a testimonial to Dr Ernest L. Stebbins, the former commissioner of health who, with his predecessor Dr John L. Rice, "displayed a keen interest and encouraged the Association in all its undertakings." *The New York Times* and *New York Herald Tribune* covered the event at the Hotel Martinique.

In congratulatory comments, Dr Iago Galdston, the ex-



PUBLIC HEALTH WORKERS INVITED

DECEMBER 3RD, 1936

FIRST PROGRAM MEETING

OF THE

PUBLIC HEALTH ASSOCIATION OF NEW YORK CITY

AFFILIATED WITH THE

AMERICAN PUBLIC HEALTH ASSOCIATION

GEORGE WASHINGTON HOTEL

23RD STREET AND LEXINGTON AVENUE

20 SPEAKERS

AFTERNOON SESSIONS AT 2 PM | DINNER SESSION 6.30 PM

FOR FURTHER INFORMATION INQUIRE OF

FRANK KIERNAN, Secretary

386 FOURTH AVENUE

NEW YORK CITY

PHONE CALEDONIA 5-2240

FIGURE 2. Announcement of first meeting of Public Health Association of New York City, 1936 (PHANYC archives).

ecutive secretary of the Committee on Medical Information of the New York Academy of Medicine, characterized PHANYC as a "lusty pre-adolescent" and wished it a "speedy maturation." He believed that "It is incumbent upon us to convert the motley brotherhood of public health workers into a well-defined, well-integrated, functionally rational and intellectually disciplined profession."

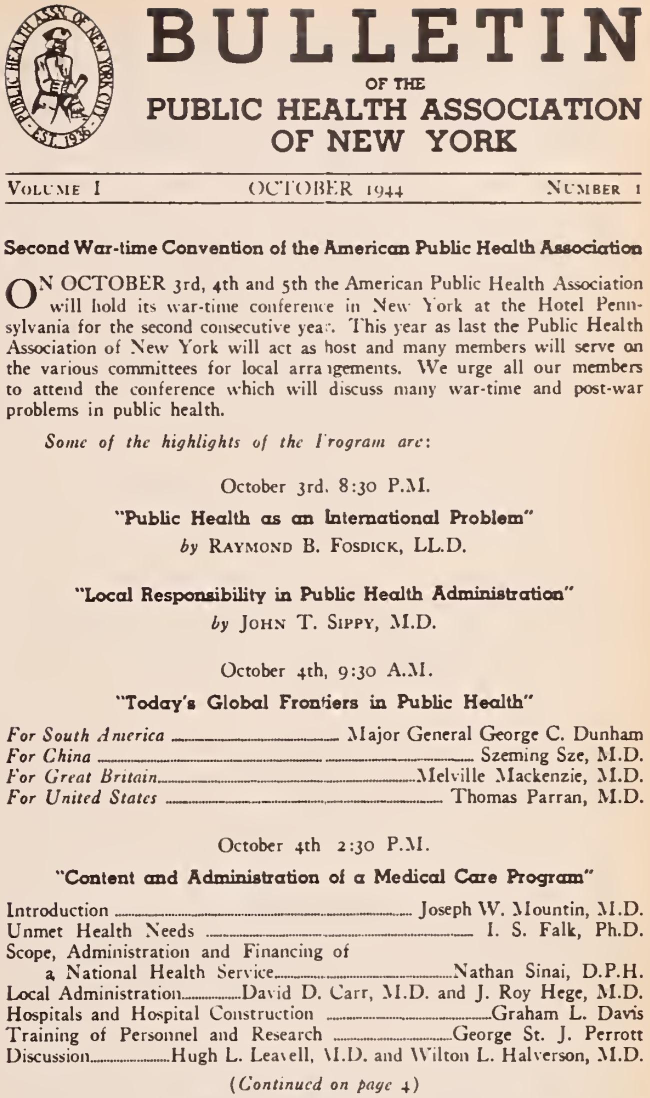
Dr Donald B. Armstrong said that the association "has helped to focus both professional and public opinion upon the outstanding needs and the more important developments of public health, not only in New York City, but elsewhere."

SUPPORTING ORGANIZATIONS

Leadership by active professionals at the New York City Department of Health unquestionably provided the strength for PHANYC's foundation. More than a dozen of the organization's 39 presidents have been former commissioners or high-ranking members of New York City government (Table II).

From the inception of the association, there was active corporate agency and institutional support in addition to that from the Department of Health. The Metropolitan Life Insurance Company hosted executive board meetings and otherwise encouraged the fledgling organization. Edward M. Dolinsky and Clarence Pearson at Metropolitan continued this tradition, as did Theodore Allison in more recent years.

Frank Kiernan was the Director of the New York Tuberculosis and Health Association and served assiduously as secretary-treasurer, delegate to the APHA Governing Council, and president by the tenth anniversary. Hazel Corbin, RN, general director of the Maternity Center, was an initial officer and then president, and provided strong leadership of nursing representation and support, as does Ruth Lubic, RN, the current director.



BULLETIN
OF THE
PUBLIC HEALTH ASSOCIATION
OF NEW YORK

VOLUME I | OCTOBER 1944 | NUMBER 1

Second War-time Convention of the American Public Health Association

ON OCTOBER 3rd, 4th and 5th the American Public Health Association will hold its war-time conference in New York at the Hotel Pennsylvania for the second consecutive year. This year as last the Public Health Association of New York will act as host and many members will serve on the various committees for local arrangements. We urge all our members to attend the conference which will discuss many war-time and post-war problems in public health.

Some of the highlights of the Program are:

October 3rd, 8:30 P.M.

"Public Health as an International Problem"
by RAYMOND B. FOSDICK, LL.D.

"Local Responsibility in Public Health Administration"
by JOHN T. SIPPY, M.D.

October 4th, 9:30 A.M.

"Today's Global Frontiers in Public Health"

For South America	Major General George C. Dunham
For China	Szeming Sze, M.D.
For Great Britain	Melville Mackenzie, M.D.
For United States	Thomas Parran, M.D.

October 4th 2:30 P.M.

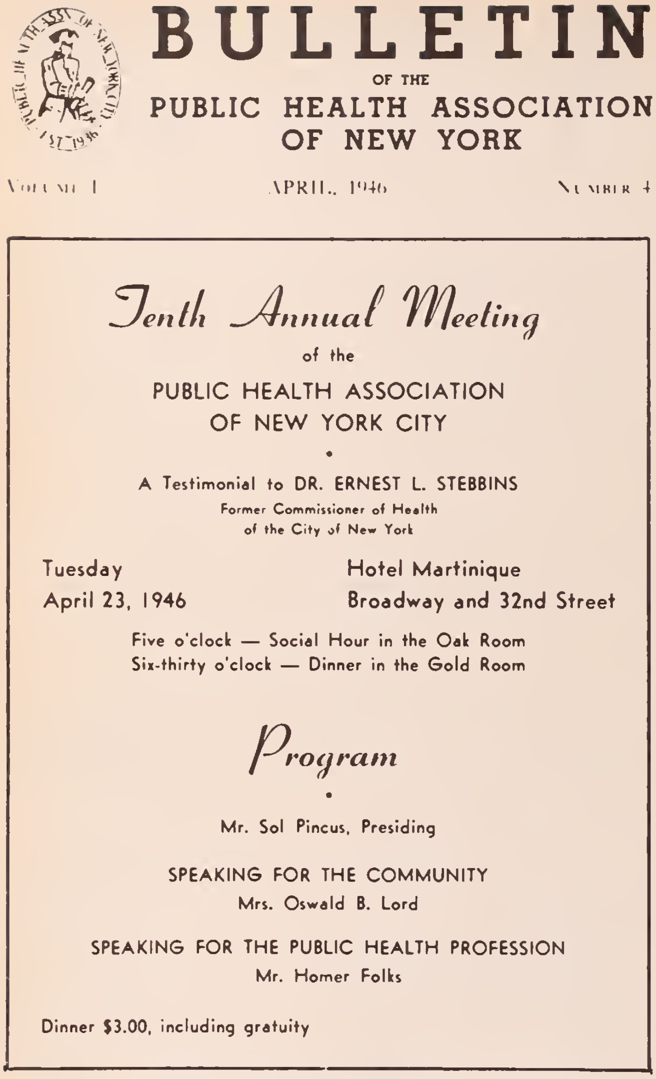
"Content and Administration of a Medical Care Program"

Introduction	Joseph W. Mountin, M.D.
Unmet Health Needs	I. S. Falk, Ph.D.
Scope, Administration and Financing of a National Health Service	Nathan Sinai, D.P.H.
Local Administration	David D. Carr, M.D. and J. Roy Hege, M.D.
Hospitals and Hospital Construction	Graham L. Davis
Training of Personnel and Research	George St. J. Perrott
Discussion	Hugh L. Leavell, M.D. and Wilton L. Halverson, M.D.

(*Continued on page 4*)

FIGURE 3. Front page of first *Bulletin*, October 1944, Volume 1, Number 1 (PHANYC archives).

Educational institutions provided scientific credibility to PHANYC's missions and professional leadership to the association. New York University was ably represented in the early days by Helen C. Mayer, PHD, associate professor of education in the School of Education, and the dean of the College of Engineering, Professor Thorndike Saville. Additionally, Haven Emerson, MD, professor of public health, Delamar Institute of Public Health at Columbia University, former Commissioner of Health of New York City (1914–1918), and active member of the board of health (1934–1956), contributed to policy and action in these early days. These ivory-tower-based idealists, along with the New York Academy of Medicine, provided evidence that issues could be identified and appropriately studied and discussed, and that valid solutions and information could be disseminated for action by those with



BULLETIN
OF THE
PUBLIC HEALTH ASSOCIATION
OF NEW YORK

Volume I   April, 1946   Number 4

Tenth Annual Meeting
of the
PUBLIC HEALTH ASSOCIATION
OF NEW YORK CITY

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A Testimonial to DR. ERNEST L. STEBBINS
Former Commissioner of Health
of the City of New York

Tuesday
April 23, 1946

Hotel Martinique
Broadway and 32nd Street

Five o'clock — Social Hour in the Oak Room
Six-thirty o'clock — Dinner in the Gold Room

Program

•

Mr. Sol Pincus, Presiding

SPEAKING FOR THE COMMUNITY
Mrs. Oswald B. Lord

SPEAKING FOR THE PUBLIC HEALTH PROFESSION
Mr. Homer Folks

Dinner $3.00, including gratuity

FIGURE 4. Tenth Annual Meeting notice in the *Bulletin*, April 1946, Volume 1, Number 4 (PHANYC archives).

TABLE II. Public Health Association of New York City Past Presidents

Donald B. Armstrong, MD	1936
Ralph Muckenfuss, MD	1937
Hazel Corbin, RN	1938–1939
Arthur I. Blau, MD	1940–1941
Helen C. Manzer, PhD	1942
Leveritt D. Bristol, MD, DrPH	1943–1944
Sol Pincus	1945
Frank Kiernan	1946
John L. Rice, MD	1947
Iago Galdston, MD	1948–1950
Harry S. Mustard, MD	1951–1952
Roscoe P. Kandle, MD	1953–1954
Duncan Clark, MD	1955
George Reader, MD	1956
Samuel Dooley, MD	1957–1958
Sylvan S. Furman	1959
Morton Schweitzer, PhD	1960
George James, MD	1961
Milton Terris, MD	1962
Alonzo S. Yerby, MD	1963–1964
Francis R. Gearing, MD, MPH	1965
Arthur Bushel, DDS, MPH	1966
Charlotte Muller, PhD	1967
Mary C. McLaughlin, MD, MPH	1968
Milton Lowenthal, MD	1969
Louis L. Feldman	1970
Lawrence Bergner, MD, MPH	1971
Marvin Lieberman, PhD	1972
Alden N. Haffner, OD, PhD	1973
Benjamin Wainfeld, MD	1974
Lloyd Novick, MD	1975
Peter B. Terenzio	1976
June J. Christmas, MD	1977
Beatrice Kresky, MD	1978
Samuel Wolfe, MD, DrPH	1979
Florence Kavaler, MD, MPH	1980–1981
Victor W. Sidel, MD	1982–1983
Frances A. McVey, RN, MA	1984–1985
Walter E. Brown, PhD	1986–1987
Michael E. Clark, MA	1988–1989

power and influence.

A host of other organizations have always been part of the greater PHANYC sphere of influence. Included among these are Blue Cross and Blue Shield of New York, Citizens Committee for Children, Milbank Memorial Fund, Women's City Club, New York Tuberculosis and Health Association, Community Service Society, and Visiting Nurse Association.

Board composition has evolved and parallels community trends to include citizen and lay participation in health planning and policy stimulated by the Office of Economic Opportunity (OEO) and community mental health legislation. Over the past 15 years, specific attention was directed to the ethnic and racial composition of the board to reflect better New York City's diversity and the special needs of these communities. On the current board are professionals working in the New York City and New York State Health Departments, the New York City Health and Hospitals Corporation, local hospitals, the Greater New York Hospital Association, the Academy of Medicine, United Hospital Fund, and academics from Columbia, State University of New York Health Science Center at Brooklyn (Downstate), Hunter College, Empire State College, and several community action agencies. Areas of specialization among board members include a full range of policy and practice activities concentrating on alcoholism, drug abuse, occupational health, ambulatory care, long term care, and maternal and child health, to indicate a few.

RELATIONSHIPS WITH THE AMERICAN PUBLIC HEALTH ASSOCIATION

PHANYC delegates to APHA's governing council have been active participants in the lively discussions at the annual meetings. Their representation of PHANYC viewpoints on various national position papers has clarified the issues, heated the debate, influenced voting, and earned PHANYC the respect of other affiliates.

At the national level, PHANYC's voice was prominent in testifying on the impact on health of federal budget transfers of funds to the military from human services; of foreign policy impinging negatively on health in the Third World, South Africa, Latin America, Cuba, Nicaragua;

of cold-war missile races and the buildup of nuclear weapons.

As one of the largest affiliates, PHANYC's influence was particularly felt in APHA discussions leading to adoption of a policy favoring the establishment of a National Health Service as key to resolving questions of equity, accessibility, and financing of health services to the American people.

PHANYC AWARDS

A great number of unsung PHANYC and agency members and citizens of New York City contribute to progress in public health. In 1955, PHANYC's first merit awards were presented to nine members of the New York City Department of Health for services beyond the purview of the respective official position held. In 1958, the Haven Emerson Award was initiated as the association's highest honor to those persons reaching the pinnacle of accomplishment in public health or in fields closely involved with it.

PHANYC developed a long term relationship with the Clara Barton High School in Brooklyn, awarding cash prizes to students for theme papers on important health problems or approaches to health improvement. There is a large minority enrollment at this school, and these teenagers are encouraged to pursue health careers and to be aware of the interrelationships of health and social problems. Board members have acted as role models, and with teachers jointly judge the contestants.

ADDRESSING CRITICAL ISSUES

Topics addressed in PHANYC's scientific subject meetings and annual gatherings indicate timely attention to critical issues in public health (Table III).

TABLE III. Meeting Topics—Selected Years

1938	Studies in Tuberculosis
1940	Water Supply for New York's Millions
1941	Three Diseases of Importance in the Aging Population: Diabetes, the Heart Diseases, and Cancer
1942	Public Health, Social and Legal Problems Connected with Prostitution
1945	Vitamins, Sulfonamides, Pencillin: Discussion of Problems of Current Importance
1948	Home Care as an Extension of Hospital Service
1955	Long-Term Illness in New York City
1958	What Can We Say to the Public about Radiation, Smoking, Obesity, Auto Accidents
1960	Alcoholism Today: Its Public Health Importance
1965	Health's Responsibility in Racial Integration
1968	The Concept of the Assistant Physician
1971	Urban Health Crisis and Recruitment of Minority Students
1973	Patients' Rights: Facts or Fancy
1974	Mental Health: A Service in Crisis
1975	Nursing Homes: A Look Into the Future
1978	Adolescent Pregnancies
1980	Protecting Workers' Health
1981	Prison Health Conditions: Implications for the Public Health
1983	DRGs: Reimbursement for the Future
1985	Low Birth Weights—A Public Health Issue

During the 1960s, in concert with much of the nation, PHANYC's attention was directed toward the impact of racial and ethnic discrimination on the health and social structure of American society. Health issues under major discussion included the promotion of family planning services; the encouragement of new health professions, such as nurse practitioner and physician assistant; improvement of salaries and working conditions for nurses who play the key service roles in public services. Consumer participation and involvement in Office of Economic Opportunity (OEO) centers, neighborhood family health care centers, community mental health programs, and hospital based ambulatory care gained prominence as did the implementation of the Medicaid program to reduce barriers to utilization and availability of services.

Joint sponsorship by PHANYC and the New York State Public Health Association of a state-wide conference on "Barriers to Utilization of Health Services," March 13–14, 1967, in Albany, brought together more than 160 participants

> to call attention to existing discrepancies in health status and medical care between the various socio-economic groups throughout New York State, and to urge and make recommendations for the institution of appropriate measures to ameliorate such inequities.[11]

In the 1970s, many of PHANYC's policy discussions and statements were directed at proposed federal legislation, including support for national health insurance as a natural expansion of the Medicare/Medicaid programs; and the federal Emergency Service Act for effective emergency pre-hospital care response systems for New York City. Dr June Jackson Christmas, former New York City Commissioner of Mental Health and then PHANYC's president, served on President Jimmy Carter's Transition Committee on Health.

PHANYC board members have consistently represented the organization on the Mayor's Organizational Task Force on Health Planning, the Comprehensive Health Planning Agency, and the Health Systems Agency of New York City, and many other task forces, committees, and technical advisory groups integral to planning health services and policy in New York City.

WOMEN'S HEALTH ISSUES

In 1977, PHANYC organized a Committee on Women's Concerns. Programs and speakers addressed selected topics such as battered women, domestic violence, self-care, reproductive health care, use of federal funds for abortion, and health care problems of lesbians. This committee participated in the Mayor's Task Force on Rape and also developed a Task Force in Occupational Health. Responsive to needs raised by members, sessions were also held on the topic "How Can Women Achieve Leadership Positions in the Health Care Field" and on "Understanding Power" to help women achieve effectiveness in their professional as well as personal lives.

Providing opportunities for membership networking with other women was an essential element in the success of this PHANYC effort.

THE 1980S

PHANYC's emphasis for the 1980s has been the public policy concerns to which the organization is dedicated: issues of the proprietization of health care institutions; the roles of medical education; voluntary and municipal hospital systems; the problems of financing health care and the emerging challenges of for-profit HMOs; the role of the academic health center.

There is a critical awareness of foreign policy issues as they relate to health—in Asia, Africa, and Latin America—as well as the ever-present spectre of militarization and nuclear war.

The historical record of the organization's struggles, educational activities, and policy positions provides testimony to PHANYC's role as a "professional conscience for the health and welfare community and an advocate for progressive change." [12]

Moreover, "It has been an advocate for quality and access; it has been a critic of injustice and discrimination. It continues to remind the public sector and private and voluntary institutions benefitting from public funds of a shared responsibility for the health of the public." [13]

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Vesical calculus, lithotrity, and Napoleon III

JAIN I. LIN, MD

Vesical calculus has afflicted human beings since ancient times. Without sufficient anatomical knowledge, medical pioneers boldly performed lithotomy, one of the earliest known surgical techniques, to remove the bladder stones. There is evidence indicating that perineal lithotomy was practiced by the ancient Egyptians and Indians.[1] This operation, and others similar, was also mentioned in Hippocrates' oath, which states that a physician will not cut for the stone and will leave the operation to those so trained. The first accurate description of perineal lithotomy is found in *De Medicine* written by Celsus of Rome around 30 AD.[2] Lithotomy as a procedure remained virtually unchanged for the next 18 centuries, except for minor modifications and the introduction of new instruments. However, at the end of the 19th century, the high mortality rate associated with lithotomy due to postoperative infection forced surgeons to seek a safer method for removing bladder stones. Lithotrity became the operation of choice.

LITHOTRITY

Lithotrity, or the crushing of bladder stones, was said to have been known to Ammonius of Alexandria about 230 BC.[1] The operation was also recorded in the writings of Byzantine physicians. Later, two 17th century Italian physicians, Santorio and Cinci, won fame by inventing stone-crushing. But, it was Jean Civiale (1792-1867), the foremost urologist of his day in France, who was credited with introducing lithotrity as a sound clinical technique.[3] As a second year medical student in 1817, Civiale started a series of experiments in crushing bladder stones. After perfecting his technique and instruments, he succeeded in operating on his first patient in 1823. His instruments were formidable and mainly consisted of a straight metal catheter with a claw-shaped end by which the stone was seized and held in position[4] (Fig 1). Then a rod was passed through the catheter and used to forcibly break the stone. The fragmented stones were then expelled by normal bladder action. Civiale limited his operation to five minutes in length and to stones less than 2 cm in diameter. In his lifetime, he treated 2,200 patients with vesical calculi, 1,600 of whom were operated on by lithotrity. He attributed his success to meticulous preoperative preparation.[3]

After strong advocacy by Civiale, the value of lithotrity was gradually recognized by the rest of the European medical community. Many surgeons began to practice lithotrity, simultaneously trying to improve on Civiale's instruments and technique. Among these was Sir Henry Thompson (1820-1904) (Fig 2).[3] While he was assistant surgeon at the University College Hospital of London, he

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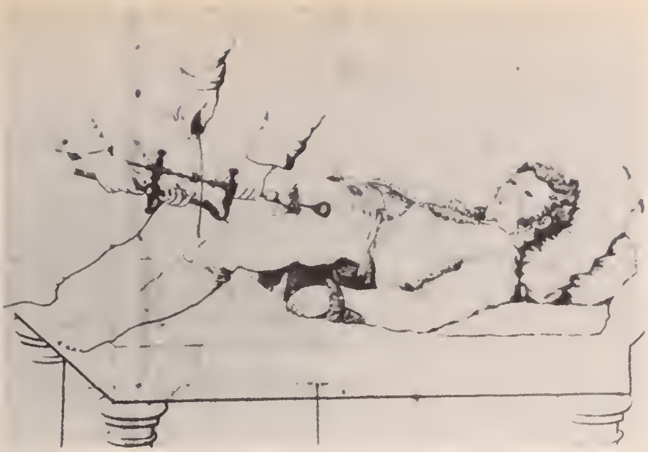

FIGURE 1. Lithotrity (reproduced from Civiale[4]).

distinguished himself in genitourinary surgery by publishing treatises on pathology, the treatment of urethral stricture, and prostatic disease. In 1858, he visited Paris and studied Civiale's technique in bladder stone removal by using a large, powerful, evacuating catheter, immediately followed by the use of a special suction apparatus. His reputation was so great that in 1863, he was called upon to treat Leopold I, King of the Belgians, whose bladder stone Civiale had failed to remove 18 months previously. Thompson successfully crushed the king's stone with one stroke and was subsequently knighted by Queen Victoria for his skill.[5] Nine years later, Thompson was summoned again to treat another European ruler, Napoleon III, who had been troubled with stones for many years. This time Thompson did not succeed.[3,5]

FIGURE 2. Sir Henry Thompson (reproduced with permission from Power[3]).

NAPOLEON III

Napoleon III (1808–1873) was a nephew of Napoleon Bonaparte (Fig 3). Although he was not as brilliant as his famous uncle, he created the Second Empire in France, which lasted almost 20 years.[6] During his reign, he played an important role in international politics, such as allying with Great Britain to stop Russian expansion in the Crimea, supporting the unification of Italy, and helping to establish the Red Cross. Nonetheless, he was not a healthy man. In addition to being afflicted with rheumatism, gout, hemorrhoids, and skin sensitivity, Napoleon III suffered in later life from chronic cystitis caused by bladder stones. In 1853, one year after he became the emperor, a rumor spread in Paris that he had bladder trouble. In 1855, Jobert de Lamballe, a French physician, diagnosed the problem as bladder spasm.[7] In 1861, the emperor's private physician, Henry Conneau, suggested a recuperative visit

FIGURE 3. Napoleon III (reproduced with permission from Williams[8]).

to Vichy. In retrospect, recuperating at Vichy was ill advised, since its fountain was alkaline and rich in minerals that might have aggravated stone formation. In 1864, the emperor experienced hematuria while observing military maneuvers at the Camp de Chalons.[7]

On August 15, 1865, Baron Larry, one of the emperor's physicians, was summoned to see him during the night because the emperor was having difficulty in urinating. After questioning the emperor about his symptoms and observing the sand in his urine, Larry came to the conclusion that the emperor was suffering from bladder stones. He advised the emperor against going to Vichy for recuperation, but the emperor persisted. On July 29, 1866, another physician, Franchois-Gabriel Gullon, probed the emperor with a tiny rubber device and then had him get into a warm bath into which the emperor was able to urinate a little.[8(p178)] After probing three times, the emperor's difficulty in urination seemed to improve. In the last half of 1869, the emperor experienced hematuria, dysuria, and pyuria.

At this time, his defense minister, Marshal Niel, was stricken with bladder stone. Auguste Nelaton, the inventor of the rubber catheter, decided to treat the marshal with lithotrity. Soon after treatment, Niel died of complications.[8(p126)] Niel's death gave the emperor good reason to lose confidence in the treatment of the bladder stone. On June 19, 1870, on the recommendation of a relative, Anna Murat, the emperor agreed to be examined by Germain See, a clinical professor at the Paris Medical School.[8(p134)] Professor See was stunned by the emperor's physical weakness. After the examination, he told the emperor he had a bladder stone and asked to discuss treatment with his physicians. The emperor reluctantly agreed. (The emperor's bladder ailment had never been disclosed to the public, which had been informed that the emperor had a rheumatism.[7])

On July 1, a grand medical consultation took place at the residence of Conneau. In addition to Conneau, Corvisart, Nelaton, Ricord, Fauvel, and See attended the meeting. See insisted that the emperor required an exploratory sounding as a preliminary step to an operation to crush the stone.[8(p138)] But the other physicians favored a more conservative approach. At the end of the meeting, See wrote a report that was supposed to be delivered by Conneau to the emperor for review. His report stated that frequent episodes of hematuria, pyuria, dysuria, and interruption of urination all strongly indicated that the emperor had cystitis due to a calculus that required further exploration and treatment.[8(p139)] Apparently, Conneau did not show the report to the emperor, who, despite ill health, went to the front line when the Franco-Prussian war broke out two weeks after the medical consultation. Years later, Empress Eugenie said that Conneau never mentioned the seriousness of the emperor's cystitis to her.[7]

A Prisoner at Sedan. Instead of riding a horse to lead his troops, as was his custom, the emperor took a train and carriage to the battlefield.[9(p563)] He frequently got off from the horse, whose jolting increased his pain. He was seen to cling to a tree in agony because of his difficulty in urination. The ill-prepared French troops were soon overrun by the well-equipped Prussian army. On September 1, 1870, Napoleon III and his army were trapped at Sedan. During the siege, he intentionally exposed himself to enemy fire. The following day, he ordered his armies to hoist a white flag to avoid any unnecessary sacrifice. Within one day, the emperor and 100,000 soldiers became prisoners of war. As soon as the news of capitulation reached Paris, the Second Empire collapsed and a Republican government was established.[9(p570)]

Ill-Fated Treatment. Napoleon III was released in the spring of 1871 after a peace treaty was signed between France and Prussia.[9(p581)] He chose exile in Great Britain and lived with his family at Camden Place, Chislehurst, in Kent. His bladder condition apparently remained stable until the summer of 1872. After learning of Napoleon's illness, the Prince of Wales immediately sent his private physician, William Gull, to see him. Gull recommended that his colleague Thompson examine the emperor. After his examination, Thompson informed Napoleon that he had a bladder stone that required immediate exploration. Napoleon, however, refused. Subsequently James Paget, the best known surgeon of the day, visited him and advised him to accept the treatment prescribed by Thompson. In December, Napoleon experienced hematuric episodes associated with perineal pain. Conneau consulted with Paget, Thompson, and Gull. Given the overwhelming advice to do so, Napoleon finally agreed to allow Thompson to perform a sounding, which disclosed a pigeon's-egg-sized stone in the bladder.[8(p164)]

On January 2, 1873, Thompson first performed a lithotrity on Napoleon, who was anesthetized by chloroform. Because of its large size, Thompson failed to crush the stone completely. After the operation, Napoleon passed a large amount of blood, pus, and fragments of a phosphate stone. On January 6, Thompson performed a second lithotrity.[8(p166)] Soon after the second operation, Napoleon suffered fever and chills. The following day, more blood appeared in the urine. Napoleon became lethargic and stuporous. Thompson began to suspect that the fragments might be obstructing the prostatic urethra. On January 9, a third operation was arranged to clear the prostatic urethra. However, Napoleon's general condition quickly deteriorated, with gradual loss of consciousness.[8(p166)] "Etais-tu a Sedan?" were his last words, murmured to Conneau. He expired at 10:45 AM at the age of 64.

Autopsy. The following day, the Empress Eugenie granted permission for an autopsy to be performed on Napoleon. The postmortem was performed by the celebrated anatomical pathologist, Burdon Sanderson of London University, and attended by many physicians previously involved in Napoleon's care. The autopsy disclosed subacute inflammation of both kidneys, bilateral dilation of the renal pelvis, severe hydronephrosis of the left kidney with atrophy of the parenchyma, bilateral hydroureter, and subacute inflammation of the urinary bladder and the prostate. In addition, a stone, weighing about 0.75 oz and measuring $1\frac{1}{4} \times 1\frac{5}{16}$ in was found in the bladder. The stone consisted of urates with a coating of phosphates.[10,11] The other organs demonstrated no remarkable findings. Sanderson concluded that the cause of death was circulatory and renal failure precipitated by the patient's deteriorated condition. In the end, all physicians except Gull signed their names to the autopsy report, which was subsequently published in the *British Medical Journal* and

London Times.[8(p169)] Back in his office, Gull independently issued a statement that he thought the phosphate layer of stone was the result of previous cystitis, not the cause of it, but that otherwise he agreed with all the postmortem findings.

Controversy. When the news of Napoleon's death was announced, it caused an uproar in the French community. The newspapers and French physicians criticized Thompson for what they considered to have been a bungled operation. J. Lapeyrere argued that in view of the size of the stone and the age of the patient, lithotomy and not lithotrity should have been the treatment of choice. He also pointed out that the interval between lithotrities was too short for the debilitated patient to recuperate between the procedures. Another rumor circulated that Napoleon died of an overdose of chloroform. This additional medical controversy was further aggravated by Gull's refusal to sign the report, which fueled public suspicion that there was disagreement among Napoleon's physicians regarding treatment.[8(p172)] Nonetheless, based on the autopsy findings, there is no doubt that Napoleon died of uremia and sepsis. Finally, one historian made a fitting remark on Napoleon's tragic death: "Whatever the cause of his physical death, in an historical sense, he had already died at Sedan." [9(p589)]

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SOME PRACTICAL POINTS IN THE TREATMENT OF STONE IN THE BLADDER, WITH AN ANALYSIS OF EIGHT NEW CASES

. . . In the management of cases of vesical stones, as in that of other surgical ailments, the exercise of sound judgment is quite as important as the highest degree of operative skill, and the two should go hand in hand. It seems to me unwise to rely entirely upon any single method of treatment or of operation, for, in reality, there is no exclusively *best* method in surgery, no particular one that should be used in all cases, even of a kind. It is often said, and perhaps with much truth, of surgeons who have some pet operation, that they select their cases for this operation, and reject all others that appear unfit for it, or even unpromising. Judicious surgeons ride no hobby, but select, from among the many known operative procedures, that which, after due consideration and study of each case, is likely to be indicated. Some authorities take the ground that the operator should select the operation with which he is most familiar. They speak of excellent lithotomists who are bad lithotriptists, and *vice-versa*. Can this be called sound teaching? Suppose in a certain case, the particular operation of the one-operation surgeon to be contraindicated, could any but the worst result be anticipated? The one-operation man may be an excellent operator, but may be very unsuccessful with his one operation, and many of his patients die, while another surgeon, who may not operate so brilliantly, but makes a proper selection of operation, cures nearly all his cases. Is not every surgeon who wishes to treat calculous patients, in duty bound to labor hard and long to familiarize himself with all the known methods, and if he cannot master them all, is he justified in undertaking any? If he acts in accordance with the principles of sound surgery, he cannot be a pure lithotomist, lithotriptist, or perineal lithotritist, nor can he even have any particular hobby in lithotomy, regarding one method or another, but is competent to choose the operation which appears, after mature consideration, most suitable to the case. We all know that one patient will bear immediate surgical operation, be it lithotomy or lithotripsy, while another, of the same age, and, on a superficial examination, apparently in the same state, will be killed by precisely the same treatment as that resorted to in the first case, whereas, a few days of rest and other preparatory treatment, would have ensured success. Is it not better to prepare all cases for operation?

Sound judgment and experience, I will repeat, are the only guides in such circumstances. . . .

JOHN W. S. GOULEY, MD
(*Trans Med Soc State NY* 1877; 171-181)

Temporal arteritis and intestinal perforation

Report of three cases

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Temporal or giant-cell arteritis is an adult-onset, focal granulomatous inflammatory disorder of small- and medium-sized arteries which primarily affects the cranial vessels but which may in more severe cases affect virtually any arterial system. The cause of this disorder is unknown but its dramatic clinical response to corticosteroids suggests an underlying immune mechanism.

This report describes three patients with biopsy-proven temporal arteritis in whom intestinal perforation or fistula formation developed following resolution of cranial manifestations of the disease on steroid therapy.

CASE REPORTS

Case 1. A 75-year-old woman experienced progressive blurred vision and loss of visual fields in February 1984. A temporal artery biopsy in May 1984 demonstrated giant-cell arteritis. No extra-ocular symptoms were noted, and the medical history was unremarkable for gastrointestinal disease. Oral prednisone, 100 mg/day, was initiated, with immediate improvement in symptoms. After one week of steroid therapy, the patient complained of mild periumbilical pain. Physical examination disclosed diffuse abdominal tenderness with mild rebound, although the patient was afebrile. A plain film of the abdomen demonstrated free air under the diaphragm. At emergency laparotomy, a perforated duodenal ulcer was found and oversewn. The postoperative course was unremarkable, and prednisone was gradually tapered to 15 mg/day over the next six months. No additional gastrointestinal problems were encountered, and the patient currently remains symptom free.

Case 2. A 74-year-old woman presented in March 1985 with a three-week history of headaches and scalp tenderness. No extracranial complaints were noted. The erythrocyte sedimentation rate was 134. The medical history was remarkable only for cholecystectomy for cholelithiasis, mild hypertension, and a previous abdominal hysterectomy in 1951 for intractable uterine bleeding. Prednisone, 60 mg/day, was started, and after two months the symptoms completely resolved. Steroids were gradually tapered to 15 mg/day. In August 1986, the patient complained of feculent vaginal discharge but had no fever, nausea, vomiting, abdominal pain, or rectal bleeding. An upper gastrointestinal radiographic series was normal, and barium enema studies revealed small diverticuli only. An exploratory laparotomy revealed a colovaginal fistula. A salpingo-oophorectomy, resection of rectosigmoid, sigmoid colostomy, and Hartmann pouch were performed on August 14. No evidence of giant-cell arteritis was found in the resected portion of the intestine. Prednisone was decreased to 10 mg/day. The colostomy was uneventfully reversed in February 1987, and the patient remains symptom free.

Case 3. A 79-year-old woman with a history of mild hypertension and arthritis was diagnosed as having giant-cell arteritis after presenting with left eye blindness in March 1984. Oral prednisone, 100 mg/day, was started, and symptomatic improvement occurred within one week. Steroids were tapered to 15 mg/day due to the development of thrombocytopenia, which ultimately required gammaglobulin for control, although the patient was otherwise symptom free. There was no history of gastrointestinal disease or extra-ocular manifestations of giant-cell arteritis. In August 1984, the patient complained of generalized malaise and was noted to have an abdominal pelvic mass. No fever or leukocytosis was present. Barium enema was remarkable for mass effect only. An exploratory laparotomy the following day revealed spontaneous perforation of the small intestine with an abdominal wall abscess and generalized peritonitis. Resection and drainage of the abdominal wall abscess, omentectomy, and multiple resections of the jejunum with staple reanastomosis were performed. Pathologic analysis of the resected portions of intestine showed necrosis and inflammation, but no evidence of giant-cell arteritis.

The postoperative course was marked by the development of septic shock, adult respiratory distress syndrome, and enterocutaneous fistula formation, which necessitated prolonged ventilatory support, antibiotic therapy, and intravenous hyperalimentation. Prednisone was tapered to 10 mg/day, and the patient was discharged after 45 days in the intensive care unit. Readmission in March 1985 for sepsis and jaundice again resulted in an emergency laparotomy for peritonitis, subsequently complicated by a subcapsular liver hemorrhage, disseminated intravascular coagulation, and septic-hypovolemic shock. Despite intensive resuscitative efforts, the patient died shortly after surgery.

DISCUSSION

With corticosteroid therapy giant-cell arteritis frequently pursues a relatively benign, indolent course which often results in complete clinical remission and allows resolution of ocular and extracranial symptoms. Symptom response often occurs within two to three days of initiation of therapy, although rarely overwhelming systemic involvement results in a fatal outcome. The gastrointestinal symptoms of temporal arteritis often consist of abdominal pain and diarrhea. Intestinal perforation or fistula formation per se has not been previously reported to occur as a result of or in association with this disease.

On the microscopic level, giant-cell arteritis can be identified by marked inflammation with intimal proliferation followed by degeneration and fragmentation of the internal elastic membrane with the appearance of multinucleate giant cells. These pathologic lesions appear in a segmental fashion rather than

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along the entire length of the vessel.[1] When such change involves the coronary and mesenteric arteries, the resulting ischemia and subsequent infarction may cause cardiac and gastrointestinal dysfunction in addition to the classic headaches and visual impairment that are produced by cranial vessel involvement.

All patients in this series had biopsy-proven giant-cell arteritis, an absence of extracranial manifestations of disease, a dramatic response of ocular and cranial symptoms to oral prednisone therapy, and some form of intestinal perforation within six months of treatment. Although abdominal pain and diarrhea have frequently been reported as intestinal manifestations of giant-cell arteritis at the time of diagnosis, intestinal perforation and/or fistula formation has not been previously reported as either a presenting sign or complication of this disease, as it has with rheumatoid arthritis,[2] even on steroid therapy.[3]

The absence of histologic evidence of giant-cell arteritis in the resected portion of intestine in two of the three cases may be the result of corticosteroid therapy. Reversal of arterial lesions has in fact been demonstrated by serial angiography in patients with documented giant-cell arteritis following successful steroid therapy. Giant-cell arteritis and Crohn disease have also been associated,[4] but no evidence of the latter was found in the present cases.

More disturbing, though, is the relative paucity of severe symptoms and fever in these patients despite overt intestinal perforation and the absence of known risk factors for spontaneous perforation or fistula formation. This may also in part be due to prolonged or high-dose corticosteroid therapy. Physicians caring for patients with giant-cell arteritis need to be aware of the possibility of intestinal perforation in such patients despite notable initial improvement in symptoms on steroids. Furthermore, despite excellent long term control of symptoms on low-dose prednisone and a lack of known gastrointestinal disease, physicians must keep occult intestinal perforation in mind for those patients with giant-cell arteritis in clinical remission who present with vague abdominal pain, or a clinical picture less severe than that of the classic "acute abdomen."

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Pulmonary oncocytoma

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Oncocytomas are rare tumors composed of epithelial cells that have abundant eosinophilic granular cytoplasm. Since their first description in the salivary gland in 1926,[1] oncocytomas have been described elsewhere in the body including the lung. Fechner and Bentinck[2] first described a pulmonary oncocytoma with ultrastructural confirmation in 1973. Thus far, seven cases have been reported in the English literature, all of which were confirmed by electron microscropic studies. Immunohistochemical investigations were not performed in any of these cases. We present another case of this rare tumor and, in addition, we present the results of the associated immunohistochemical studies.

CASE REPORT

A 74-year-old man was brought to the emergency room for slurred speech and weakness on the right side of the face. A computerized axial tomographic (CAT) scan of the head revealed a small lacunar infarct in the left basal ganglia. As part of the general work-up, a chest film was obtained which revealed a 3.0- to 3.5-cm oval mass in the right lower lobe (Fig 1). Computed tomography of the chest revealed a mass in the same area (Fig 2) which contained a small calcific component in its right lateral border. Appropriate management was given for the cerebral ischemic infarct. A right lower lung lobectomy was subsequently performed, and the patient's postoperative course was uneventful. Neurologic deficits cleared by the time of discharge.

The lobectomy specimen consisted of a wedge-shaped portion of lung tissue measuring 6.5 × 4.0 × 2.7 cm in maximum dimension. The pleural surface was unremarkable. On palpation, a semifirm mass was noted. Sections showed a well-demarcated, tan brown mass measuring 2.5 cm in greatest diameter. Focal areas of hemorrhage and calcification were noted. The remaining lung parenchyma appeared congested.

Microscopic examination showed an alveolar arrangement of tumor cells having abundant, finely granular, eosinophilic cytoplasm and uniform small, centrally located hyperchromatic, oval to round nuclei with prominent nucleoli (Fig 3). Mitoses were rarely seen. Narrow fibrous septae separated the nests of tumor cells. Focal areas

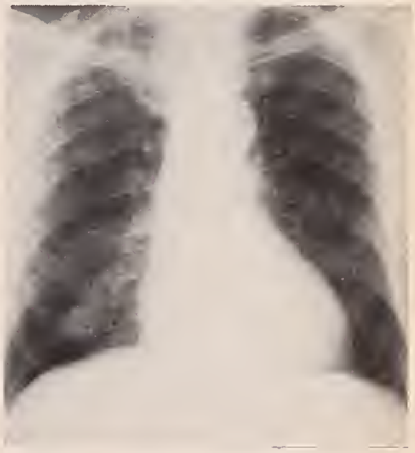

FIGURE 1. Chest film (anterior-posterior view) showing a circular nodule at the periphery of the right lower lung field.

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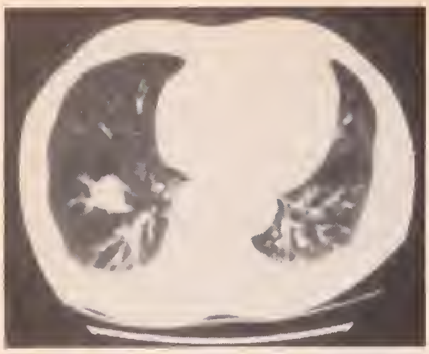

FIGURE 2. Computed tomography of the chest showing a radiodense nodule in the right lower thoracic cavity.

with an organoid pattern were also noted. Grimelius staining showed focal positivity in the tumor.

On ultrastructural analysis, the cells had distinct cellular borders with few desmosomes. The nuclei were round to oval with dispersed chromatin and prominent nucleoli. The cytoplasm was filled with numerous mitochondria and contained a few nonmembrane-bound electron dense granules ranging from 100 to 150 nm in diameter (Fig 4). These are smaller than the usual neurosecretory granules.

Immunoperoxidase staining using the peroxidase-antiperoxidase technique was performed on paraffin sections from the tumor. Stains for desmin, neuron specific enolase, epithelial membrane antigen, and S-100 were negative. Stains for cytokeratin and vimentin were equivocal. Appropriate positive and negative controls were utilized.

DISCUSSION

Stohr and Risak[1] were the first to describe a salivary gland tumor in 1926 which consisted of oncocytes. However, it was Jaffe[3] in 1932 who introduced the term "oncocytoma" for a benign salivary gland tumor that had a predominantly oncocytic population. Since then, oncocytic tumors have been described elsewhere in the body, and ultrastructural criteria have been defined to distinguish true oncocytomas from tumors with oncocytoid features.

Various cytoplasmic organelles can result in an oncocytic appearance on light microscopy, including hypertrophy of the smooth endoplasmic reticulum, abundance of lysosomes, or secretory granules. The true oncocyte, however, shows mitochondrial hyperplasia on electron microscopy. Thus, true oncocytomas include only the following: Warthin tumor, oxyphil cell adenoma of the parotid gland, oncocytic adenoma of the pituitary gland, Hürthle cell tumor of the thyroid, fibrolammellar oncocytic hepatoma, intracystic oncocytic papilloma of the breast, renal oncocytoma, ovarian oncocytoma, and bronchial oncocytoma.[4]

Bronchial oncocytomas are extremely rare pulmonary tumors and were first described by Hamperl[5] in 1937 using only light microscopy. He then considered it to be a type of bronchial carcinoid. Black,[6] in 1969, later described the ultrastructural features of a "bronchial oncocytoma;" he noted large argyrophilic dense core vesicles in the cytoplasm as opposed to the smaller secretion granules he found in another case of "bronchial carcinoid." The term oncocytoma was apparently a misnomer, since his case lacked the strict ultrastructural criterion of mitochondrial hyperplasia.

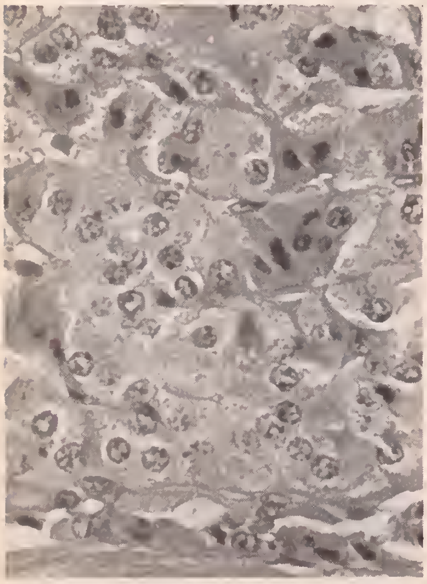

FIGURE 3. Tumor cells with uniform polygonal eosinophilic granular cytoplasm and round to oval nuclei (hematoxylin-eosin stain; original magnification ×1,350).

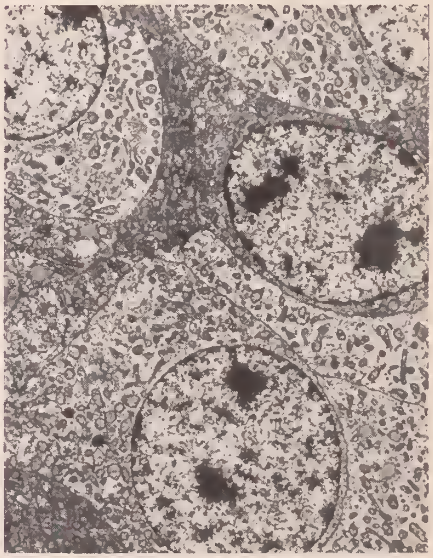

FIGURE 4. Electron micrograph showing tumor cells with numerous mitochondria within the cytoplasm. A few electron dense granules are also found (uranyl acetate-lead citrate stain; original magnification × 2,800).

Fechner and Bentinck[2] in 1973 first described a bronchial neoplasm composed of true oncocytes that exhibited an increase in mitochondria on electron microscopy. The authors interpreted the tumor described by Black as being an acinic cell tumor of bronchial origin that was neither an oncocytic carcinoid nor an oncocytoma.

Thus far, seven patients with oncocytoma in the lung have been reported in the English literature (Table I). Since not all tumors are bronchial in location, it is only fitting to use the general term "pulmonary oncocytoma." These patients, including the present one, ranged in age from 22 to 75 years, the median age being 53 years. All except one patient were male.[7] Five gave a history of smoking. Presenting symptoms were nonspecific and included malaise, low back pain, and chest pain. Chest radiographs usually revealed a well circumscribed density with a predilection for the right side. Only in one reported case was the lesion located on the left. The majority of tumors were bronchial in location. In three cases (cases 2, 4, 8; see Table I), the tumor was located in the lung parenchyma, and no relation to the bronchus was identified on the gross specimen. Bronchoscopy revealed partially occluding, solid polypoid masses in those tumors that were bronchial in location. A preoperative diagnosis of carcinoid was made in three cases (cases 1, 4, 6).

On macroscopic evaluation, the tumors were mostly single, except in one case[7] which consisted of multiple nodules. They were moderately firm, from 1.0 to 3.5 cm in diameter, and showed a solid homogeneous cut surface of varying colors. Focal hemorrhage and calcification were seen in the present case. Histologic and ultrastructural analysis demonstrated the classic oncocytic features of eosinophilic granularity in the cytoplasm due to mitochondrial hyperplasia. In two cases, however, membrane-bound dense bodies were noted. Fechner and Bentinck[2] identified membrane-limited dense bodies but clarified that these were smaller (60 nm) than neurosecretory granules found in carcinoid. Fernandez and Nyssen[8] also found few membrane-bound dense bodies but did not characterize these granules, so the possibility exists that their case is not a true oncocytoma but rather an oncocytic carcinoid. The present

TABLE I. Cases of Pulmonary Oncocytoma with Electron Microscopic Confirmation Reported in the Literature

Author	Physical Examination	Chest Film	Bronchoscopic Findings	Gross Appearance
Fechner & Bentinck[2]	68 yr; male; smoker; malaise	Right upper lobe infiltrate	Narrowed right upper lobe bronchus	3.5-cm solid yellow-tan mass
Santos-Briz et al[16]	22 yr; male; smoker	Round sharp shadow right lower lobe; Possible hydatid cyst	—	3.0-cm uniform red brown, firm mass
Fernandez & Nyssen[8]	30 yr; male; smoker; sudden cough & hemoptysis	Coin lesion right lower lobe	Cherry red mass blocking right lower bronchus	1.5-cm oval pink mass
Tesluk & Dajee[18]	53 yr; male; smoker; low back pain	Right lower lobe spherical mass; aspirate; possible carcinoid	—	Fragments of tan-gray firm tissue
Nielsen[7]	40 yr; female; weight loss, pneumonia	—	Right lower lobe bronchial tumor	Multiple nodules, 10–15 mm, solid cut surface
Cwierzyk et al[19]	57 yr; male; smoker; chest pain	Left lung root infiltrate extending to left upper lobe	Partial occlusion left upper lobe bronchus; cytology questionable carcinoid	1.5-cm polypoid mass
Warter et al[20]	75 yr; male	Left lower lobe infiltrate	Nodule on wall of right main bronchus	3.0-mm tan pink nodule
Present case	74 yr; male; stroke victim	Right lower lobe density	—	2.5-cm tan yellow mass; focal hemorrhage and calcification

case also had few electron dense granules but they were not membrane-bound and were of a size smaller than the usual neurosecretory granules. It is therefore felt that this is a bona fide oncocytoma.

The origin of oncocytomas is a matter of controversy. Oncocytes can be identified in normal aging bronchial mucous glands.[9-11] Hamperl[5] believed it to be a sign of degenerative or metaplastic change. Sun[12] speculated that oncocytomas are an "intracellular neoplasm of mitochondria" or, in other words, a "mitochondrioma." Ghadially,[4] however, proposed the possibility that mitochondria in the tumor cells are biochemically defective and thus undergo compensatory hyperplasia. There may likewise be an increased lifespan of the organelle or decreased rate of elimination.

There is an even greater controversy as to whether pulmonary oncocytoma is merely a variant of carcinoids or whether it belongs to a separate category. Spencer,[11] for instance, describes the bronchial oncocytomatous carcinoid as being distinct from the bronchial oncocytomatous adenoma, though he adds that most "bronchial oncocytomas" reported are probably bronchial carcinoids, as in the case cited by Black.[6] Carter and Eggleston[13] agree with this distinction.

Furthermore, a number of bronchial neoplasms have been described as being composed of a biphasic population of oncocytic and carcinoid cells. This leads one to suppose that these two tumors actually comprise a spectrum of a single entity that has varying degrees of oncocytic and carcinoid differentiation. Walter et al[14] and Sklar et al[15] each described a bronchial tumor composed of a mixture of pure oncocytes and pure carcinoid cells as defined by electron microscopy. Walter et al[14] also found cells of "intermediate type" having few neurosecretory granules and abundant mitochondria. The common origin of carcinoid cells and oncocytic cells from a single progenitor is thereby postulated.

At present, the distinction of oncocytic and carcinoid cells rests purely on ultrastructural grounds. It is felt that these biphasic tumors described are, as their authors claim, merely evidence of oncocytic metaplasia of bronchial carcinoids, and this is by virtue of the presence of neurosecretory granules. Once these are identified in a tumor, then it fulfills the morphologic criterion for the diagnosis of carcinoid, whether or not oncocytic changes are seen. In the absence of these granules and until the origin of oncocytes is clarified, we believe that the distinction still holds.

The immunohistochemical properties of pulmonary oncocytomas have never been fully characterized. Only Santos-Briz et al[16] and Nielsen[7] resorted to fluorescence microscopy and detected no serotonin or catecholamines. Immunoperoxidase stains were not used, but theoretically should aid in distinguishing carcinoid from oncocytoma. Neuron specific enolase was negative in the tumor described here, which is consistent with a non-carcinoid mass. Epithelial membrane antigen was negative, although Heyderman et al[17] showed that bronchial mucous glands contain epithelial membrane antigen. Staining of oncocytes was, however, not mentioned. So little immunohistochemical investigation has been performed on normal and neoplastic oncocytes that it is difficult to make a conclusion based on our results. We feel strongly, however, that immunohistochemical studies might shed more light on the nature of these cells, and also that of the various dense bodies found in oncocytomas, and may even resolve the controversy regarding cell origin.

Clinically, oncocytomas of the lung have proven to be benign in behavior except for the case described by Nielsen,[7] which was locally infiltrative and had parabronchial lymph node metastasis.

Oncocytomas are subclassified as benign or malignant; although clinical behavior is difficult to predict from light microscopic and ultrastructural findings alone. Immunohistochemical studies may aid in the diagnosis, but the ultimate answer lies in a close follow-up of cases and in meticulous sampling of tumors to detect non-oncocytic elements.

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Common duct stone with associated *Clonorchis sinensis* infection

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Parasitic infection is a known cause of common bile duct obstruction. *Ascaris lumbricoides* and *Fasciola hepatica* are the parasites most commonly implicated.[1,2] *Clonorchis sinensis*, however, is not commonly seen in Western countries.[3] *Clonorchis* infection frequently results in cholangiohepatitis often associated with secondary *Escherichia coli* infection, and may obstruct the common bile duct if associated with biliary stricture, stones, or tumor.[2] The following is a report of a patient with gallstones, a common bile duct stone, and associated active *Clonorchis* infection of the bile ducts.

CASE REPORT

A 59-year-old male Chinese immigrant was admitted to the hospital complaining of epigastric and right upper quadrant pain, associated with nausea and vomiting of one day's duration. He denied weight loss, jaundice, acholic stools, or a history of hepatitis. He had lived most of his life in Hong Kong and had immigrated to the United States three years previously.

Physical examination revealed a thin, nonicteric Oriental man. His temperature was 37°C, and the vital signs were stable. There was voluntary guarding over the right upper quadrant, with minimal rebound tenderness over the epigastrium and right upper quadrant. Rectal examination revealed brown stool negative for blood. The chest film and electrocardiogram were within normal limits. The abdominal films were normal. The white blood cell count was 15,000 k/mm^3 with 81% segmented neutrophils, 1% stab, 16% lymphocytes, and 2% monocytes. Laboratory studies demonstrated a hemoglobin of 12.3 g/dL; hematocrit, 38.3%; normal electrolyte levels; blood urea nitrogen, 17 mg/dL; glucose, 106 mg/dL; amylase, 30 U; bilirubin, 1.5 mg/dL; alkaline phosphatase, 88 U/L; serum glutamic-oxaloacetic transaminase, 71 U/L; serum glutamic-pyruvic transaminase, 154 U/L. A subsequent white blood cell differential count revealed 30% eosinophils.

An abdominal ultrasound displayed multiple calculi in the gallbladder, a common bile duct measuring 9.5 mm, and a 6-mm stone in the right hepatic duct (Fig 1). A DISIDA nuclear scan revealed partial cystic duct obstruction consistent with either chronic or acute cholecystitis. A computerized axial tomographic scan demonstrated a dilated gallbladder with stones surrounded by fluid (Fig 2), dilated intrahepatic ducts, a dilated common bile duct, and a normal pancreas.

At laparotomy, a dilated, chronically inflammed gallbladder containing multiple stones was found and removed. On opening the common bile duct, a large number of 3 × 15-mm leaf-like structures and bile poured out. After introducing the choledoschope into the common bile duct, we were able to see many more parasites, some of which were adherent to the walls of the smaller intrahepatic biliary radicles. Some of these parasites were extracted with forceps and many more were flushed out. A single stone was visualized and extracted from the distal common bile duct. A T-tube was placed in the common duct. After surgery, many more of the parasites drained through the T-tube.

On the second day after surgery, the patient received a one-day treatment regimen with praziquantel, 25 mg/kg/day in four divided doses.[4] A T-tube cholangiogram performed on the seventh day after surgery showed prompt emptying into the duodenum, absence of filling defects, and ductal

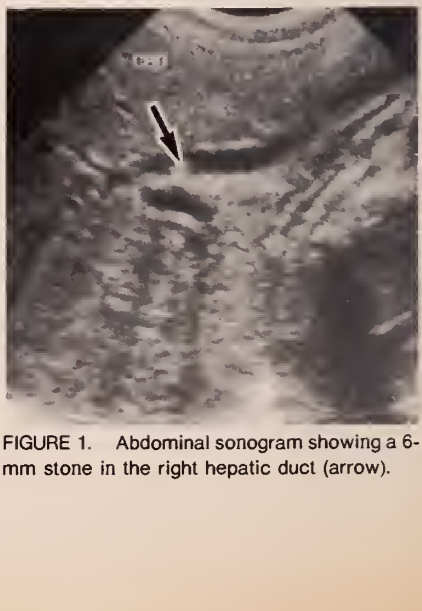

FIGURE 1. Abdominal sonogram showing a 6-mm stone in the right hepatic duct (arrow).

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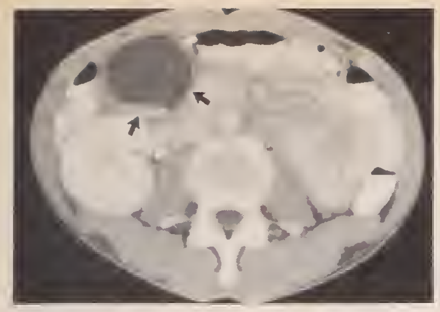

FIGURE 2. Abdominal CAT scan showing dilated gallbladder with multiple stones (arrow) in gallbladder and fluid around gallbladder wall (arrow). Dilated intrahepatic ducts not seen on this film slice.

scalloping with cystic dilation of small intrahepatic ducts (Fig 3). The patient was discharged with instructions to have his stool cultured for ova and parasites.

DISCUSSION

Clonorchis sinensis was first described by McConnel[5] in 1875, and its three-month life cycle was described by Faust[6] in 1927. This fluke is endemic in South Korea, Japan, Formosa, Kwangton Province of South China, and Indochina.[7] In Hong Kong, 65% of the population over one year of age are infected with *Clonorchis sinensis.* The parasite has a life span of 20 to 35 years.

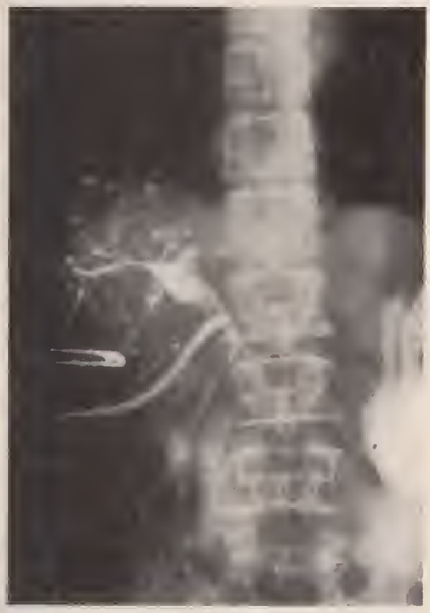

FIGURE 3. T-tube cholangiogram performed on day seven after surgery. Shows ductal scalloping with cystic dilation of small intrahepatic ducts.

The 15 mm × 4 mm adult fluke lives in the terminal biliary radicles. The unhatched eggs pass through the intestine into fresh water, where the eggs hatch into miracidia which penetrate the snail species *Parafossarulus striatulus* (China), *Parafossarulus japonicus,* and *Bulinus fuschiana* (Japan). Within the snails, sporocytes and rediae develop which produce cercaria (the free swimming larval stage of a trematode consisting of body and tail) that escape into the water. The cercaria burrow between the scales of fresh water fish into the muscles, and are ingested by humans who eat the uncooked fish. The cercaria undergo exocystosis in the human stomach and migrate via the ampulla of Vater into the peripheral branches of the bile ducts where they mature.

The worms travel the biliary channels by firmly anchoring their ventral suckers, thereby desquamating epithelium, projecting their bodies full length, attaching their oral suckers and further inching their bodies forward. They then burrow into the exposed ductal stroma adjacent to the peribiliary vascular plexuses, and live off the glucose and protein contained in the tissue fluid exuded from the denuded surfaces of the intrahepatic ducts.[2]

As a result of the biliary de-epithelialization by the parasite, crypt formation, hyperplasia of epithelium, and periportal connective tissue fibrosis develop. The parasites may crowd and intermittently form into biliary plugs causing stasis, bacterial infection (*E coli*), cholangitis, and cholangiohepatitis.[8] A glue-like material found within the lining epithelium and biliary ducts incorporates denuded cells with the clonorchis ova or with dead adult worms to form bile thrombi or stones. *C sinensis* has also been implicated as a cause of cholangiocarcinoma.[1,2]

In an autopsy study of 31 patients from an endemic region, primary choledocholithiasis and *C sinensis* were found in 20.[9] The 8% incidence of obstructive jaundice in a series of 500 patients reported by Hou[9] was not attributed to duct blockage by the organism itself. Generalized biliary dilation was present in only 1.6% of the patients and was a sequela of stone, stricture, or tumor caused by chronic inflammation due to the parasite. Lee and Hirst[10] reported two Chinese patients with clonorchal cholelithiasis and choledocholithiasis obstructing the common bile duct. In the patient presented here, there was dilation of the intrahepatic ducts, cholelithiasis, acute cholecystitis, and choledocholithiasis, as well as clonorchiasis.

Prior to the availability of the trematocidal drug praziquantel, the surgical treatment of cholangiohepatitis due to clonorchiasis consisted of biliary-enteric diversion by either choledocho-jejunostomy or choledocho-duodenostomy. Praziquantel has a specific effect on cell wall permeability, which induces contraction of the parasite by vacuolization and disintegration of the parasite's tegument. Side effects include malaise, headache, dizziness, abdominal discomfort with or without nausea, and increase in body temperature. On rare occasions urticaria has been noted.

Stool examinations for ova and parasites should be carried out on immigrants or travelers from areas of endemic parasitoses, as the diagnosis of parasites is confirmed by finding eggs in the stool. Since this was a surgical emergency there was insufficient time to perform stool examinations, and had clonorchis eggs been found on admission, the management might have been the same.

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Testicular malignancy associated with massive inguinal adenopathy and life threatening hemorrhage

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Testicular tumors occur with an increased propensity in the setting of cryptorchidism.[1,2] Whereas neoplastic spread is usually directed along retroperitoneal lymphatics, correction of an undescended testis by orchiopexy may change the classic pathways of spread, resulting in unusual lymphatic diversion.[3-6] We report on a patient with extensive disease that had spread to both groins. The degree of tumor involvement was considered atypical for any inguinal disease process that we were familiar with or that had previously been reported in the literature.

CASE REPORT

A 27-year-old man underwent a right orchiopexy via an inguinal approach at age 11. He was well until two weeks prior to medical evaluation by his family physician, when he noted the development of scrotal swelling followed by massive bilateral inguinal enlargement. A biopsy taken from the left inguinal region at the referring hospital suggested that the lesion was sarcomatous in nature. Such a pattern of extensive spread to one or both groins had previously been reported in the presence of spermatic cord sarcoma.[7]

Because of uncontrollable hemorrhage from the region of the right groin mass, hospital transfer was initiated. In order to control the active bleeding noted on presentation at North Shore University Hospital, extensive compression dressings were applied to the groin masses.

On examination at this time, the patient was found to be a thin male in minimal distress. A large scrotal mass indistinct from the testes and measuring 7 cm in diameter was present. Even larger 10–15 cm groin masses were present bilaterally. Active arterial bleeding was present from both necrotic groin lesions, but was more marked on the right. The chest radiograph was normal. Hemoglobin was 7.7 g/dL. The pulse rate was 120/min. Transfusions were promptly administered.

A computed tomographic (CT) scan of the abdomen and pelvis failed to show retroperitoneal extension of the disease; rather, all metastases from the scrotal lesion appeared to be to the groins (Fig 1). Pelvic aortography demonstrated neovascularization of the right inguinal mass from the internal iliac and deep femoral arteries. The bleeding was managed by compression dressings and transfusions while a treatment approach was contemplated. Radiation therapy with 600 rads was administered in an attempt to bring the bleeding under control. A review of submitted slides received 48 hours after admission suggested that the initial diagnosis of sarcoma was incorrect and that the tumor was actually an embryonal cell carcinoma. Tumor markers showed the alpha-fetoprotein level to be 7,400 U/mL (normal to 22 U/mL), and the beta-human chorionic gonadotropin (HCG) level less than 4 ng/mL.

The lesion was considered unresectable, and combination chemotherapy for metastatic germ cell tumor was instituted. The drugs employed included cisplatin, vinblastine, and bleomycin. The week following chemotherapy, there was a rapid decrease in any signs of bleeding from the groin masses. Tumor regression was very apparent over the next two weeks. The patient subsequently received three additional courses of chemotherapy, after which time alpha-fetoprotein was undetectable. Because of the presence of a small but persistent tumor mass in the groin and scrotal regions, the patient was readmitted for surgical resection.

A right radical orchiectomy with in-continuity right superficial inguinal node dissection was performed. Redundant right scrotal skin was used to cover the resected inguinal region where skin was taken with the primary lesion. A left inguinal dissection was also performed. The left testicle could now be palpated, and it appeared normal. On microscopic evaluation, the right testis revealed a malignant mixed germ cell tumor with teratoma, endodermal sinus tumor, and embryonal carcinoma. The left inguinal specimen showed metastatic disease in 1 of 12 inguinal nodes (Fig 2). The right inguinal mass showed no evidence of residual malignancy. Immunoperoxidase staining of the tumor was negative for both beta-HCG and alpha-fetoprotein. A fourth cycle of chemotherapy with cisplatin, bleomycin, and VP-16 was given after surgery because of the presence of residual disease. The patient remained free of disease two years after completion of therapy.

DISCUSSION

Metastasis of scrotal germ cell tumors to regional nodes in the retroperi-

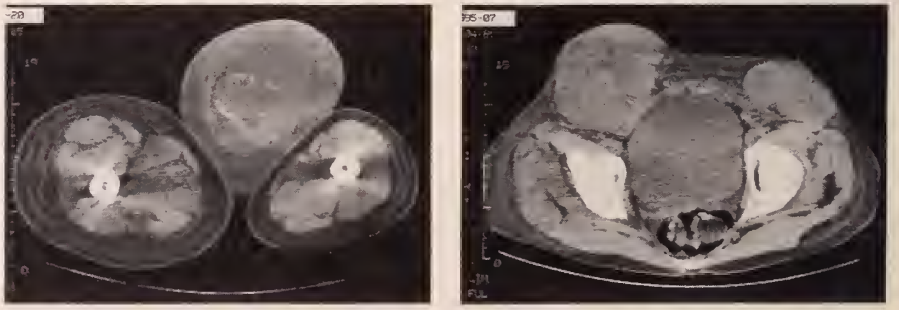

FIGURE 1. Pelvic CT scan showing a large scrotal mass (left) and massive bilateral inguinal masses (right).

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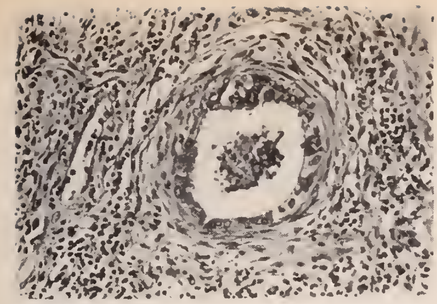

FIGURE 2. Light photomicrograph showing metastatic teratoma to one of 12 left inguinal lymph nodes. The node is fibrotic and depleted of lymphocytes (hematoxylin-eosin stain; original magnification × 150).

toneum is well known. Groin metastases more typically suggest spread from primary intrascrotal sarcoma.[7] However, as in this case, prior orchiopexy changes the natural history of evolving testicular tumors. Prior scrotal surgery alters lymphatic drainage, changing the pathway away from retroperitoneal lymphatics. Inguinal lymph node involvement, when occasionally seen in association with testicular neoplasms, represents a worsening of prognosis for the patient.[3-6] Extensive involvement of groin nodes, as seen in the present case, is unusual both for metastatic nodal involvement as well as for primary nodal disease. Batata et al[8] reported a 7% incidence of inguinal metastasis when testicular tumors developed after correction for cryptorchidism. The findings of groin metastasis in association with an intrascrotal lesion can frequently result in a mistaken clinical diagnosis. The patient reported here demonstrated that the performance of an orchiopexy at age 11 contributed to the development of a non-seminomatous germ cell carcinoma, and that the scrotal surgery resulted in subsequent inguinal metastasis.

The need for orchiopexy in the treatment of an undescended testicle is not questioned. It can preserve fertility, allow for physical examination to detect early neoplastic change, and is of cosmetic benefit. When an undescended testis is detected, correction by age one is recommended, because ultrastructural abnormalities are apparent by that age.[9] If cryptorchidism is detected late, many investigators feel that orchiopexy should be performed no later than the age of ten. Orchiectomy is advised after this age because the risk of the subsequent development of testicular cancer is so great.[1,4,12]

The optimal management of patients with palpable inguinal disease is uncertain. Morse et al[10] believe platinum based chemotherapy is justified as the primary treatment after radical orchiectomy, with groin dissection reserved for residual disease. Klein et al[6] advocate treatment with chemotherapy when groin disease and retroperitoneal disease exist simultaneously. However, when palpable groin nodes exist without evidence of retroperitoneal disease, the choice of surgery or chemotherapy as the initial treatment remains debatable.[6]

In this patient, the initial concern of controlling the bleeding necessitated the use of localized radiation therapy. Post-irradiation combination chemotherapy was employed for three cycles of the drugs. On completion of the third course of therapy, there remained residual bilateral palpable inguinal nodes and a persistent right testicular mass. It was felt that a fourth course of therapy would be most effective following tumor debulking. Surgical resection here was necessary for minimizing tumor burden and to allow for evaluation of the effect of the already administered chemotherapy.

On microscopic evaluation, residual tumor was noted in the specimen, in spite of normal levels of alpha-fetoprotein and lactate dehydrogenase (LDH). However, the marked reduction in these markers following the chemotherapy is indicative of a significant decrease in tumor burden. The chemotherapeutic management of this patient took advantage of the many advances made in the oncologic treatment of germ cell tumors. The initial alpha-fetoprotein and LDH levels placed the patient in a previously defined poor prognostic group.[11,12] Combination chemotherapy with cisplatin, bleomycin, and vinblastine was administered for three cycles, followed after surgery by one course of cisplatin, bleomycin, and VP-16. This type of regimen has been useful in poor prognostic situations.[13] Using a progressive decline in alpha-fetoprotein as a guide, chemotherapy was given until a complete clinical response was achieved, reaching a level of zero by the eighth week of treatment. This rate of decline in serum tumor markers is consistent with previous reports and predicts a favorable outcome.[14] Despite the undetectable alpha-fetoprotein, the surgical specimen did show residual tumor, which indicates that a clinical response determined by markers alone must be further evaluated, especially when bulky lesions persist as in this case.

Immunocytochemical studies in the resected specimen from this patient were negative, suggesting that selection of surviving tumor cells following chemotherapy favored those that were negative for alpha-fetoprotein. Experimental regimens utilizing non-cross-resistant drug combinations have been developed to try to overcome the different sensitivities that may exist in the varying tumor populations. While these salvage regimens have been developed for patients with persistent disease, durable remissions have been difficult to achieve.[15]

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Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Himself, Mrs Plucker, and the rites of spring

TO THE EDITOR: It was with great joy that I read your special article in the February 1989 issue, entitled "Himself, Mrs Plucker, and the rites of spring."[1] I had the opportunity of being one of the "spring training" doctors for the Los Angeles Dodgers in February of 1969. I was a budding surgical resident and my tour with the Dodgers also counted as annual leave. However, being a devout Dodger and baseball fan, I could not resist and eagerly volunteered.

Like yourself, and like the others who followed me, I found Mrs Plucker to be a wonder. She functioned as mother-hen, confidant, stand-in for the "doc," lovely and kindly grandma, and angel of mercy. I became very fond of her and together we spent many happy hours in treating, evaluating, and reminiscing. She was so kind and so helpful and so understanding. I recall her starched uniform always glistened in the Florida sun. You could often hear her rustling by before you could see her. She was very patient with my sophomorism and taught me great lessons in the art of listening and being practical.

Others who followed behind me were also touched by this lovely lady. I mourn her passing, and today 20 years later I still think of her and the marvelous experience of being at Vero Beach. Your article returned me to that time and place with ease and grace. I thank you very much.

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Abuse of the elderly

TO THE EDITOR: The article by Eric Cassell[1] on abuse of the elderly in the March issue of the *Journal* is one of the most poignant discussions of this topic that I have ever seen.

One important abuse of the elderly patient in the nursing home which Cassell does not mention, however, is that of overmedication with psychotropic drugs. This is done in order to make the individuals concerned behave in a docile and compliant manner. Attentive listening and appropriately prescribed psychotherapy are much less frequently dispensed in such settings. One of the sequelae of this depersonalized approach to the elderly is the fact that over half of the medication related deaths in the nation occur in patients over the age of 60 years.

Clearly the misuse of the prescription pad in nursing homes, and in the treatment of the elderly in general, poses a public health problem which must be stopped. It also represents a systematic form of abuse of older citizens which can no longer be tolerated. Many of the individuals I am referring to are not the incompetent elderly persons whom Cassell describes in his article. Hence the physicians involved in their treatment are able to form a relationship with their patients. Only through a return to a personal approach to the care of our older patients can we hope to end the abuse and misuse of them which has prevailed for so long.

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Physician recredentialing

TO THE EDITOR: One cannot help but take exception to the article written by Drs Gellhorn and Cherkasky, concerning the issue of physician recredentialing.[1] One should recall that this proposal, by Governor Mario Cuomo, was suggested as a means of containing the malpractice crisis in New York State. In fact, the authors state that it "was a logical follow-up of the administration's medical malpractice law."[1] In other words, the entire notion of recredentialing seems to have arisen from the assumption that the malpractice problem in New York State was and is caused by the physicians in the state. Does anyone among your readership who does not work for the State of New York really believe this to be the case? And given this false and twisted assumption upon which the recredentialing proposal was initially based, can we, as physicians, meekly go along with it?

Finally, in agreement with the points made by Dr Sherman in his article in the same issue of the *Journal*,[2] I would suggest that the real issue in this case is not the competence of New York State physicians, but that of control. Who will control the practice of medicine in New York State? Considering the forces against us, it is difficult to be optimistic.

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The future of medicine: A resident's perspective

TO THE EDITOR: As a second year resident, I have spent the past five years listening to the doom and gloom prophecy of our profession's establishment. As a spectator, I have witnessed our feeble attempts to recoup social, ethical, and financial losses. The dictums of a generation which led medicine to its present predicament, distressingly still prevail. It is time for medicine's newest generation to assert itself and for the old guard to take heed. Experience is most valuable when imposed upon fresh ideas and innovation. As a representative of medicine's future, I would like to relay some observations and set forth some suggestions, so that they may best serve our mutual interest, the betterment of American medicine.

Medicine, a dynamic entity, behaves in accord with the tenets of Darwinism. To be successful in the practice of medicine, we must assess our environment and adapt accordingly. This environment may be an army field station, inner city emergency room, or an overly restrictive society. Our present method of adaptation includes the taking of evasive action when faced with an ethical or economic dilemma. The dogma of western medical practice must be reevaluated from the perspective of today's American society in anticipation of further movement along present trends. We must readdress both societal expectations of our profession as well as our professional obligations to that society.

The near fatal mistake of America's auto industry offers a valuable lesson. To interact successfully with society, we must listen closely to its desires and opinions. The Occidental community has insidiously swayed from the Judeo-Christian beliefs that prevailed when modern medical ethics were originally fashioned. Three of the most important changes in American society are our misbelief that Americans regard the sanctity of human life above all else, that we cannot place a price upon a human life, and that Americans desire a near infinite level of health care. These are falsehoods, in my opinion.

All clinicians are familiar with both the living will and the do-not-resuscitate order. These are two of the most overt examples of the change regarding sanctity of human life. Many of us are making these decisions daily, yet they remain most uncomfortable. Our profession has not yet fully integrated these ideas into its social norms of conduct. There are numerous examples of this change in America's outlook. Capital punishment has returned to many states, abortion even after an age of fetal viability remains legal, and speed limits have been raised to 65 mph. America is clearly telling us that it does not always regard life above all else. Although we should not necessarily agree with or accept society's newest thinking, we *must* appreciate it as a change in societal values, and understand it.

The medical profession still operates under the false assumption that we have not, and cannot, place a price on human life. In many instances, for example, the workings of the federal government, it is impossible to consider all human lives infinitely valuable. In fact, many government agencies put a clear and exact monetary value on a generic human life. According to Steven Waldman, in his *Newsweek* article, "Putting a Price Tag on Life,"[1] a human life has a present value of anywhere from $208,000 (assuming future mortality discounting) to $8.5 million, depending on the respective government agency. When dictating policy in the cost versus benefit mode, loss of human life must be expressed as a cost, and this cost must be calculated in dollar terms. For example, if an Occupational Safety and Health Administration (OSHA) industrial inspection (OSHA uses a figure of $2–5 million per life) detected a situation that might eventually result in the loss of two lives, but rectification of the situation would require a $20 million investment, the loss of life would be deemed acceptable,[1] and no rectification would be required. Medicine is still struggling over this issue. Presently, we are fortunate to have enough latitude left in the system to accommodate ultra-high-cost care. This elasticity, however, is quickly dwindling.

Money is often representative of one's desires. Thus the greater the desire for something, the higher the monetary value. Therefore, we can look towards economics to uncover societal desires. A basic paradigm of clinical thought is that people desire the highest level of health care available. This is simply not true. Aggregate America values its health care at 10.9% of its total wealth, as manifested by our industry's portion of the gross national product (GNP). We are willing to pay a prize fighter $8 million for ten or 15 rounds; we therefore value his services approximately 80 times that of providing a little girl with a liver transplant. If health care were without cost, surely Americans would demand a near infinite amount. Costs, however, are not zero. Americans are not willing to allocate all of their assets to health care. We cannot provide total care without total societal commitment. We should listen to what society is telling our profession. Society voices the desire for improved health care, but it is not willing to make the sacrifices necessary to achieve a higher level. Certainly one could argue that those in emergent need would make all of the necessary sacrifices. The parched desert traveler might make the necessary sacrifice to preserve his life, and trade his family fortune for a sip of water. But aggregate America would certainly not be expected to trade its total wealth for an ounce of water.

Having addressed the issue of society's expectations of us, let us turn to the question of what we owe society. Our responsibilities as physicians are simple. There are six: we should act justly; we should act responsibly; we should act ethically (although the particulars in this matter are hotly debated, the basic principles are generally well accepted); we should treat the poor benevolently; we should maximize the efficiency of providing care; and we should be good at what we do.

These are our responsibilities as physicians. We should live up to our responsibilities, including offering high quality health care at a fair price.

It is our responsibility to be fair, but it is society's responsibility to decide how much health care it wants. It must decide how much it wants to sacrifice for a given level of care. It is not our responsibility to subsidize a prize fighter's $8 million per fight salary. Nor should we subsidize big business by lowering health care costs, lowering their labor costs and thereby maximizing profits. We should not subsidize the Pentagon by personally absorbing a percentage of the GNP. We are letting society take advantage of our strong professional ethics and general sense of benevolence. At my most cynical moments, I often think that when the budget is at hand, the Washington bureaucrats are saying, "Let's cut physician reimbursement; if we don't pay them they'll do it for free . . . and if they don't volunteer . . . we'll force them." Why let the politicians "have their cake and eat it too" at our personal expense?

Presently, the politically active aspect of the medical establishment is making a feeble attempt to thwart government control and preserve physician reimbursement. The government asserts control over the profession of medicine because the government gives us money. In the end, government control costs us money, as well as increased gastric acid production.

Let me offer a new approach, one diametrically opposed to our present one. Let's lobby for decreased Medicaid reimbursement. Let us treat the poor as we should, and make charity care charity care once again. (The issue of hospital reimbursement should be left to the hospital administrators.) Medicaid reimbursement barely covers a clinician's operating costs. It merely acts as a foot in the door for government regulation. To reduce government regulation, we must start by getting their hands out of our pockets. The benefits of this approach are not merely in the short term. The long term ramifications are staggering. Decreased government involvement will cause the spectre of socialized medicine (the ultimate form of bureaucratic intervention) to lower its ugly head, and a deemphasis on reimbursement might bring our profession back to its previous level of public esteem. Most importantly, the feeling of benevolence would pervade the practice of medicine once again. Improved government relations with regard to reimbursement might even lead to positive malpractice legislation.

I am well aware that getting the entire medical profession to lobby for decreased physician reimbursement is difficult or nearly impossible. What is important is that there are alternate ways of viewing our situation. Too often the medico-political establishment's short term approach lacks long term scope.

In sum, we are the sole gatekeepers of the nation's third largest industry. Instead of controlling the industry we are letting the industry control us.

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ERRATA

Please note the following corrections to the article, "The history of Kashin-Beck disease," published in the June 1989 issue (89:343–351):

Page 346, column 2, line 6: Eugene V. Beck was born in 1865.

Page 349, column 1: The first full sentence should read: "A monograph by a Chinese, Hung Pao-Yuan, emanated from Takamori's department and was published in Japanese in 1945.[41]"

FROM THE LIBRARY

EDITORIAL: THE PERSONALITY OF MEDICINE

During recent years, organized medicine has devoted a great deal of its time to combating the political and social forces that would relegate the physician to the role of a mercenary in the practice of medicine. Such forces would have us forget the need and importance for the preservation of the individual physician-patient relationship in our democratic way of life. Fortunately, medicine has been successful to the present and the freedom of the physician and patient has been preserved.

What will happen in the future? The answer lies within the character and principles not only of the practicing physician today but also the doctors of tomorrow who are being trained in our hospitals, medical and undergraduate schools. In the past few decades, there has been a tendency on the part of medical education to stress scientific knowledge as an end in itself. The product of such a training may be capable of ministering to the ills of the human body but not to the human hearts, minds, and emotions of his patients. Is it any wonder then that the patient's attitude towards his physician changes? In the eyes of the layman, medicine becomes a commodity subject to the whims of a changing society. If we are to preserve the freedom of the physician and the patient, mere opposition to the inroads of socialistic ideologies and systems will eventually fail unless medicine sees to it that the physician is more than just the purveyor of scientific knowledge. As history tells us, societies are more often destroyed from within than from without.

At the recent annual meeting of the American Medical Association, Dr. David B. Allman, president, devoted his entire inaugural address on June 4, 1957, to the "personality of medicine." He stressed that the physician "must be both scientist and humanitarian." Although the physician is primarily a man of science, he must be constantly concerned over human personal values. He must recognize his obligations both as a physician and citizen—the latter requiring his "active interest in community affairs and public issues." Dr. Allman concluded by indicating "the need for a new revolution—a revolution against opportunism, expediency and materialism; a return to the original American ideals of freedom, personal responsibility, individual initiative, and, above all, faith in God." Certainly Dr. Allman is to be commended for his clarification of the problem confronting medicine and wholeheartedly supported in his effort to preserve the best in the personality of medicine.

(*NY State J Med* 1957; 57:2338)

Body and soul: Doctors tell all

When Doctors Get Sick. Edited by Harvey Mandell, MD, and Howard Spiro, MD. 464 pp. New York, Plenum Publishing Corp, 1987. $25.00 (hardcover)

M.D.: Doctors Talk About Themselves. By John Pekkanen. 294 pp. New York, Dell Publishing, 1988. $18.95 (hardcover)

The two volumes discussed in this essay review will rest well on the bookshelves of those physicians who care enough about their profession to experience concern for the bodies and souls of their flagging colleagues. They may also be of more than passing interest to those in the profession who spend more time than they would like to admit wondering why they ever went into medicine, whether or not they want to stay in medicine, and what price was paid, physically and spiritually, for their foray into this most eventful of professions. Both books, if read as thoughtfully as the subject matter would demand, are painful at times, but have the potential to be fiercely engaging, if not seductively compelling. Together they offer a mirror, cracked though it might be, to the personal glories and devastation wrought by eating of the fruit of the tree of medical knowledge.

The idea of a collection of essays by physicians who have become sick and have done battle with the forces of hospital medicine is not a new one. *When Doctors Are Patients*,[1] published by Pinner and Miller in 1948, and long accepted as a classic of medicine, is unfortunately obtainable now only through the assistance of a book-find service. In that original volume, two physicians at Montefiore Hospital asked their colleagues to write about their experiences of being sick. They were rewarded with a series of thoughtful essays describing the emotions, feelings, behaviors, and thought processes of a group of high powered healers struck down by the great visceral common denominators. Nausea, pain, diarrhea, and vertigo were but a few of the progenitors of human misery over which they could be masters no longer. How they dealt with the ego blows constituted the fascination of this volume.

The physicians of 40 years ago would prove past masters at self-deception, denial, hubris, shoddy manipulation of their caretakers, and overt subversion of their medical care. They chose friends as their physicians instead of those most qualified. They kept secrets of vital import to their care from spouses, surgeons, and consultants alike. Often unable to accept human limitations, they cheerfully went about their rounds trailing their own IVs behind them. All in all, a sorry performance, tempered only by their willingness to admit the idiocy of it all, and to enlist the reader's sympathy by covertly implying that were the tables to be turned, so would be the behaviors of the reader.

The redeeming feature of this volume was the genuine spirit of introspection, philosophical musing, and search for understanding which ran like a common thread throughout. I found much of this missing in the modern update of that volume, a distressing reflection not only of how medicine has changed in the interim, but how much its practitioners have changed as well. This may, however, be the bias of the reviewer who, like an old cinema buff, despises all remakes of the old classics. Libris caveat emptor.

When Doctors Get Sick is a bigger and fatter volume, with no fewer than 50 contributions. It is entirely too swollen, and at times the editing is poor, with "Britishisms" (both of spelling and slang) retained unmodified and untranslated. Drs Mandell and Spiro tapped personal friends and previously published contributors in this country and abroad, but might have obtained more richness and variety had less informal methods been used to develop the collection.

As with all essay collections, content and style are variable, with some contributions that simply cannot stand on their own merit. Yet even the poor essays contribute in their own way, reflections of how doctors may fail their patients simply by failing themselves. One is reasonably certain that those who woodenly report their operative notes and laboratory values, with limited affect and no sense of the significance of what has happened to them, will tend to relate to patients in the same way, as unreactive objects to be manipulated until cured.

What seemed most impressive was the preoccupation of today's doctors with the technical details of their illnesses. Many of the essays read like talking charts, progress notes come to life through the eyes of a new narrator, the patient. The editorial content, is, of course, what we crave most. Today's doctors report their distress over the staggering array of treatment options, their struggle to get their physicians to agree on an appropriate course of therapy, their difficulties in accepting opinions with which they disagreed. They recount their struggles with ancillary hospital personnel, bureaucratic regulations, and their frustrated desire for privacy. They all appear to have taken copious mental notes on what happened to them. In this regard, they bear little resemblance to the previous generation, who gave far more individualistic, impressionistic, and less literal accountings of their experiences.

This may, however, be artifactual. The essays appear to follow a preordained rubric undoubtedly given them by the editors. One can almost "hear" the directions given, so stereotypically are they followed. "Please tell us how you got sick and what happened to you. Did religion help you? As a result of your experiences do you find yourself relating to patients differently? How has your philosophy of life changed as a result of your illness?" Unfortunately, many of the questions posed seemed to be beyond the ken of some contributors, whose essays fall down like old soufflés when an attempt is made to answer in the contemplative mode.

A factor contributing to the general deflation after each story is told may be that today's doctors seem less articulate in general. If the feelings are there, they appear less accessible to the writer and therefore unreportable to the reader. When expressed, they are often disappointing. (Several physicians, for example, reported that after their ordeals they lost tolerance for the complaints of patients who suffered less than they. No concept of the infinite variety of individual response to illness had been assimilated.)

Despite these very real limitations, many of the essays are truly gripping. The sections written by sufferers from diseases of the gastrointestinal system were of teaching quality. In particular, four essays written by men and women living since early youth with ulcerative colitis were especially striking. The essay by Dr Judith Brice should be required reading for all students of liaison psychiatry as she graphically portrays what it means to be an unpopular, feared, and demanding patient whose pain is seemingly beyond the comprehension of ordinary mortals, and who is roasted by those to whom she should feel closest by virtue of a shared profession.

Amusingly, what remained most unchanged was the great need of physicians to maintain control, participate nosily in their care, and admonish hospital personnel whenever mistakes occurred. This was not done without guilt and careful consideration of the repercussions. Also unchanged was the ubiquitous denial, which led today's physician reporters stoically to endure the most horrific signs of illness for weeks, months, before ambling over to find out if it was all their imagination. One doctor hangs up his own x-ray films, surreptitiously shot for him by a technician friend, only to confront a massive tumor. Another bleeds copiously per rectum for several months, hurriedly changing his whites in the men's room, and presumably not even dallying to wash his hands. In view of the still contemporary need to have a diagnosis, and the avidity with which even the most horrendous verdicts were accepted by worried physicians, it is too bad that the editors did not include an essay or two by sick physicians for whom no definitive diagnosis has ever been made.

Many of the essays in this volume are deeply moving, forcing the reader to identify head on with the writer, and confront the question that nags at us all: How will I react when "it," whatever "it" is, hits me? Jack Lewis' recounting of his terminal and unrelenting struggle with acute myelogenous leukemia rends the heart. Another chilling narrative is Richard E. Thompson's ordeal with a benign giant cell tumor of the sacrum. William B. Ober manages to introduce one of the few attempts at levity, as he describes the indignities of hemorrhoidectomy. The cool intellectualism of the anonymous "R. F. Spooner" recounting what he has learned from Crohn's disease is impressive. Also memorable is John A. McCool's story of the headaches of a brain tumor which he did not bother to report lest it interfere with his medical school classes and cause him to lose face with study partners. A psychotic tone poem of great beauty is the contribution of Michael Rose, written during a hospitalization for an acute manic state.

For these essays, and others, we are indebted to editors Mandell and Spiro, who gleefully report that they are thrilled to be in print with this book. It is obviously a collaborative endeavor which they put together with great affection. It should last us for another few years.

In the final analysis, *When Doctors Get Sick* is less impressive for what it actually says than for what it makes us think about. If each of us had been asked to contribute to such a volume, what might we have had to say? How truly wise would we appear in the eyes of our colleagues and our patients? And what hideous price did we pay in studying medicine, that holds us hostage to fears unknown to others, and intensifies our anguish at the moment our own vital organs and imperfect remedies betray us?

Mr Pekkanen is a senior and experienced writer who was twice winner of the prestigious National Magazine Awards. His book, *M.D.: Doctors Talk About Themselves*, was designed to sell a vast number of copies. It undoubtedly will, appealing as it does to a wide variety of readers, including doctor bashers, hospital groupies, devotees of lives of the rich and famous, and assorted psychological voyeurs. None of this prevents it from being a profitable book for doctors to read. I found it lying on the desk of the editor of this *Journal*. Picking it up with idle curiosity, I flipped pages. Suckered in, I sat down to have a better look. Forty minutes later the secretary interrupted me from the depths to say, "Dr Bluestone, are you still here?" I had lost all track of time or space. It would be ungenerous and pretentious of me to pan Mr Pekkanen's book now.

The picture on the cover is of a young resident sagging against a wall with his eyes closed, a surgical mask drooped about his thyroid, and a look of weary and classic suffering on his face. To my tutored eye, this suggests either that he is contemplating when he will finally get off duty, or how long it will be before he can offer his bowels some relief. Our hearts go out to him, and to his anonymous colleagues who contributed to this book.

To create this work, Mr Pekkanen must have followed a huge assortment of physicians into their respective locker rooms, plied them with just enough ethanol to loosen their tongues, and then recorded their candid vignettes of memory and impression on reams of tape. What results is a series of one- to three-page first person narratives, each standing alone, and separated by asterisks which remind the reader that it is time to take a break before the next mini-story comes to life. Thank goodness for the asterisks! They proved necessary on more than one occasion to keep me from hastily passing on. In fact, the whole book elicited a constant struggle over whether I should just keep reading because the narrative was so impelling, or I should pause and reflect while the full impact was still rolling around inside my brain.

Mr Pekkanen's doctors tell all. What they say is upsetting, funny, endearing, inspiring, revolting, but in every case solid with the heavy ring of truth. They talk about their cases, their training, their finances, their practices, their colleagues. Oh, do they tell on their colleagues! These mini-memoirs are so intimate one has the uncomfortable feeling that one has no business hearing this, that at any moment the screen will be dropped and we will be left as eavesdroppers with our cheeks turning crimson. And right on, we say, as they gripe, reminisce, rail at the bosses, taunt the red tape compliance ladies, sneer insults at colleagues for whom they have no earthly use. They bleed for us, drops of bright red blood, as they survey the wreckage of their once proud profession, and tell us how they keep trying to talk their kids out of it any more. They confess their guilt and shame over errors, laying their overactive superegos right at our feet for us to step on. They tell us how they got through their residencies, the way Solzhenitsyn got himself out of the Gulag.

One tells us of a hideous prank he plays on a patient to get even with a colleague for whom he has no love. A female surgeon complains that the only referrals she gets from male colleagues are breasts to be removed. A psychiatrist excoriates his colleagues for leaving the sickest patients to the least qualified and remaining in lucrative private practices for the worried well. Another describes how he bites the inside of his cheek to stay awake as a whining patient drones on and on, session after session. An anesthesiologist describes his love affair with fentanyl. An inner city emergency room doctor tells how he lost his liberalism, after what he's seen, and what rapport he has with the cops who share his loathing for their human cargo. A neonatologist wonders what he's doing, particularly when the parents have long since abandoned their infant.

On and on. The book is gripping, I am telling you. I brought it home and started reading aloud to my husband as he was making dinner. I entertained him from the preparation of his meticulously sliced salad through to dessert, hoping I wouldn't get tomato sauce on it. We couldn't put it down.

One thing that puzzles me is how Mr Pekkanen managed to get such a wide variety of doctors to come out sounding as if they were all the same person. It must have taken a good bit of editing for him to distill the essence of each person's contribution into a distinctive and swift-moving narrative style. I know doctors. I know how they talk and how they write, and there's no way these remarks could have been the slightest bit verbatim. But granting a certain poetic license, we are content to be swept up by the narratives and merely ponder the finished product.

If it is true that the sign of a good writer is the ability to articulate thoughts that the reader has always had but has been unable to actually put into focus for himself, the doctors writing through this author are very good indeed. They echo the despair felt by physicians practicing in America today, despair for the future of their profession, for the resolution of dilemmas that seem larger than life, for the seemingly insurmountable obstacles to a meaningful relationship with the patient. They also echo the wonderful feelings still possible through the practice of medicine, although these voices are growing weaker and are not as plentiful. They manage to do it in a way which makes us smile, recognize, and say, Aha! I am not alone. For this reason, I believe it has a truly cathartic value for physicians who tend to remain isolated in their pain. All in all, we have here an opportunity for a glimpse into the soul of medicine today. Those who prefer to wait for the paperback edition, which will undoubtedly follow as the night the day, should be advised that this is quintessential briefcase/pocketbook reading. Use it for awaiting trains, interviews, and doctor's appointments. Assuming, of course, that you set your wrist alarm first.

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Consulting Editor

Reprinted from the March 1989 issue of Epidemiology Notes (*Vol. 4, No. 3*), *published by the Division of Epidemiology, New York State Department of Health, Albany, NY.*

Alcohol and public health: Implications for New York State

This issue of *Epidemiology Notes* initiates a three-part series on the public health significance of alcohol abuse in New York State. The series will describe beverage alcohol consumption patterns and the dimensions of alcohol-related morbidity and mortality in the state. The impact that alcohol abuse has on health care services and factors which contribute to the problem of alcohol abuse will be discussed, as will opportunities for health care provider and public health sector involvement in alcohol abuse prevention and control.

Alcohol Abuse—A Definition

Alcohol abuse can be defined as consumption which places the drinker at increased risk for illness, injury or death. Abuse is not limited to the chronic dependence on alcohol. Abuse can also describe other consumption patterns, such as drinking to intoxication. Abuse pertains also to situations, such as excessive consumption prior to operating a motor vehicle. In addition, alcohol abuse can relate to consumption at any level, in any setting by those at special risk, such as pregnant women. Millions of New York State residents, most of whom are not alcoholics, abuse alcohol. This pervasive health risk behavior presents a unique challenge to preventive health.

Alcohol Consumption

The consumption of beverage alcohol is a common phenomenon, more than twice as common as cigarette smoking, and is woven into many social interactions of adults and adolescents (Fig 1).

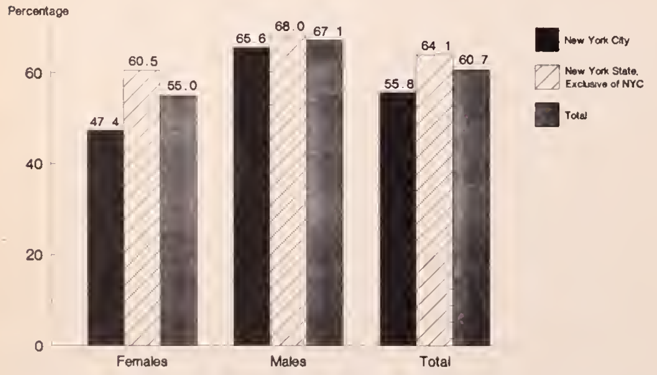

FIGURE 1. Percentage of respondents reporting consumption of at least one alcoholic beverage in the previous 30 days (Source: NYS Alcohol Tracking Survey, April 1987).

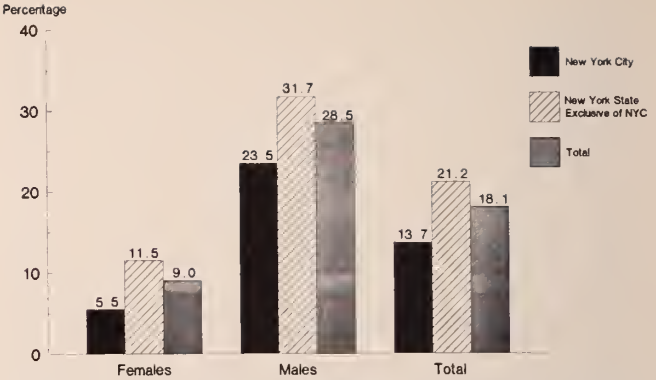

FIGURE 2. Percentage of respondents reporting consumption of five or more drinks at one time in the previous 30 days (Source: NYS Alcohol Tracking Survey, April 1987).

New York State residents consume approximately 450,000,000 gallons of beverage alcohol per year. During 1987, individuals 14 years of age and older consumed an average of 25.4 gallons of beer, 3.8 gallons of wine and 2.3 gallons of distilled spirits. Per capita consumption is at approximately the same level as it was 25 years ago, although it has decreased slightly in the 1980s. The per capita consumption of alcohol beverages is a useful measure of the dimensions of community risk, since it is associated with such behavioral consequences as driving while intoxicated as well as to poor health outcomes (eg, cirrhosis).

Drinking five or more alcoholic drinks over a short time period, "binge drinking," generally produces a blood alcohol content above the legal intoxication level (0.1%) and elevates the risk of unintentional or intentional injury. Frequent intoxication also serves to increase the risk for long-term adverse health consequences. Figure 2 indicates that almost one out of every five state residents surveyed in 1987 acknowledged binge drinking within the past month. Drinking to intoxication is much more common among males. Twenty-one percent of adult males and 7% of adult females (a projected total of almost 1.7 million people) reported that they had driven a motor vehicle in

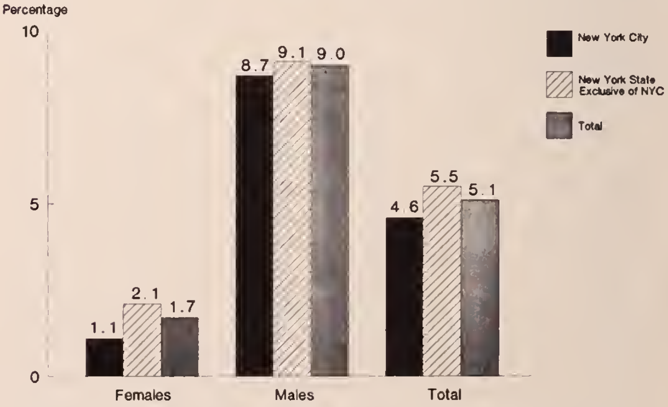

FIGURE 3. Percentage of respondents reporting average daily consumption of two or more alcoholic beverages in the previous 30 days (Source: NYS Alcohol Tracking Survey, April 1987).

the past year while feeling the effects of alcohol.

A widely used threshold definition of heavy drinking is the consumption of an average of two or more alcoholic beverages each day, including nondrinking days. Approximately one in 20 residents reported engaging in this behavior when surveyed in 1987 (Fig 3). Again, males predominate in this health risk behavior.

Health Outcomes

Mortality. New York State death certificate data for 1985 have been examined to estimate the proportion of deaths among residents attributable to alcohol consumption. A methodology to calculate alcohol involvement reported by the Minnesota Department of Health was employed in the analysis.[1] Table I summarizes the results of the analysis. More than 6,500 New York State residents died in 1985 of alcohol-related causes. Reflecting consumption patterns, approximately 70% of alcohol-associated deaths were among men.

Years of potential life lost (with age 65 as the standard) provide a measure of the prematurity of death. More than 100,000 years were sacrificed to alcohol abuse in 1985, more than 33% greater than that attributable to smoking.

Morbidity. Mortality and years of potential life lost represent only the tip of the iceberg of the total health impact of alcohol abuse. Other severe health outcomes include illnesses and injuries requiring hospitalization. An analysis of hospital discharge data for 1986 was conducted by applying the same attributable risk as in the mortality analysis. This yielded an estimate of approximately 153,000 alcohol-related discharges entailing more than 1.3 million hospital patient days at an annual cost in excess of $500 million (Tables II, III).

TABLE I. Estimated Alcohol-Related Mortality, NYS, 1985

Diagnostic Category	Total Deaths	Percent Alcohol Related	Number of Alcohol-Related Deaths
Injuries			
Motor vehicle crashes	2,041	50	1,021
Falls	883	41	362
Suicide	1,544	26	401
Injuries caused by fire	305	42	128
Homicide	1,758	42	738
Drownings	196	30	59
Watercraft injuries	48	35	17
Total Injuries	6,775	40	2,726
Digestive Diseases			
Alcoholic cirrhosis	1,133	100	1,133
Other cirrhosis	1,803	50	902
Acute pancreatitis	156	41	64
Chronic pancreatitis	17	67	11
Total Digestive Diseases	3,109	68	2,110
Neoplasms			
Cancer of the oral cavity	716	47	337
Cancer of the esophagus	786	75	590
Cancer of the larynx	368	49	180
Cancer of the liver	647	15	97
Total Neoplasms	2,517	48	1,204
Mental Disorders			
Alcoholic psychosis	15	100	15
Alcoholism (dependent)	372	100	372
Alcohol abuse	33	100	33
Total Mental Disorders	420	100	420
Other			
Alcoholic cardiomyopathy	66	100	66
Total Other	66	100	66
Grand Total	12,887	50% (approx)	6,526

TABLE II. Hospital Discharges Reported to SPARCS in 1986

Diagnosis Category	Total Number	Percent Alcohol Related	Number Alcohol Related	Average Length of Stay (Days)
Alcohol dependence	70,154	100	70,154	8.4
Alcohol abuse	10,462	100	10,462	9.5
Alcohol psychosis	4,404	100	4,404	9.4
Alcohol cirrhosis	4,891	100	4,891	12.8
Alcohol cardiomyopathy	1,303	100	1,303	10.8
Alcohol poisoning	1,153	100	1,153	4.1
Fetal alcohol syndrome	106	100	106	22.8
Chronic liver disease	11,686	80	9,349	11.0
Acute pancreatitis	8,550	41	3,506	11.8
Chronic pancreatitis	2,564	67	1,718	10.6
Cancer of the esophagus	2,278	75	6,709	15.7
Cancer of the lip	125	47	59	6.5
Cancer of the larynx	1,891	49	927	13.9
Cancer of the liver	1,291	15	194	12.9
Pulmonary TB	4,801	25	1,200	17.6
Falls	42,859*	40	17,144*	8.0*
Burns	5,587	42	2,347	12.4
Drownings	196	30	59	5.5
Motor vehicle	21,736*	15	3,260*	8.0*
Assaults	7,322*	45	3,295*	8.0*
Self inflicted	357*	32	114*	8.0*
All other injuries	100,524*	11	11,058*	8.0*
Totals	304,240	approx 50%	153,412	

* Estimated.

TABLE III. Total Length of Stay and Total Hospital Cost of Alcohol Related Hospital Discharges Reported to SPARCS, 1986

Diagnosis Category	Length of Stay in Days	Average Cost Per Day	Total Cost
Alcohol dependence	589,536	$343	$202,436,200
Alcohol abuse	98,907	390	38,547,484
Alcohol psychosis	41,661	343	14,289,723
Alcohol cirrhosis	62,521	425	26,561,282
Alcohol cardiomyopathy	14,121	518	7,308,267
Alcohol poisoning	4,749	484	2,297,533
Fetal alcohol syndrome (stays in excess of three days only)	2,204	281	618,938
Chronic liver disease	103,670	393	40,730,380
Acute pancreatitis	41,493	401	16,656,771
Chronic pancreatitis	18,262	401	7,323,062
Cancer of the esophagus	26,816	393	10,538,688
Cancer of the lip	400	393	157,200
Cancer of the larynx	12,936	393	5,083,848
Cancer of the liver	2,507	393	985,251
Pulmonary TB	21,134	393	8,305,662
Falls	137,666*	427	58,783,382*
Burns	18,846	427	8,047,242
Drownings	474	427	202,398
Motor vehicle	26,178*	427	11,178,006*
Assaults	26,459*	427	11,297,993*
Self inflicted	915*	427	390,705*
All other injuries	88,796*	427	37,915,892*
Totals	1,340,251	$380	$509,655,907

* Estimated.

Approximately 6.1% of all hospital discharges in New York State in 1986 were alcohol-associated, accounting for 7.8% of the total cost of providing hospital care statewide. In addition, an estimated 762,000 alcohol-associated emergency room visits occurred in 1986, at a cost of more than $86 million. Long term treatment and rehabilitative care further add to health care costs of alcohol abuse.

Reference

1. Parker DL, et al: The social and economic costs of alcohol abuse in Minnesota, 1983. *AJPH* 1987; 77: 982-6.

Two-dose measles immunization recommendations

Measles continues to be a significant public health problem in New York State despite ten years of efforts to eliminate indigenous measles. From 1985 through 1988, an average of 455 cases of measles occurred each year in the state. A substantial proportion of these cases have occurred in previously vaccinated people. The failure of the current one-dose measles vaccination policy to achieve its goal is an experience mirrored in other states. The failure to administer the first dose of measles vaccine at the appropriate age has been responsible for part of the problem in some areas.

Considerable evidence in the medical literature indicates that a two-dose measles vaccination schedule should be a significant improvement over the current single dose policy. The second dose of measles vaccine should protect the 2–10% of children who failed to respond to the first dose. Individuals receiving two doses of measles vaccine have been shown to have a significantly reduced risk of contracting measles.

Considerations of herd immunity also suggest that a two-dose schedule should increase the level of immunity in the population to the point where secondary spread can be limited. At least ten European countries have adopted a two-dose schedule which has resulted in virtual elimination of indigenous measles cases in those countries. The Immunization Practices Advisory Committee (ACIP) of the US Public Health Service and the Red Book Committee of the American Academy of Pediatrics will be meeting soon to consider whether a two-dose measles policy will be recommended nationally.

Since measles is a preventable disease with the potential for serious morbidity and possible mortality, major efforts have been expended in the control of recent outbreaks in schools and on college campuses. These efforts have been costly not only in terms of the disruption of school activities but also in terms of the costs of vaccination campaigns to halt the outbreaks.

After review of these issues and the New York State experience with measles, the Department of Health, in consultation with state and national experts including representatives of the US Centers for Disease Control and District II (New York State) of the American Academy of Pediatrics, has endorsed a two-dose measles immunization policy. The policy will require a second dose of measles vaccine be given to children entering kindergarten beginning in September 1990. The specific recommendations are as follows:

- Individuals born since January 1, 1957, who do not have a physician-documented history of measles or serologic confirmation of measles immunity should receive two doses of measles vaccine for maximum protection against measles. However, it is not necessary to re-immunize all these people now unless there is the likelihood of exposure to measles (eg, measles cases occurring in the same school, anticipated travel to a measles-affected area, employment in a medical field, etc).
- Recommended schedule for New York State: The first dose of measles vaccine should be given at 15 months of age and the second dose should be given at four to six years of age simultaneously with the DTP#5-OPV#4 immunizations (at separate injection sites). In New York City, where measles has been endemic in very young children, the New York City Health Department recommends the first dose of measles vaccine be given at 12 months of age. The minimum interval between the first and second doses should be three months. These new recommendations should be instituted now for children who will enter kindergarten in the fall of 1990. The Department of Health will provide vaccine for the second dose for children seen in public clinics.

- Requirement for school entry: All children entering kindergarten will be required to show proof of receipt of two doses of measles vaccine or other acceptable proof of measles immunity for school entry beginning September 1, 1990. Thereafter, all children born on or after January 1, 1985 who enter grades 1-12 will also be required to show proof of receipt of two doses of measles vaccine in order to cover children coming from out-of-state.
- The use of MMR (measles-mumps-rubella) vaccine is recommended for all vaccine doses to increase protection against all three vaccine preventable diseases.
- Emphasis should be placed on the importance of children's receiving their first dose of measles and other vaccinations on time at the recommended ages.

This article was adapted from a letter recently sent by the Commissioner of Health and the District II (New York State) Chairman of the American Academy of Pediatrics to pediatricians, family practitioners, local health units and regional health directors in New York State. Further information may be obtained by contacting your local health department or the Department of Health Immunization Program at (518) 473-4437.

Bicycle Helmet Brochure

The New York State Department of Health has a brochure available entitled *Head Out With A Helmet*. This brochure contains information concerning bicycle safety and what to look for when buying a helmet. Limited copies of the brochure may be obtained by contacting:

Publications
New York State Department of
Health
Empire State Plaza
Albany, NY 12237

AIDS: A GUIDE FOR THE PRIMARY PHYSICIAN

Edited by King K. Holmes, MD, and Arno G. Motulsky, MD. 66 pp, illustrated. Seattle, University of Washington Press, 1988. $15.00 (paperback)

Disease expression of human immunodeficiency virus (HIV) disease has often been viewed as that of an iceberg. Symptomatic disease, ie, AIDS or the AIDS-related complex (ARC), represents only the visible portion of the iceberg—the area above the water line. Asymptomatic disease represents that vast majority of those infected and is reflected by the portion of the iceberg below the water line. Two processes will contribute to increasing the amount of disease above the water line—progression of disease and increased antibody testing for HIV. Consequently, with greater numbers of patients identified, the care for these individuals will fall on primary care practitioners, chiefly internists. This book attempts to present to the practitioner recent information on the diagnosis, treatment, and psychosocial aspects of managing AIDS patients.

The articles that appear in this volume originally appeared as an edition of *University of Washington Medicine*, published in Winter 1987. Most of the authors are from that institution, and the text reflects this. This is not a deficit, however, since many of the authors have been responsible for substantial contributions to the literature on the epidemiology, virology, and chemotherapy of HIV disease. The book is divided into 13 chapters, and appears at first glance to cover all the important issues that accompany HIV disease. The first two chapters cover the epidemiology of AIDS in the United States and the Pacific Northwest, and then go on to detail the public health response. This is followed by three chapters on HIV virology, laboratory diagnosis of HIV infection, and management of the clinical manifestations of HIV-induced immunosuppression. There are then several chapters on the psychosocial and ethical aspects of the disease, an overview of the infection control issues in AIDS, and finally a global perspective on the disease.

Comprehensive as this outline is, however, it is inadequate preparation for the clinician who will be attempting to manage patients with symptomatic disease. The chapter on the clinical manifestations and approach to management of opportunistic infections demonstrates this inadequacy most clearly. The entire issue of *Pneumocystis carinii* prophylaxis is covered in one paragraph with no discussion of the dosages of the various drugs that have been employed. Inadequate discussion is given to the issue of the medical workup of an individual identified as seropositive. No guidance is given regarding the workup of the seropositive patient with an abnormal chest radiograph. In contrast, however, discussions of the ethical and psychosocial issues are well presented and highlighted by the use of actual cases.

In summary, there is a great need for a comprehensive text describing the clinical management of patients with HIV disease, as well as the public health, social, and ethical issues. While this book may serve as a good introduction for those clinicians who are beginning to confront HIV disease, it is clearly inadequate as a reference for questions regarding clinical management. There is as yet no useful recent text that meets this challenge. One hopes to see this soon.

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UNDERSTANDING THE ALCOHOLIC'S MIND. THE NATURE OF CRAVING AND HOW TO CONTROL IT

By Arnold M. Ludwig, MD. 188 pp. New York, Oxford University Press, 1988. $16.95 (hardcover)

The theme of Dr Ludwig's book is clear enough in the title, which suggests that controlling drinking problems begins with understanding how alcoholics think about alcohol and how they crave it. In the preface, the author suggests that alcoholics recover by "adopting certain characteristic ways of thinking and behaving" (p8). The purpose of the book is to elaborate on these ways by describing what takes place during the process of recovery. It is suggested that both clinicians and those needing the book for their own personal benefit or "cure" will find the book helpful.

The author, a professor of psychiatry, has obviously talked to many people with serious drinking problems, and many who have utilized Alcoholics Anonymous (AA) as the road to recovery. He also has published many articles in the field of alcoholism. His book is useful for physicians who wish to understand alcoholism and the recovery process, and to gain anecdotal evidence of this process, which is presented by using the words of recovering alcoholics. Because many conflicting theories of alcoholism have in common an agreement about the cognitive processes of the drinker, the author adopts a cognitive-behavioral approach first popularized by D. Meichenbaum in 1979 (*Cognitive-Behavior Modification: An Integrative Approach*, New York, Plenum, 1977). He then describes how drinkers set themselves up for relapse, what craving is all about, and how they attempt to control this craving.

The strength of this book, its authentic recapitulation of the mental processes drinkers utilize as they "hit bottom" and begin their recovery with the help of AA, is also its weakness. First, the objective and scientific is interspersed unevenly with the subjective, anecdotal presentations. The serious practitioner will have trouble applying such statements as this: "The motivation to quit drinking does not always occur at the moment individuals opt for sobriety, for it is possible that the will to recover may grow slowly in intensity until, with the passage of time, it eventually hardens into a firm implacable resolve" (p128). Or (the final sentence of the book, p134), "With this orientation to life, intoxication is unnecessary." These statements reflect beliefs held firmly by recovering alcoholics, and in no way is their importance for the drinker diminished. On the other hand, they represent a highly subjective approach to recovery which does not work for everyone. The author himself states that the spiritual AA program, which affects many aspects of life other than the ingestion of alcohol, is not to be embraced by everyone. Second, there have been important additions to the understanding of the drinker in the last ten to 15 years, not only in the biological area, but in the understanding of the drinker as a person who is indeed affected by a family and other systems. The author notes these approaches briefly but dismisses them rather abruptly. Such important authors in the field as Duncan Stanton, Peter Steinglass, David Berenson, or Edward Kaufman are not cited at all. Nevertheless, a cognitive-behavioral approach to drinking in an individual context, supported by a clinically rich description, can be of great value to those many physicians faced with individual patients who are drinking too much.

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POST-TRAUMATIC THERAPY AND VICTIMS OF VIOLENCE

Edited by Frank M. Ochberg, MD. 384 pp. New York, Brunner/Mazel Publishers, 1988. $40.00 (hardcover)

For the past five years I have been treating a victim of terrorist violence. This individual has taught me in a poignant and painful way, the meaning of terrorism, the expectation of violent death, and the terrible and slow process of rehabilitation and return to normal life. I wish that I had had an opportunity to read this book five years ago, before I began my therapeutic work with this patient.

The editor, Frank M. Ochberg, MD, has been a friend of mine for many years. I have watched him develop in his research with the victims of violence. This book brings together insights by experts from different disciplines in understanding and treating such victims. Moving from the discussion of the post-traumatic stress disorder to the problems of translating these phenomena into psychological terms, Dr Ochberg discusses the definitions of bereavement, victimization, autonomic arousal, death imagery, and negative intimacy (a term that he has coined to include situations such as kidnap, rape, and assault). Building upon these five paradigms, Dr Ochberg provides models of post-traumatic stress disorder, animal studies, and treatment. I am particularly impressed with the section, "Principles of Post-Traumatic Therapy."

The chapter by Dr Van der Kolk on the psychopharmacologic treatment of post-traumatic stress is one of the finest I have encountered, and it has given me a new perspective on the sometimes appropriate use of psychoactive medications in the acute phase. Even though popular teaching over the last 20 years has held that there is little or no role for psychoactive drugs in the acute phase, Dr Van der Kolk makes a very cogent argument for flexibility in applying this axiom. I am also impressed by the chapter on the Vietnam veteran with the emphasis on post-traumatic family therapy.

There is a great deal more that could be said about this book. Hardly any part of it is without merit, and it should be read *in toto* by anyone who has the desire or opportunity to reach out to help some of these victims of our modern world.

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DISEASES OF THE COLON, RECTUM, AND ANAL CANAL

Edited by Joseph B. Kirsner, MD, and Roy G. Shorter, MD. 724 pp, illustrated. Baltimore, Williams & Wilkins, 1988. $115.00 (hardcover)

This book, edited by Drs Kirsner and Shorter, is an excellent and thorough one concerned with diseases of the rectum and colon. While the different chapters are written by different experts, there is a uniformly high quality of writing throughout and there clearly has been an effort to avoid excessive repetition. The illustrations are excellent, including a number of color endoscopic photographs. Chapters on inflammatory bowel disease, immunology, bacterial flora, and endoscopy are quite good and the topic of colon carcinoma is thoroughly examined, including chapters on epidemiology, polyp management, and dysplasia.

There are a few minor criticisms which would be applicable to any book of this scope. Ulcerative colitis and Crohn disease are lumped together under the rubric, inflammatory bowel disease (IBD). While they are similar in many areas, the risk of colon cancer and the issue of colon cancer surveillance are different in these conditions. Also, the section on the surgical approach to IBD would have benefited from more discussion, especially from the point of view of the gastroenterologist. The chapter on colonoscopy is, in general, excellent, but the indications for this procedure are probably exaggerated since there still is a place for barium enema in such problems as abdominal pain and constipation. In addition, constipation and pseudo-obstruction syndromes deserve more thorough coverage.

The colon is an organ which is far more complex in terms of its bacterial ecology and metabolic function than was previously appreciated. It is also the focus of a number of diseases that have recently been emphasized such as *Campylobacter* colitis and ischemic colitis. Colon cancer is a major public health problem. An extensive review of all of these topics is welcome. This book is an excellent source of information for gastroenterologists and rectal and colon surgeons and is highly recommended.

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COLOUR ATLAS OF DERMATOLOGY

By Professor L. K. Bhutani. 209 pp, illustrated. St Louis, Mo, Ishiyaku Euro-America, Inc, Publishers, 1988. $42.50 (hardcover)

This book was written by Professor Lalit K. Bhutani, head of the Department of Dermatology and Venereology at the All-India Institute of Medical Sciences in New Delhi.

The book is divided according to disease groups into 19 chapters, all of which are excellent. My favorites are the chapters on leprosy (full spectrum), tuberculosis of the skin, parasitic infestations, sexually transmitted diseases, and genodermatoses. These will be especially helpful to Western physicians who lack exposure to those conditions, but who may find themselves confronted with imported tropical cases. The common general dermatoses are fully covered in the other chapters. There is a special chapter, too, for unusual disorders.

Appropriately brief, concise but complete, descriptions of each disease condition appear on the left-hand page, describing the incidence, clinical features, histopathology (with photomicrographs), and treatment. There are some misspellings.

On the right-hand page opposing are color photographs, all taken by the author himself. These photographs are of excellent quality and are simply breath-taking in their beauty, sharpness, and instructiveness. One photograph is indeed worth a thousand words. One minor flaw is the mixed horizontal and vertical orientations of some of the photographs, undoubtedly done in an effort to accommodate the usual four in one page.

Very appropriately, there is an 11-page chapter on therapeutics at the end of the book. This deals with general principles of dermatologic treatment and the author's choices of formulary and prescriptions for specific dermatoses. And, very thoughtfully, there is a glossary of clinical dermatologic terms (depicted by photographs) and histologic terms.

Finally, in this age of rising prices, the cost of the book is a steal. For a similar color atlas, one can pay double or triple the price of this one. I recommend this book very highly to all general practitioners, family practitioners, internists, pediatricians, dermatologists, residents, and medical students.

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LIVER TUMORS: MULTIDISCIPLINARY MANAGEMENT

Edited by W. John B. Hodgson, MD. 323 pp, illustrated. St Louis, Mo, Warren H. Green, Inc, 1988. $47.50 (hardcover)

With the recent development of new technology and effective treatment regimens, interest in the diagnostic and therapeutic management of liver tumors has justifiably been increasing. Hodgson has put together an authoritative team of experts with extensive experience in the evaluation of patients with these disorders, based on the New York Medical College program. The purpose of this book is to compile scientific and clinical information concerning the interdisciplinary aspects of primary and metastatic hepatic neoplasms. Once armed with this vast amount of information, the clinician will be assisted in formulating an organized approach to their management. This book is an extremely useful addition to the medical literature because of the expertise of the authors and also because of the comprehensive nature of its contents.

The book begins with a detailed description of the vascularity of liver tumors, with elegant photographs of hepatic and neoplastic microcirculation. A discussion of early detection of liver tumors is followed by diagnostic evaluations including liver scintiscanning, computed tomographic (CT) scans, and angiography. The major content of the book addresses surgical management and includes discussions of incisions, surgical anatomy, hepatic artery ligation, surgical aspects of intrahepatic chemotherapy, hepatic resections, and pre- and post-operative management. The multidisciplinary approach is maintained with chapters on chemotherapy and radiation therapy of primary and metastatic liver malignancies. The final chapter brings together all the clinical information and presents an algorithm to assist the physician in planning total management of these patients.

Overall, the information presented is of high quality, accurate, and is written in a clear, readable manner. Certain sections of the book could have been strengthened. The section on tumor markers would benefit from a more extensive discussion of carcinoembryonic antigen (CEA), which still remains the only practical marker of gastrointestinal malignancies. The detailed discussion of colloid liver scintiscanning, while of historical interest, is perhaps of less clinical significance as it is yielding to CT scans and magnetic resonance imaging (MRI) as a diagnostic tool. The presentation of CT scans is worthy of any radiology textbook. I would have appreciated more information on MRI as we are finding that this new modality is identifying disease in patients with false negative CT scans. The surgical discussions cover all aspects of background data and thoroughly discuss all options for operative management in practical terms. The chapter on chemotherapy would be more complete with a more in-depth discussion of leucovorin and 5-FU pharmacology, as this combination is now considered standard treatment and is being recommended for adjuvant therapy for colorectal cancer. The book is well organized in the content it presents. The style and technical features, including figures, tables, and references, are extremely well done.

Clearly, this book ranks as one of the most desirable books on liver tumors and will be very useful, not only as a reference book, but also as a companion and guide for the clinician who is caring for patients with these diseases. The overall stated objectives, which are to present an alternative to therapeutic nihilism and to provide a wide range of treatment options, are clearly met by the authors.

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EXERCISE AND SPORT SCIENCES REVIEWS

Edited by Kent Pandolf, PhD. 551 pp, illustrated. New York, Macmillan Publishing Company, 1988. $48.00 (hardcover)

For the third consecutive year, Kent P. Pandolf, PhD, of the US Army Research Institute of Environmental Medicine, has been chosen to edit *Exercise and Sport Sciences Reviews*, an annual publication of the American College of Sports Medicine. Like its predecessors, the 16th volume reviews clinical and basic science research topics in exercise and sports medicine. Ten general topics are represented: psychology, motor control, athletic medicine, rehabilitation, biochemistry, exercise physiology, biomechanics, growth and development, environmental physiology, and sport sociology. Clinicians, students, educators, and scientists with interest in the sciences of sport and exercise are the target audience.

Volume 16 consists of 15 chapters written by 24 internationally recognized authorities. The first four chapters are devoted to the structure and function of muscle. Disuse atrophy, fatigue, force, and calcium-mediated events are presented in detail. Chapter 5 deals with the biomechanics of the spine. While the presentation is loaded with physics formulae, clinical correlations are discussed with clarity and force, thus making a difficult but important subject meaningful.

The next six chapters cover a broad range of topics including rehabilitation, athletic medicine, and physiology. A short section on knee cartilage reviews normal and pathologic structure and function. Thereafter, a lengthy chapter presents common and exotic sports-related dermatoses. The endocrine and neuroendocrine responses involving the hypothalmus; the pituitary, adrenal, thyroid, and sex glands; and the pancreas during exercise in stressful environments are discussed in detail in the following chapter. The effect and role of exercise in diabetes (type I), end-stage renal disease, and old age is summarized over an additional three chapters. The material provides ample information for a detailed exercise prescription, including indications, contraindications, risks, and benefits for patients in these categories.

Chapters 11 and 12 consider the psychologic impact of self-confidence and biofeedback on athletic performance. While research in these areas is scant, available evidence suggests that positive attitude and psychophysiologic training have palpable benefits requiring further study. The relatively unbiased section on gender and sport traces the development and current status of "women in sport." The individuality of adolescent changes in growth and performance is nicely appraised in the concluding chapter of the review.

The only major drawback to this volume, like others of the series, is that although extremely comprehensive with respect to the chosen topic area, the material represents only a small part of a very exciting field. It is likely that the reader will finish the volume desirous of similar reviews of other topic areas in exercise and sport science.

In summary, this volume represents a balanced synthesis of current clinical and basic scientific information in the field of exercise and sport. It will serve not only as a ready resource in clinical practice, but also as a standard reference in academe.

RICHARD B. BIRRER, MD
Consulting Editor

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PERFECTING THE WORLD. THE LIFE AND TIMES OF DR. THOMAS HODGKIN, 1798–1866

By Amalie M. Kass and Edward H. Kass. 624 pp, illustrated. New York, Harcourt Brace Jovanovich, Publishers, 1988. $34.95 (hardcover)

Many physicians, including myself, know very little about Thomas Hodgkin, except the fact that he described a malignant disease of the lymph glands. Professors Amalie and Edward Kass take us back into the early 19th century and tell the story of the life of this marvelous man in a very elegant manner. Their subject was a well-to-do Quaker with far reaching ideas, obviously ahead of his time, that endeared him to many and made enemies of more than a few. His life was full and productive. He experienced acclaim and rejection but remained strong in his beliefs and persistent in his desire for social reforms. His accomplishments include a short period as an apothecary's apprentice followed by a career as physician, teacher, and humane practitioner. He was probably the first to describe a defect in the aortic valve, appendicitis, and peritonitis. He also advocated preventive medicine. Dr Hodgkin traveled extensively, usually as a companion to a dear friend, Moses Montefiore. He objected to the oppression of Jews, American Indians, and black slaves and favored equal medical treatment for the rich and the poor. He set high standards for the education and training of physicians, but was devastated when Guy's Hospital failed to promote him. I was amazed to read that Thomas Hodgkin was the first to advocate health insurance. Hodgkin was also the first to introduce the stethoscope to England, and gave the first course in "pathologic anatomy."

This book will appeal to oncologists, social workers, and some teachers of preventive medicine. Others will find the reading very slow. Like so many before and after him, Thomas Hodgkin did not succeed in "perfecting the world."

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Lyme disease imitating a sports injury

An 11½-year-old female cyclist and swimmer sought treatment at a sports medicine clinic for pain in her left knee of seven months' duration (*Physician and Sportsmedicine* 1989; 17:135–136, 141). Two months prior to her first symptoms, she had bumped her knee while on a family camping trip. She had gone camping on several occasions during the previous year. Her history provided no further clues as to the cause of the knee pain.

One month after the pain began, x-rays of the knee taken by her family physician were normal. At this time, she began to use crutches. Four weeks later, a bone scan of the left knee performed by an orthopedic surgeon was also normal. She now relied on a knee immobilizer in addition to the crutches. Two months later, pain and swelling developed in her right knee. For the next three months, treatment consisted of bilateral knee immobilizers and physical therapy. Other concurrent medical symptoms included frequent headaches and throat infections, reported by the patient as strep throat.

Physical examination at the sports medicine clinic demonstrated "tenderness at the apex of both patellae and slight bilateral synovial thickening in the knees." Results of laboratory studies led to the diagnosis of Lyme disease. After one month of antibiotic therapy, 40 mg/kg/day of tetracycline, the pain and swelling in her knees had disappeared.

Lyme disease, caused by the spirochete *Borrelia burgdorferi*, is a multisystemic disorder that can imitate many other diseases. Initial symptoms vary and can include erythema chronicum migrans, fatigue, fever, chills, headache, sore throat, and arthralgia. If allowed to progress, Lyme disease is associated with symptoms related to cardiac disease such as palpitations, dizziness, and chest pain, as well as neurologic symptoms such as memory lapse, sleep disorders, and back pain. *B burgdorferi* is transmitted via infected ticks.

"Clinicians need to be suspicious of systemic illness when joint symptoms fail to respond to ordinary measures, such as physical therapy . . . . Sports medicine practitioners should be aware that mechanical injury is not the only cause of joint pain, and recurrent headaches and malaise may be symptoms of underlying disease."

Bicycle helmets greatly reduce risk of head and brain injury suffered in accidents

It seems obvious that wearing a helmet should help protect a bicycle rider from head and brain injuries suffered in accidents. Yet the majority of bicyclists apparently do not wear helmets, and children in particular infrequently don protective head gear when bicycling. Finding insufficient data to demonstrate statistically the protective effect of helmets, Thompson et al designed a case-control study to assess the effectiveness of bicycle helmets in preventing head and brain injuries (*N Engl J Med* 1989; 320:1361–1367).

The study population encompassed patients from five hospitals in the Seattle area. The case group comprised patients treated in an emergency room for head injuries resulting from a bicycle accident. There were two control groups: an emergency room control group composed of patients treated in an emergency room for bicycle-related injuries other than head injuries; and a population-based control group, which consisted of persons involved in bicycle accidents who may or may not have been injured or required medical care. The authors defined head injury as those injuries to the forehead, scalp, ears, skull, brain, or brain stem which a helmet could realistically prevent. Cases of concussion or more severe brain dysfunction were categorized as brain injuries.

During the one-year study period, the five hospital emergency rooms treated 776 people for bicycle-related injuries. Eighty-six percent of these patients responded to the authors' questionnaire, resulting in 235 case patients. Of these patients with head injuries, 42.1% suffered brain injuries. "Of the case patients with head injuries, 143 (60.9 percent) were children . . . .Sixty-five percent of the 99 brain injuries and 68 percent of the severe brain injuries . . . occurred in children under the age of 15." Only 4% of the 99 case patients with brain injuries wore helmets.

Helmet use overall was low: 7.2% of case patients and 23.8% of emergency room control patients. Bicyclists over 25 years old were far more likely to wear helmets.

Statistical analysis led to the following conclusions: bicycle helmets produced a 75% decrease in the risk of head injury and reduced the risk of brain injury by 85%. These compelling findings combined with the knowledge that, in 1985, for example, 574,000 emergency room visits and 1,300 deaths in the United States were attributable to bicycling injuries, emphasize the importance of urging all bicyclists to wear helmets. "Helmets are particularly important for children, since they suffer the majority of serious head injuries from bicycling accidents," stress the authors.

New test identifies persons at risk for diabetes

By analyzing the DNA extracted from white blood cells, researchers believe they can now identify persons at increased risk for developing Type I diabetes (*Science News* 1989; 135:353–368). Early recognition of this risk factor could help physicians develop the means to prevent the destruction of insulin-producing beta cells in the pancreas.

Currently, scientists believe that the body's own immune cells attack and destroy the beta cells in response to some "triggering factor," such as a virus or other environmental factor. Although there is no cure for Type I diabetes, research is focusing on immunosuppressive drugs that could block the destruction of beta cells. Such immunosuppressive agents would be most effective if treatment is begun in the early stages of the disease process. This new test, developed at the University of Pittsburgh School of Medicine, pinpoints persons with a genetic abnormality that scientists believe makes beta cells more susceptible to attack by immune cells.

People with this specific genetic alteration are "107

NEWS BRIEFS are compiled and written by Vicki Glaser.

times more likely than others to develop Type I diabetes." In families with a history of Type I diabetes, the new test could help identify children at increased risk for the disease.

High hopes for new anemia drug

The Food and Drug Administration (FDA) recently approved a genetically engineered form of erythropoietin to treat the anemia that accompanies chronic kidney failure (*The New York Times*, June 2, 1989). Clinical tests performed with the new drug have demonstrated that it could replace the need for blood transfusions in some patients who undergo kidney dialysis, and it could prove useful in combatting the anemia that occurs with AIDS and cancer.

The drug, epoetin alfa, marketed by Amgen, Inc (Thousand Oaks, Calif) under the name Epogen, stimulates the bone marrow to make red blood cells. Erythropoietin, a naturally occurring hormone synthesized in the kidney, is in short supply in patients with kidney failure. These patients must undergo multiple blood transfusions, risking infection, toxic levels of iron, and immune reactions, to prevent life threatening anemia.

While treatment of anemia in patients with chronic kidney failure is the intended purpose of this drug, doctors are already speculating on several other uses of epoetin. For example, early testing with epoetin in patients with acquired immunodeficiency syndrome (AIDS) has demonstrated its potential for treating the anemia caused by AZT, a drug used to lessen the symptoms of AIDS. Patients suffering from cancer, and those undergoing radiation or chemotherapy, may become anemic and could benefit from this new drug. Other proposed uses include stimulating "red blood cell production in patients expecting surgery, allowing them to donate more of their own blood to be stored before the operation. It could also be given to surgical patients after their operation to help them recover from their blood loss."

Treatment with epoetin entails intravenous administration three times a week, at an estimated cost of $4,000–$8,000 a year for each patient. Complications noted during clinical trials included high blood pressure, headache, chest pain, and, occasionally, seizures, all of which were common among patients with kidney failure who did not receive the drug. Development of epoetin was part of the FDA's orphan drug program, which supports drug development for diseases or disorders that affect 200,000 or fewer people in the United States.

Detecting malignant lesions with skin surface microscopy

The best hope for successful treatment of malignant melanoma is early detection. Clinical examination of cutaneous pigmented lesions can, at times, lead to diagnoses of benign lesions, nonmelanocytic lesions, or malignant melanoma. Other lesions require biopsy and histologic analysis for a definitive diagnosis. Only an estimated 65% of melanomas are accurately identified before surgery (*Lancet* 1989; 1:1239).

Skin surface microscopy is one method of evaluating pigmented lesions that could reduce the need for biopsy. The use of magnifications of ×6 to ×40 to examine pigmented lesions was first proposed in 1951. "At higher magnifications, the pigmentary pattern is slightly blurred; to counteract this effect, the technique of clearing the surface corneocytes by stripping with adhesive tape before applying a light film of oil was developed." A study using this technique demonstrated 85% diagnostic accuracy prior to biopsy.

Skin surface microscopy, using a standard operating microscope with a camera mounted on a side arm, is useful for distinguishing between melanocytic and nonmelanocytic pigmented lesions. The magnification enables visualization of the melanin pigment network, which appears as a "fine spider's web" along the periphery of melanocytic lesions only.

Histologic evaluation of biopsy samples is essential for differentiating benign from malignant melanocytic lesions. However, skin surface microscopy could hasten a diagnosis and limit the need for biopsy in some patients.

Home-infusion company flourishes with doctors' capital

Home-infusion companies provide intravenous drug and nutrient therapy to patients in their homes. The cost savings of home infusion compared to a hospital stay will help boost the annual growth of the home-infusion market by 20% over the next five years, according to industry analysts (*Business Week*, May 22, 1989, pp 92–93).

In 1984, two out-of-work medical-products salesmen recognized the market potential and established T^2 Medical, an Atlanta-based home-infusion company with 37 centers nationwide. Probably the most profitable home-infusion company in the US, T^2 boasts sales of about $35 million, with earnings of about $7 million this fiscal year.

The key to T^2's success was the clever marketing strategy developed by its founders. The capital for each infusion center was provided by local physicians, who, as owners and investors in the company, were motivated to steer patients to the local infusion centers. Competitors such as Caremark, a division of Baxter Healthcare Corp, acknowledged the effectiveness of this approach and began offering partnership and financial arrangements for referring physicians.

However, Congress subsequently passed a law, which will go into effect in 1990, that prohibits infusion centers owned by referring physicians from receiving Medicare payments. Under the new law, Medicare payments are permitted if each physician owns no more than 5% of a center to which he or she refers patients. T^2 is using stock sold in a public offering last year to buy back doctors' shares of the centers. Physicians will still own 50% of the company.

Although industry watchers predict continued growth for the home-infusion market, Congress has plans to further restrict investments by participating physicians, and the competition among home-infusion companies is heating up, with competitors now forced to offer more services and better prices to woo physicians and patients.

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Irving Harris Beckwith, MD, East Pembroke. Died December 28, 1988; age 86. Dr Beckwith was a 1928 graduate of the Cornell University Medical College, New York. He was a Diplomate of the American Board of Obstetrics and Gynecology and a Fellow of the American College of Surgeons. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

John Hans Birnbaum, MD, New York. Died April 7, 1989; age 94. Dr Birnbaum was a 1922 graduate of Friedrich-Wilhelms Universitaet Medizinische Fakultaet, Berlin, Germany. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Joseph Brody, MD, Forest Hills. Died January 26, 1988; age 86. Dr Brody was a 1929 graduate of the Cornell University Medical College, New York. His memberships included the American Geriatrics Society, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

William Thomas Carrington, MD, Neponsit. Died May 11, 1989; age 82. Dr Carrington was a 1932 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Robert Johnson Craft, MD, New Rochelle. Died May 3, 1989; age 89. Dr Craft was a 1927 graduate of the Howard University College of Medicine, Washington, DC. His memberships included the American Academy of Family Practice, the New York County Medical Society, and the Medical Society of the State of New York.

Charles De Bold, MD, Poughkeepsie. Died May 9, 1989; age 75. Dr De Bold was a 1941 graduate of the Hahnemann Medical College of Philadelphia, Pennsylvania. He was a Fellow of the American College of Surgeons. He was a member of the American Geriatrics Society, the Dutchess County Medical Society, and the Medical Society of the State of New York.

Joseph Fisher, MD, New York. Died October 26, 1988; age 80. Dr Fisher was a 1933 graduate of the New York Medical College, New York. His memberships included the American Rheumatism Association, the New York County Medical Society, and the Medical Society of the State of New York.

Samuel Bergman Frank, MD, San Diego, California. Died May 4, 1989; age 80. Dr Frank was a 1933 graduate of the Case Western Reserve University School of Medicine, Cleveland, Ohio. He was a Diplomate of the American Board of Dermatology, Inc, and a Fellow of the American College of Physicians. His memberships included the New York Academy of Medicine, the New York State Dermatological Society, the American Academy of Dermatology, the Society for Investigative Dermatology, the American Society of Tropical Medicine and Hygiene, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Sidney Freed, MD, Bethpage. Died May 22, 1989; age 70. Dr Freed was a 1944 graduate of the University of Toronto Faculty of Medicine, Toronto, Canada. He was a member of the American Geriatrics Society, the Nassau County Medical Society, and the Medical Society of the State of New York.

August Herman Groeschel, MD, Sea Girt, New Jersey. Died April 23, 1989; age 81. Dr Groeschel was a 1931 graduate of the Columbia University College of Physicians and Surgeons, New York. His memberships included the New York Academy of Medicine, the American Public Health Association, the New York County Medical Society, and the Medical Society of the State of New York.

Ernest E. Gyarmati, MD, North Bellmore. Died April 16, 1989; age 73. Dr Gyarmati was a 1952 graduate of the Orvosi Fakultas Tudomanyegyetem, Budapest, Hungary. He was a Fellow of the American Academy of Family Practice. His memberships included the New York Academy of Medicine, the American Psychiatric Association, the Nassau County Medical Society, and the Medical Society of the State of New York.

Aron Harel, MD, Forest Hills. Died April 15, 1989; age 67. Dr Harel was a 1956 graduate of The Hebrew University, Hadassah Medical School, Jerusalem, Israel. His memberships included the American Psychiatric Association, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Hedwig Koenig, MD, Newtown, Pennsylvania. Died April 14, 1989; age 94. Dr Koenig was a 1929 graduate of the Johns Hopkins University School of Medicine, Baltimore, Maryland. She was a Diplomate of the American Board of Pediatrics. Her memberships included the New York Academy of Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Harry Konterwitz, MD, Bedford, Massachusetts. Died May 14, 1989; age 85. Dr Konterwitz was a 1928 graduate of the University of Nebraska College of Medicine, Omaha, Nebraska. He was a Fellow of the American College of Chest Physicians. His memberships included the New York Cardiological Society, the American Thoracic Society, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Isaak Kuschner, MD, Oak Park, Michigan. Died April 1, 1989; age 92. Dr Kuschner was a 1934 graduate of the Universitaet Wien, Medizinische Fakultaet, Wien, Austria. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Hugh McHugh, MD, Larchmont. Died March 1989; age 81. Dr McHugh was a

1936 graduate of Temple University School of Medicine, Philadelphia. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the New York Academy of Medicine, the American Psychiatric Association, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

John Anthony Malgieri, MD, Crestwood. Died March 21, 1989; age 74. Dr Malgieri was a 1942 graduate of the New York Medical College. His memberships include the Association of Life Insurance Medical Directors of America, the Bronx County Medical Society, and the Medical Society of the State of New York.

De Witt Clinton Mead, MD, Fulton. Died April 17, 1989; age 84. Dr Mead was a 1933 graduate of Rush Medical College, Chicago, Illinois. He was a member of the Medical Society of the County of Oswego and the Medical Society of the State of New York.

Kaare Kristian Nygaard, MD, White Plains. Died April 23, 1989; age 86. Dr Nygaard was a 1929 graduate of the Medisinske Fakultet Universitetet i Oslo, Oslo, Norway. He was a Fellow of the American College of Surgeons. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Raphael W. Ollenstein, MD, Cranbury, New Jersey. Died March 11, 1989; age 90. Dr Ollenstein was a 1928 graduate of the Medizinische Fakultaet, Albert Ludwigs Universitaet, Freiburg im Breisgau, Baden-Wurttemberg, Germany. His memberships included the American Academy of Family Physicians, the Bronx County Medical Society, and the Medical Society of the State of New York.

Theodore Wright Oppel, MD, Boulder, Colorado. Died March 14, 1989; age 86. Dr Oppel was a 1929 graduate of the University of Pennsylvania School of Medicine, Philadelphia. He was a Diplomate of the American Board of Internal Medicine and a Fellow of the American College of Physicians. His memberships included the New York Academy of Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Angelos Papatestas, MD, New York. Died February 12, 1989; age 50. Dr Papatestas was a 1962 graduate of the Faculty of Medicine, Aristotelian University of Thessaloniki, Thessaloniki, Greece. He was a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons. His memberships included the New York State Surgical Society, the Society of Surgical Oncology, the Association of Academic Surgery, the New York County Medical Society, and the Medical Society of the State of New York.

Henry Poster, MD, Bronx. Died May 17, 1989; age 81. Dr Poster was a 1934 graduate of the St Louis University School of Medicine, St Louis, Missouri. He was a diplomate of the American Board of Radiology. His memberships included the New York Roentgen Society, the Bronx County Medical Society, and the Medical Society of the State of New York.

Julius Rock, MD, Rochester. Died April 14, 1989; age 79. Dr Rock was a 1936 graduate of the University of Rochester School of Medicine-Dentistry. He was a Diplomate of the American Board of Obstetrics and Gynecology and a Fellow of the American College of Obstetricians and Gynecologists. He was a member of the New York Academy of Medicine, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Pilar H. Roncal, MD, Middletown. Died March 18, 1989; age 50. Dr Roncal was a 1962 graduate of the Faculty of Medicine and Surgery University of Santo Tomas, Manila, Philippines. She was a member of the Medical Society of the County of Orange and the Medical Society of the State of New York.

Lawrence Jacob Roose, MD, New York. Died April 22, 1989; age 77. Dr Roose was a 1937 graduate of the New York University School of Medicine. He was a Diplomate of the American Board of Psychiatry and Neurology. He was a member of the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

David Rothstein, MD, Southbury, Connecticut. Died April 28, 1989; age 88. He was a 1925 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Zundel Sokolowicz, MD, Miami, Florida. Died May 11, 1989; age 79. Dr Sokolowicz was a 1938 graduate of the Charles Univerzita Fakulta of Ped-Gen Medicine, Praha, Czechoslovakia. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Hans J. Sowka, MD, Wilmington. Died March 14, 1989; age 76. Dr Sowka was a 1938 graduate of the Medizinische Fakultaet der Universitaet Heidelberg, Heildelberg, Baden-Wurttemberg, Germany. He was a Fellow of the American Academy of Family Physicians. He was a member of the Medical Society of the County of Essex and the Medical Society of the State of New York.

Henry Stern, MD, Laguna Hills, California. Died March 6, 1989; age 87. Dr Stern was a 1928 graduate of the Universitaet Wien, Medizinische Fakultaet, Wien, Austria. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Nestor J. Totero, MD, New York. Died May 6, 1989; age 82. Dr Totero was a 1934 graduate of New York Medical College. His memberships included the American Psychiatric Association, the Academy of Psychosomatic Medicine, the Association for the Advancement of Psychotherapy, the Bronx County Medical Society, and the Medical Society of the State of New York.

Stanley L. Wallace, MD, Brooklyn. Died June 20, 1989; age 65. Dr Wallace was a 1946 graduate of Bowman Gray School of Medicine of Wake Forest University, Winston-Salem, NC. He was a Fellow of the American College of Physicians and a member of the American Rheumatism Association, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Tobias M. Watson, MD, Palos Verdes, California. Died April 3, 1989; age 85. Dr Watson was a 1927 graduate of the Hahnemann Medical College of Philadelphia. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Marshall H. Wood, MD, Ossining. Died April 3, 1989; age 85. Dr Wood was a 1930 graduate of the Albany Medical College of Union University. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Tryphosa R. Worcester, MD, Crownsville, Maryland. Died April 19, 1989; age 93. Dr Worcester was a 1925 graduate of the University of Michigan Medical School, Ann Arbor, Michigan. She was a member of the New York County Medical Society and the Medical Society of the State of New York.

Leonard Jan Bruce-Chwatt, MD, FRCP
1907–1989

Dr Leonard Jan Bruce-Chwatt, a frequent contributor and peer reviewer for the *Journal*, passed away in England on May 18, 1989, at the age of 82. In April 1989, Dr Bruce-Chwatt was awarded the Laurence Redway Award for Excellence in Medical Writing by the Medical Society of the State of New York for his article, "Cinchona and its alkaloids: 350 years" (*NY State J Med* 1988; 88:318–322). A renowned medical historian and a leading international expert on malaria, he served for ten years (1958–1968) as Chief of Research and Technical Intelligence in the Malaria Eradication Division of the World Health Organization. Prior to this time he worked for a dozen years in Nigeria where he headed the malaria service. He later became Director of the Ross Institute at the London School of Hygiene and Tropical Medicine and served for many years as a member of the World Health Organization Expert Committee on Malaria.

Dr Bruce-Chwatt authored 430 medical papers and several books. His best known books are *Essential Malariology* (1980, 1985) and *The Rise and Fall of Malaria in Europe* (1980), which he coauthored with Julian de Zulueta. He held an MPH degree from Harvard University's School of Public Health and a DTM & H degree from the London School of Hygiene and Tropical Medicine. In 1976 Queen Elizabeth II personally conferred the honor of Companion of the Order of St Michael and St George (CMG) on him. He is survived by his wife Joan, sons Andrew and Robert, and a grandson, Rupert.

PASCAL JAMES IMPERATO, MD
Editor

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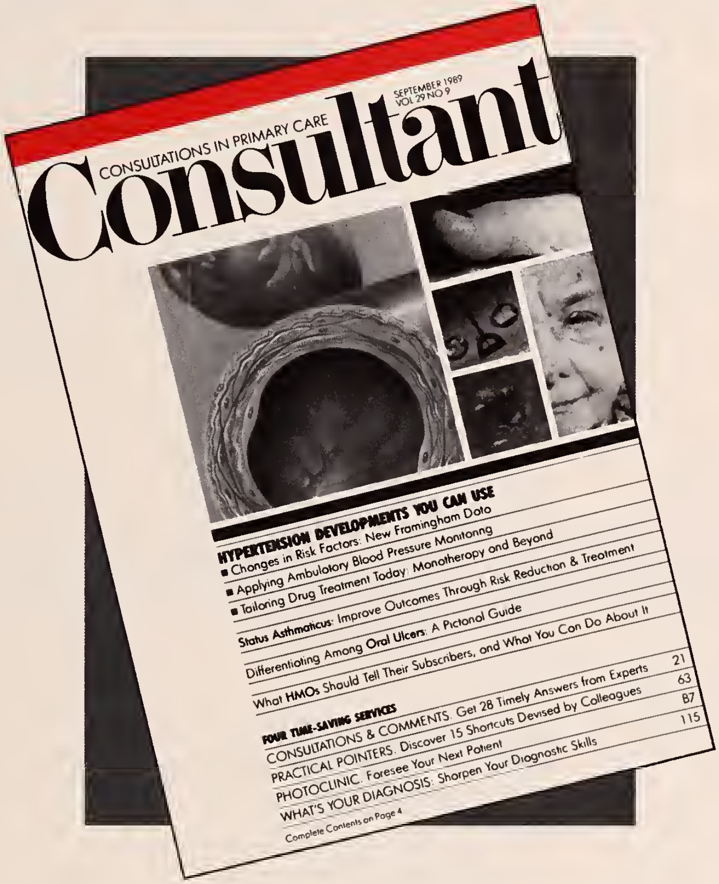

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VOL 29 NO 9

HYPERTENSION DEVELOPMENTS YOU CAN USE
- Changes in Risk Factors: New Framingham Data
- Applying Ambulatory Blood Pressure Monitoring
- Tailoring Drug Treatment Today: Monotherapy and Beyond

Status Asthmaticus: Improve Outcomes Through Risk Reduction & Treatment

Differentiating Among **Oral Ulcers**: A Pictorial Guide

What **HMOs** Should Tell Their Subscribers, and What You Can Do About It

FOUR TIME-SAVING SERVICES
CONSULTATIONS & COMMENTS: Get 28 Timely Answers from Experts 21
PRACTICAL POINTERS: Discover 15 Shortcuts Devised by Colleagues 63
PHOTOCLINIC: Foresee Your Next Patient 87
WHAT'S YOUR DIAGNOSIS: Sharpen Your Diagnostic Skills 115
Complete Contents on Page 4

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(*continued from p 6A*)

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Sept 23–25. **Applied Duplex Ultrasound: A Practical Course for Physicians and Sonographers.** Wynfrey Hotel Galleria Plaza, Birmingham. Contact: Dawne Ryals, Ryals & Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (404) 641-9773.

CALIFORNIA

Sept 7–10. **Fifth Annual Sonography Update for Physicians and Sonographers.** 15¾ Cat I Credits. Mariott Hotel and Marina, San Diego. *Sept 9–10.* **Registry Review Course—Physics, Abdomen, Gynecology, Obstetrics and Echocardiography.** 14½ Cat I Credits. San Diego Marriott Hotel and Marina, San Diego. Contact: Dawne Ryals, Ryals & Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (404) 641-9773.

Sept 15–16. **Uveitis Frontiers. Diagnostic, Medical, and Surgical Approaches to Uveitis.** Quail Lodge, Carmel Valley. Contact: Extended Program in Medical Education, University of California, Room U-569, San Francisco, CA 94143-0742. Tel: (415) 476-4251.

DISTRICT OF COLUMBIA

Sept 20–24. **18th Annual Meeting of the World Congress of Gynecologic Endoscopy.** J. W. Marriott Hotel, Washington, DC. Contact: The American Association of Gynecologic Laparoscopists, 13021 E Florence Ave, Santa Fe Springs, CA 90670. Tel: (213) 946-8774.

HAWAII

Sept 23–26. **Annual Seminar in Cardiology.** Sheraton Waikiki, Honolulu. Contact: Anne Criss, Rogers Heart Foundation, 1200 7th Ave, N, St Petersburg, FL 33705. Tel: (813) 894-0790.

MARYLAND

Sept 8–9. **Current Controversies and Techniques in Congenital Heart Surgery.** *Sept 25–30.* **Emergency Medicine Review.** University of Nebraska Medical Center. Contact: Brenda C. Ram, Continuing Education, University of Nebraska Medical Center, 42nd & Dewey Ave, Omaha, NE 68105. Tel: (402) 559-4152.

MASSACHUSETTS

Sept 6–8. **Risk Analysis in Occupational and Environmental Health.** *Sept 25–29.* **Industrial Ergonomics: Human Factors in Occupational Health & Safety.** Boston. Contact: Harvard School of Public Health, Office of Continuing Education, Dept A, 677 Huntington Ave, L-23, Boston, MA 02115. Tel: (617) 732-1171.

SOUTH CAROLINA

Sept 25–28. **Ninth Annual Postgraduate Course—Uroradiology '89.** Westin

(*continued on p 20A*)

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Calcium[1] and exercise are not enough to prevent postmenopausal osteoporosis, since estrogen deficiency is the primary cause.[2] PREMARIN is the only brand of estrogen indicated to prevent further bone loss and in a recently published study has been shown to reduce the risk of hip fractures by as much as 66%.[3]

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BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION AND PATIENT INFORMATION, SEE PACKAGE CIRCULARS.)*

PREMARIN® Brand of conjugated estrogens tablets, USP
PREMARIN® Brand of conjugated estrogens Vaginal Cream, in a nonliquefying base

> 1 ESTROGENS HAVE BEEN REPORTED TO INCREASE THE RISK OF ENDOMETRIAL CARCINOMA
> Three independent, case-controlled studies have reported an increased risk of endometrial cancer in postmenopausal women exposed to exogenous estrogens for more than one year This risk was independent of the other known risk factors for endometrial cancer These studies are further supported by the finding that incidence rates of endometrial cancer have increased sharply since 1969 in eight different areas of the United States with population-based cancer reporting systems, an increase which may be related to the rapidly expanding use of estrogens during the last decade The three case-controlled studies reported that the risk of endometrial cancer in estrogen users was about 4 5 to 13 9 times greater than in nonusers The risk appears to depend on both duration of treatment and on estrogen dose In view of these findings, when estrogens are used for the treatment of menopausal symptoms, the lowest dose that will control symptoms should be utilized and medication should be discontinued as soon as possible When prolonged treatment is medically indicated, the patient should be reassessed on at least a semi-annual basis to determine the need for continued therapy Although the evidence must be considered preliminary, one study suggests that cyclic administration of low doses of estrogen may carry less risk than continuous administration, it therefore appears prudent to utilize such a regimen Close clinical surveillance of all women taking estrogens is important In all cases of undiagnosed persistent or recurring abnormal vaginal bleeding, adequate diagnostic measures should be undertaken to rule out malignancy There is no evidence at present that "natural" estrogens are more or less hazardous than "synthetic" estrogens at equi-estrogenic doses
> 2 ESTROGENS SHOULD NOT BE USED DURING PREGNANCY
> The use of female sex hormones, both estrogens and progestogens, during early pregnancy may seriously damage the offspring It has been shown that females exposed in utero to diethylstilbestrol, a nonsteroidal estrogen, have an increased risk of developing, in later life, a form of vaginal or cervical cancer that is ordinarily extremely rare This risk has been estimated as not greater than 4 per 1,000 exposures Furthermore, a high percentage of such exposed women (from 30% to 90%) have been found to have vaginal adenosis, epithelial changes of the vagina and cervix Although these changes are histologically benign, it is not known whether they are precursors of malignancy Although similar data are not available with the use of other estrogens, it cannot be presumed they would not induce similar changes Several reports suggest an association between intrauterine exposure to female sex hormones and congenital anomalies, including congenital heart defects and limb-reduction defects One case-controlled study estimated a 4 7-fold increased risk of limb-reduction defects in infants exposed in utero to sex hormones (oral contraceptives, hormone withdrawal tests for pregnancy, or attempted treatment for threatened abortion). Some of these exposures were very short and involved only a few days of treatment. The data suggest that the risk of limb-reduction defects in exposed fetuses is somewhat less than 1 per 1,000 In the past, female sex hormones have been used during pregnancy in an attempt to treat threatened or habitual abortion There is considerable evidence that estrogens are ineffective for these indications, and there is no evidence from well-controlled studies that progestogens are effective for these uses If PREMARIN is used during pregnancy, or if the patient becomes pregnant while taking this drug, she should be apprised of the potential risks to the fetus, and the advisability of pregnancy continuation

DESCRIPTION: PREMARIN (conjugated estrogens, USP) contains a mixture of estrogens, obtained exclusively from natural sources, blended to represent the average composition of material derived from pregnant mares' urine It contains estrone, equilin, and 17α-dihydroequilin, together with smaller amounts of 17α-estradiol, equilenin, and 17α-dihydroequilenin as salts of their sulfate esters Tablets are available in 0 3 mg, 0 625 mg, 0 9 mg, 1 25 mg, and 2 5 mg strengths of conjugated estrogens Cream is available as 0 625 mg conjugated estrogens per gram

INDICATIONS AND USAGE: PREMARIN (conjugated estrogens tablets, USP) Moderate-to-severe *vasomotor* symptoms associated with the menopause (There is no evidence that estrogens are effective for nervous symptoms or depression without associated vasomotor symptoms and they should not be used to treat such conditions) Osteoporosis (abnormally low bone mass) Atrophic vaginitis Kraurosis vulvae Female castration

PREMARIN (conjugated estrogens) Vaginal Cream is indicated in the treatment of atrophic vaginitis and kraurosis vulvae

PREMARIN HAS NOT BEEN SHOWN TO BE EFFECTIVE FOR ANY PURPOSE DURING PREGNANCY AND ITS USE MAY CAUSE SEVERE HARM TO THE FETUS (SEE BOXED WARNING)

CONTRAINDICATIONS: Estrogens should not be used in women (or men) with any of the following conditions. 1 Known or suspected cancer of the breast except in appropriately selected patients being treated for metastatic disease 2 Known or suspected estrogen-dependent neoplasia 3 Known or suspected pregnancy (see Boxed Warning) 4 Undiagnosed abnormal genital bleeding 5 Active thrombophlebitis or thromboembolic disorders 6 A past history of thrombophlebitis, thrombosis, or thromboembolic disorders associated with previous estrogen use (except when used in treatment of breast or prostatic malignancy)

WARNINGS: Estrogens have been reported to increase the risk of endometrial carcinoma (see Boxed Warning) However, a recent large, case-controlled study indicated no increase in risk of breast cancer in postmenopausal women A recent study has reported a 2- to 3-fold increase in the risk of surgically confirmed gallbladder disease in women receiving postmenopausal estrogens

Adverse effects of oral contraceptives may be expected at the larger doses of estrogen used to treat prostatic or breast cancer or postpartum breast engorgement, it has been shown that there is an increased risk of thrombosis in men receiving estrogens for prostatic cancer and women for postpartum breast engorgement Users of oral contraceptives have an increased risk of diseases, such as thrombophlebitis, pulmonary embolism, stroke, and myocardial infarction Cases of retinal thrombosis, mesenteric thrombosis, and optic neuritis have been reported in oral contraceptive users An increased risk of postsurgery thromboembolic complications has also been reported in users of oral contraceptives If feasible, estrogen should be discontinued at least 4 weeks before surgery of the type associated with an increased risk of thromboembolism, or during periods of prolonged immobilization. Estrogens should not be used in persons with active thrombophlebitis, thromboembolic disorders, or in persons with a history of such disorders in association with estrogen use They should be used with caution in patients with cerebral vascular or coronary artery disease Large doses (5 mg conjugated estrogens per day), comparable to those used to treat cancer of the prostate and breast, have been shown to increase the risk of nonfatal myocardial infarction, pulmonary embolism, and thrombophlebitis When doses of this size are used, any of the thromboembolic and thrombotic adverse effects should be considered a clear risk

Benign hepatic adenomas should be considered in estrogen users having abdominal pain and tenderness, abdominal mass, or hypovolemic shock Hepatocellular carcinoma has been reported in women taking estrogen-containing oral contraceptives Increased blood pressure may occur with use of estrogens in the menopause and blood pressure should be monitored with estrogen use A worsening of glucose tolerance has been observed in patients on estrogen-containing oral contraceptives For this reason, diabetic patients should be carefully observed Estrogens may lead to severe hypercalcemia in patients with breast cancer and bone metastases

PRECAUTIONS: Physical examination and a complete medical and family history should be taken prior to the initiation of any estrogen therapy with special reference to blood pressure, breasts, abdomen, and pelvic organs, and should include a Papanicolaou smear As a general rule, estrogen should not be prescribed for longer than one year without another physical examination being performed Conditions influenced by fluid retention, such as asthma, epilepsy, migraine, and cardiac or renal dysfunction, require careful observation. Certain patients may develop manifestations of excessive estrogenic stimulation, such as abnormal or excessive uterine bleeding, mastodynia, etc Prolonged administration of unopposed estrogen therapy has been reported to increase the risk of endometrial hyperplasia in some patients Oral contraceptives appear to be associated with an increased incidence of mental depression. Patients with a history of depression should be carefully observed Pre-existing uterine leiomyomata may increase in size during estrogen use The pathologist should be advised of estrogen therapy when relevant specimens are submitted If jaundice develops in any patient receiving estrogen, the medication should be discontinued while the cause is investigated Estrogens should be used with care in patients with impaired liver function, renal insufficiency, metabolic bone diseases associated with hypercalcemia, or in young patients in whom bone growth is not yet complete If concomitant progestin therapy is used, potential risks may include adverse effects on carbohydrate and lipid metabolism.

Concomitant Progestin Use: The lowest effective dose appropriate for the specific indication should be utilized Studies of the addition of a progestin for 7 or more days of a cycle of estrogen administration have reported a lowered incidence of endometrial hyperplasia Morphological and biochemical studies of the endometrium suggest that 10 to 13 days of progestin are needed to provide maximal maturation of the endometrium and to eliminate any hyperplastic changes Whether this will provide protection from endometrial carcinoma has not been clearly established There are possible additional risks which may be associated with the inclusion of progestin in estrogen replacement regimens The choice of progestin and dosage may be important in minimizing possible adverse effects

The following changes may be expected with larger doses of estrogen.

a Increased sulfobromophthalein retention

b Increased prothrombin and factors VII, VIII, IX, and X, decreased antithrombin 3, increased norepinephrine-induced platelet aggregability

c Increased thyroid binding globulin (TBG) leading to increased circulating total thyroid hormone, as measured by PBI, T_4 by column, or T_4 by radioimmunoassay Free T_3 resin uptake is decreased, reflecting the elevated TBG, free T_4 concentration is unaltered.

d Impaired glucose tolerance

e Decreased pregnanediol excretion

f Reduced response to metyrapone test

g Reduced serum folate concentration.

h Increased serum triglyceride and phospholipid concentration

As a general principle, the administration of any drug to nursing mothers should be done only when clearly necessary since many drugs are excreted in human milk

Long-term, continuous administration of natural and synthetic estrogens in certain animal species increases the frequency of carcinomas of the breast, cervix, vagina, and liver However, in a recent, large case-controlled study of postmenopausal women there was no increase in risk of breast cancer with use of conjugated estrogens

ADVERSE REACTIONS: The following have been reported with estrogenic therapy, including oral contraceptives. breakthrough bleeding, spotting, change in menstrual flow, dysmenorrhea. premenstrual-like syndrome, amenorrhea during and after treatment, increase in size of uterine fibromyomata, vaginal candidiasis, change in cervical erosion and in degree of cervical secretion, cystitis-like syndrome, tenderness, enlargement, secretion (of breasts), nausea, vomiting, abdominal cramps, bloating, cholestatic jaundice, chloasma or melasma which may persist when drug is discontinued, erythema multiforme, erythema nodosum, hemorrhagic eruption; loss of scalp hair, hirsutism, steepening of corneal curvature, intolerance to contact lenses; headache, migraine, dizziness, mental depression, chorea. increase or decrease in weight, reduced carbohydrate tolerance, aggravation of porphyria, edema, changes in libido

ACUTE OVERDOSAGE: May cause nausea, and withdrawal bleeding may occur in females

DOSAGE AND ADMINISTRATION:

PREMARIN® Brand of conjugated estrogens tablets, USP

1 *Given cyclically for short-term use only* For treatment of moderate-to-severe *vasomotor* symptoms, atrophic vaginitis, or kraurosis vulvae associated with the menopause (0 3 mg to 1 25 mg or more daily). The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible Administration should be cyclic (eg, three weeks on and one week off) Attempts to discontinue or taper medication should be made at three- to six-month intervals.

2 *Given cyclically* Osteoporosis Female castration. Osteoporosis —0 625 mg daily Administration should be cyclic (eg, three weeks on and one week off) Female castration—1 25 mg daily, cyclically Adjust upward or downward according to response of the patient For maintenance, adjust dosage to lowest level that will provide effective control

Patients with an intact uterus should be monitored for signs of endometrial cancer and appropriate measures taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding

PREMARIN® Brand of conjugated estrogens Vaginal Cream

Given cyclically for short-term use only For treatment of atrophic vaginitis or kraurosis vulvae

The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible

Administration should be cyclic (eg, three weeks on and one week off)

Attempts to discontinue or taper medication should be made at three- to six-month intervals

Usual dosage range 2 g to 4 g daily, intravaginally, depending on the severity of the condition.

Treated patients with an intact uterus should be monitored closely for signs of endometrial cancer and appropriate diagnostic measures should be taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding

Revised October 1987

References:

1. Nilas L, Christiansen C, Rødbro P Calcium supplementation and postmenopausal bone loss *Br Med J* 1984, 289 1103-1106 **2**. Riggs BL Research Directions in Osteoporosis Scientific Workshop, National Institutes of Health, February 9-11, 1987 **3**. Kiel DP, Felson DT, Anderson JJ, et al Hip fracture and the use of estrogens in postmenopausal women *N Engl J Med* 1987,317 1169-1174 **4.** Christiansen C, Christensen MS, Transbøl I: Bone mass in postmenopausal women after withdrawal of oestrogen/gestagen replacement therapy *Lancet* 1981,1 459-461

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(*continued from p 15A*)

Resort Hotel, Hilton Head Island. Contact: Dawne Ryals, Ryals & Associates, Inc, PO Box 1925, Roswell, GA 30077-1925. Tel: (404) 641-9773.

TEXAS

Sept 29–30. **Neurology for the Primary Care Physician.** 11 Cat I Credits. Hyatt Regency Hotel on the Riverwalk, San Antonio. Contact: Marilyn Brandon, Office of Continuing Medical Education, Scott and White, 1401 S 31st St, Temple, TX 76508. Tel: (817) 774-2350.

AROUND THE WORLD

AUSTRALIA

Sept 12–16. **Annual Seminar in Cardiology.** Intercontinental Hotel, Sidney. *Sept 17–22.* **Annual Seminar in Cardiology.** Hyatt Hotel, Melbourne. Contact: Anne Criss, Rogers Heart Foundation, 1200 7th Ave, N, St Petersburg, FL 33705. Tel: (813) 894-0790.

CANADA

Sept 15–17. **Mammography and the Search for Breast Cancer.** Vancouver, British Columbia. Contact: Wende W. Logan, MD, 1351 Mt Hope Ave, Rochester, NY 14620. Tel: (716) 442-8432.

FRANCE

Sept 7–9. **Electrocardiography: Past and Future.** Beach Regency Hotel, Nice. Contact: Conference Director, The New York Academy of Sciences, 2 E 63rd St, New York, NY 10021. Tel: (212) 838-0230.

IRELAND

Sept 2–10. **Imaging, Intervention, Ireland—1989.** Dublin, Cong and New Market (Irish castles). Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077-1925. Tel: (404) 641-9773.

NEW ZEALAND

Sept 5–11. **Annual Seminar in Cardiology.** Regent Hotel, Auckland. Contact: Anne Criss, Rogers Heart Foundation, 1200 7th Ave, N, St Petersburg, FL 33705. Tel: (813) 894-0790. □

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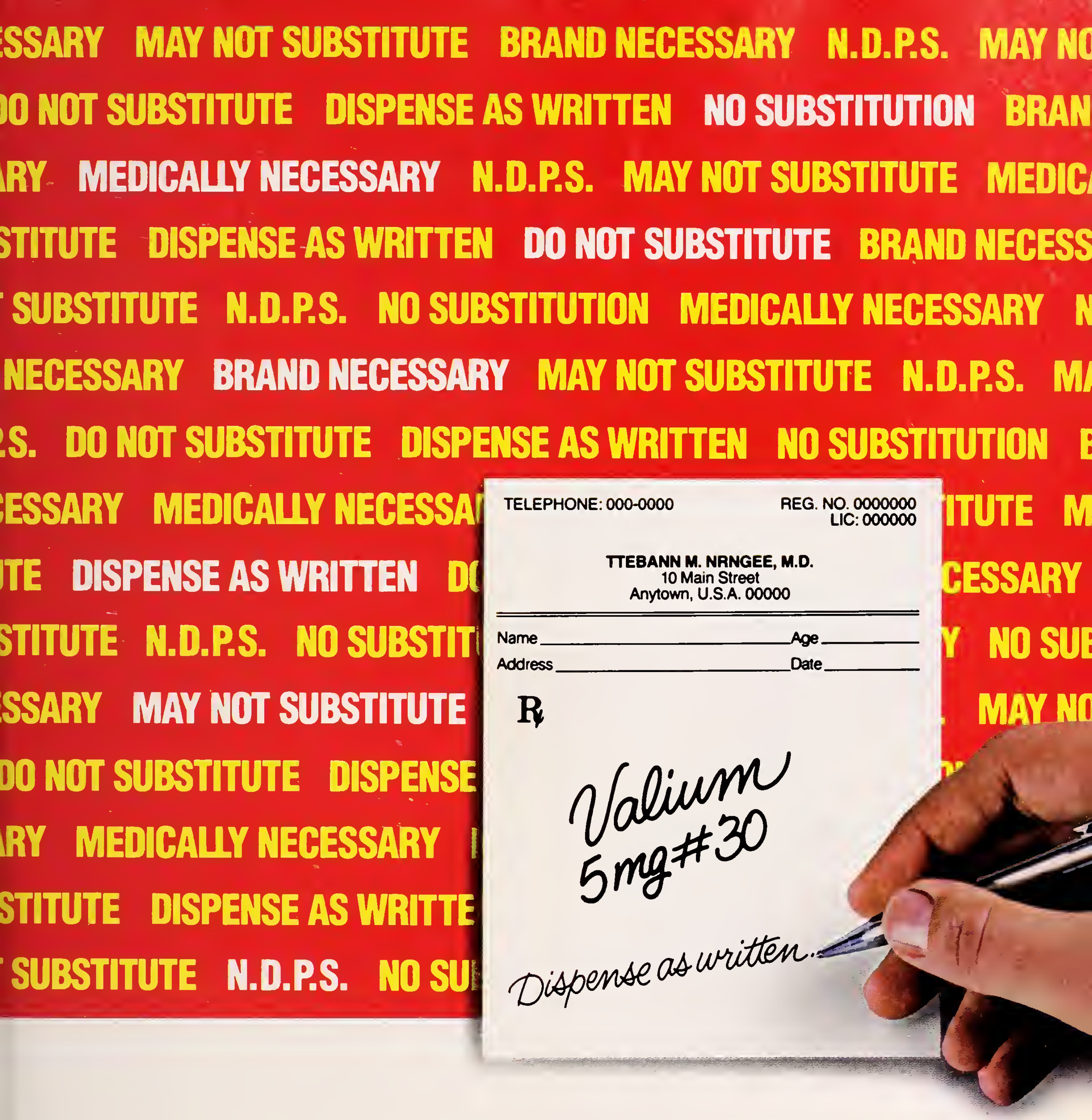
Wyeth-Ayerst Laboratories	16A, 17A, 18A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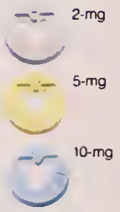

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Contents

COMMENTARIES

Ideopathic retroperitoneal fibrosis—past and present 503
THOMAS W. STEWART, JR, MD

Thyroid needle biopsy: The primary procedure 504
RONALD P. MONSAERT, MD

RESEARCH PAPER

Human immunodeficiency virus infection in a cohort of intravenous drug users in New York City: Demographic, behavioral, and clinical features 506
LAWRENCE S. BROWN, JR, MD, MPH; ALVIN CHU, MA; TOORU NEMOTO, PHD, MA; DAVID AJULUCHUKWU, PHD, MPA; BENY J. PRIMM, MD

REVIEW ARTICLE

Retroperitoneal fibrosis: A report of selected cases and a review of the literature 511
DANIEL D. BUFF, MD; MARC B. BOGIN, MD; LAWRENCE L. FALTZ, MD

SPECIAL ARTICLE

Current challenges of AIDS in New York City 517
STEPHEN C. JOSEPH, MD, MPH

HISTORY

A short history of the quack's materia medica 520
JOHN S. HALLER, JR, PHD

GUIDELINES

Student participation in mixed gender sports competition 526
COMMITTEE ON SCHOOL HEALTH AND SPORTS MEDICINE

CASE REPORTS

The clinical value of fine needle aspiration biopsy in a patient with medullary thyroid carcinoma and pseudofollicular carcinoma 527
ARTHUR BAUMAN, MD; H. T. G. STRAWBRIDGE, MD; WILLIAM A. BAUMAN, MD

Renal arteriovenous fistula in the setting of subacute bacterial endocarditis 530
THOMAS F. STEPHENSON, MD; JOHN F. McCABE, MD; JERRY J. SVOBODA, MD; MALUR R. BALAJI, MD; MICHAEL J. FEINSTEIN, MD; REVINDRA MEHTA, MD

Ultrasound diagnosis of jugular venous ectasia 532
CHRISTOPHER GRIBBIN, MD; B. NAGESH RAGHAVENDRA, MD; HOWARD B. GINSBURG, MD

Malignant lymphoma presenting with severe hypercalcemia 533
SAMUEL R. SIRIANNI, MD; PHD; MAXIMES E. MORA, MD; AMY M. SANDS, MD; DEBORAH PUCKHABER, MD; TERRY J. SMITH, MD

LETTERS TO THE EDITOR

Buck v Bell 536
THEODORE SCHUSSLER, MD, JD

Physician recredentialing 536
DAVID I. ATKINSON, MD

Complications arising from the use of ophthalmologic medications in an intensive care unit patient 537
ZVI HERSCHMAN, MD, BRIAN KAUFMAN, MD

Clinical causes of death of elderly patients in a long term health care facility 538
WILBERT S. ARONOW, MD

LEADS FROM EPIDEMIOLOGY NOTES 540

BOOK REVIEWS 544

NEWS BRIEFS 548

OBITUARIES 550

MEDICAL MEETINGS AND LECTURES 4A

THE LOWER RESPIRATORY TRACT—

More vulnerable to infection in smokers and older adults

Experience counts

Ceclor® Pulvules® 250 mg
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For respiratory tract infections due to susceptible strains of indicated organisms.

Summary.
Consult the package literature for prescribing information.

Indication: Lower respiratory infections, including pneumonia, caused by *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Streptococcus pyogenes* (group A β-hemolytic streptococci).
Contraindication: Known allergy to cephalosporins
Warnings: CECLOR SHOULD BE ADMINISTERED CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS PENICILLINS AND CEPHALOSPORINS SHOW PARTIAL CROSS-ALLERGENICITY POSSIBLE REACTIONS INCLUDE ANAPHYLAXIS

Administer cautiously to allergic patients

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics. It must be considered in differential diagnosis of antibiotic-associated diarrhea Colon flora is altered by broad-spectrum antibiotic treatment, possibly resulting in antibiotic-associated colitis

Precautions:
- Discontinue Ceclor in the event of allergic reactions to it.
- Prolonged use may result in overgrowth of nonsusceptible organisms
- Positive direct Coombs' tests have been reported during treatment with cephalosporins
- Ceclor should be administered with caution in the presence of markedly impaired renal function Although dosage adjustments in moderate to severe renal impairment are usually not required, careful clinical observation and laboratory studies should be made
- Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis
- Safety and effectiveness have not been determined in pregnancy, lactation, and infants less than one month old Ceclor penetrates mother's milk Exercise caution in prescribing for these patients

Adverse Reactions: (percentage of patients)

Therapy-related adverse reactions are uncommon Those reported include:
- Gastrointestinal (mostly diarrhea): 2.5%
- Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment.
- Hypersensitivity reactions (including morbilliform eruptions, pruritus, urticaria, and serum-sickness-like reactions that have included erythema multiforme [rarely, Stevens-Johnson syndrome] and toxic epidermal necrolysis or the above skin manifestations accompanied by arthritis/arthralgia, and frequently, fever): 1.5%, usually subside within a few days after cessation of therapy Serum-sickness-like reactions have been reported more frequently in children than in adults and have usually occurred during or following a second course of therapy with Ceclor No serious sequelae have been reported Antihistamines and corticosteroids appear to enhance resolution of the syndrome
- Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy
- As with some penicillins and some other cephalosporins, transient hepatitis and cholestatic jaundice have been reported rarely
- Rarely, reversible hyperactivity, nervousness, insomnia, confusion, hypertonia, dizziness, and somnolence have been reported
- Other eosinophilia, 2%, genital pruritus or vaginitis, less than 1%, and, rarely, thrombocytopenia

Abnormalities in laboratory results of uncertain etiology
- Slight elevations in hepatic enzymes
- Transient fluctuations in leukocyte count (especially in infants and children).
- Abnormal urinalysis, elevations in BUN or serum creatinine
- Positive direct Coombs' test
- False-positive tests for urinary glucose with Benedict's or Fehling's solution and Clinitest® tablets but not with Tes-Tape® (glucose enzymatic test strip, Lilly)

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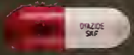

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Indications and Usage: *Hypertension or edema in patients who develop hypokalemia on hydrochlorothiazide alone, in patients who require a thiazide diuretic and in whom the development of hypokalemia cannot be risked*

This fixed combination drug is not indicated for the initial therapy of edema or hypertension except in individuals in whom the development of hypokalemia cannot be risked.

'Dyazide' may be used alone or as an adjunct to other antihypertensive drugs; dosage adjustments may be necessary

Contraindications *Concomitant use with other potassium-sparing agents such as spironolactone or amiloride, potassium supplements (except in presence of severe hypokalemia), anuria, acute and chronic renal insufficiencies or significant renal impairment, hypersensitivity to drug or other sulfonamide-derived drugs, preexisting elevated serum potassium concentration*

Warnings *Abnormal elevation of serum potassium levels (greater than or equal to 5 5 mEq/liter) can occur with all potassium-conserving diuretic combinations, including 'Dyazide' Hyperkalemia is more likely to occur in patients with renal impairment, diabetes (even without evidence of renal impairment), elderly or severely ill patients. Since uncorrected hyperkalemia may be fatal, serum potassium levels must be monitored at frequent intervals especially in patients first receiving 'Dyazide', when dosages are changed or with any illness that may influence renal function.*

If hyperkalemia is suspected, obtain an ECG and monitor serum potassium If hyperkalemia develops, discontinue 'Dyazide' and initiate thiazide therapy if needed Persistent hyperkalemia may require dialysis Monitor serum electrolytes frequently in patients with mild renal dysfunction and in diabetic patients In patients who may develop respiratory or metabolic acidosis, monitor serum electrolytes and acid/base balance frequently

Precautions *The bioavailability of the hydrochlorothiazide and triamterene components of 'Dyazide' is about 50% of the maximum obtainable with oral therapy Theoretically, a patient transferred from therapy with hydrochlorothiazide with or without triamterene might show an increase in blood pressure, fluid retention, or change in serum potassium Extensive clinical experience with 'Dyazide', however, suggests that these conditions have not been commonly observed in clinical practice (See CLINICAL PHARMACOLOGY.) Use thiazides cautiously in patients with impaired hepatic function They can precipitate coma in patients with severe liver disease, potassium depletion induced by the thiazide may be important in this connection, administer 'Dyazide' cautiously and be alert for such early signs of impending coma as confusion, drowsiness and tremor, if mental confusion increases, discontinue 'Dyazide' for a few days, attention must be given to other factors that may precipitate hepatic coma, such as blood in the gastrointestinal tract or preexisting potassium depletion. If patients develop hypokalemia, which is uncommon with 'Dyazide', increase potassium intake (i.e., with supplements or potassium-rich foods) If repeat determinations show serum potassium concentrations below 3 0 mEq/L, discontinue 'Dyazide' and initiate potassium chloride supplementation. Institute corrective measures cautiously and monitor serum potassium concentrations frequently, especially in patients receiving digitalis or those with a history of cardiac arrhythmias Diuretics may aggravate existing electrolyte imbalances, especially at high dosages or in patients on salt-restricted diets. Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Chloride replacement may be required in the treatment of metabolic acidosis If dilutional hyponatremia develops, restrict water intake In actual salt depletion, initiate sodium chloride replacement. Use 'Dyazide' cautiously in patients with a history of renal stone formation*

If hyperkalemia develops when treating for hypokalemia, take corrective measures. Also discontinue 'Dyazide' and, if appropriate, substitute a thiazide diuretic until potassium levels return to normal Do periodic BUN and serum creatinine determinations, especially in the elderly and in patients with suspected or confirmed renal insufficiency. Serum PBI levels may decrease without signs of thyroid disturbance Discontinue thiazides before conducting parathyroid function tests

Angiotensin-converting enzyme (ACE) inhibitors can elevate serum potassium, use with caution with 'Dyazide' Concurrent use with chlorpropamide may increase the risk of severe hyponatremia A few occurrences of acute renal failure have been reported in patients on 'Dyazide' when treated with indomethacin Therefore, caution is advised in administering nonsteroidal anti-inflammatory agents with 'Dyazide' Diuretics reduce renal clearance of lithium and increase the risk of lithium toxicity Clinically insignificant reductions in arterial responsiveness to norepinephrine have been reported Thiazides have also been shown to increase the paralyzing effect of nondepolarizing muscle relaxants such as tubocurarine, therefore use cautiously in patients undergoing surgery Monitor electrolytes in patients taking amphotericin B, corticosteroids or corticotropin concomitantly Thiazides may potentiate the action of other antihypertensive drugs The effects of oral anticoagulants may be decreased when used concurrently with hydrochlorothiazide, dosage adjustments may be needed 'Dyazide' may raise the level of blood uric acid; dosage adjustments of antigout medication may be needed to control hyperuricemia and gout The following agents given with triamterene may promote serum potassium accumulation and possibly result in hyperkalemia, especially in patients with renal insufficiency: blood from blood bank (may contain up to 30 mEq of potassium per liter of plasma or up to 65 mEq of potassium per liter of whole blood when stored for more than 10 days), low-salt milk (may contain up to 60 mEq of potassium per liter); potassium-containing medications (such as parenteral penicillin G potassium), and salt substitutes (most contain substantial amounts of potassium). Exchange resins, such as sodium polystyrene sulfonate, whether administered orally or rectally, reduce serum potassium concentrations by sodium replacement of the potassium, fluid retention may occur in some patients because of the increased sodium intake Chronic or overuse of laxatives may reduce serum potassium concentrations by promoting excessive potassium loss from the intestinal tract, laxatives may interfere with the potassium-retaining effects of triamterene The effectiveness of methenamine may be decreased when used concurrently with hydrochlorothiazide because of alkalinization of the urine 'Dyazide' will interfere with the fluorescent measurement of quinidine

There are no adequate and well-controlled studies in pregnant women This drug should be used during pregnancy only if clearly needed Thiazides and triamterene cross the placental barrier and appear in cord blood The use of thiazides in pregnancy requires weighing the anticipated benefit against possible hazards, including fetal or neonatal jaundice, pancreatitis, thrombocytopenia, and possibly other adverse reactions which have occurred in the adult. Thiazides appear, and triamterene may appear, in breast milk If use of the drug is essential, the patient should stop nursing Safety and effectiveness in children have not been established

Adverse Reactions *The serious adverse effects associated with 'Dyazide' have commonly occurred in less than 0 1% of patients treated with this product. Anaphylaxis, rash, urticaria, photosensitivity, cardiac arrhythmias, postural hypotension, diabetes mellitus, hyperkalemia, hyperglycemia, glycosuria, hyperuricemia, hypokalemia, hyponatremia, acidosis, hypochloremia, jaundice and/or liver enzyme abnormalities, pancreatitis, nausea and vomiting, diarrhea, constipation, abdominal pain, acute renal failure, interstitial nephritis, renal stones composed primarily of triamterene, elevated BUN and serum creatinine, abnormal urinary sediment, leukopenia, thrombocytopenia and purpura, megaloblastic anemia, muscle cramps, weakness, fatigue, dizziness, headache, dry mouth, impotence, sialadenitis Thiazides alone have been shown to cause the following additional adverse reactions. paresthesias, vertigo, xanthopsia, transient blurred vision, allergic pneumonitis, pulmonary edema, respiratory distress, necrotizing vasculitis, exacerbation of lupus, aplastic anemia, agranulocytosis, hemolytic anemia In neonates and infants. thrombocytopenia and pancreatitis—rarely, in newborns whose mothers have received thiazides during pregnancy*

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MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices. Information must be submitted at least three months prior to the event.*

OCTOBER 1989

AROUND THE STATE

BRONX

Oct 25. **The Sam Sheppard Murder Trial Revisited.** Lehman College. Contact: Mark L. Taff, MD, President, New York Society of Forensic Sciences, 130 Gold Pl, Malverne, NY 11565. Tel: (516) 887-4691.

COOPERSTOWN

Oct 11. **Magnetic Resonance Imaging.** 3 Cat I Credits. Bassett Hall Conference Center. Contact: Charlotte L. Hoag, Medical Education Coordinator, Office of Medical Education, The Mary Imogene Bassett Hospital, One Atwell Rd, Cooperstown, NY 13326-1394. Tel: (607) 547-3926.

EAST MEADOW

Oct 19. **Cooley's Anemia: New Developments and Therapeutic Strategies for the Next Decade.** 4 Cat I Credits. Nassau County Medical Center. Contact: Dr Dominick Sabatino, Department of Pediatrics, Nassau County Medical Center, East Meadow, NY 11554. Tel: (516) 542-2177.

MANHATTAN

Oct 4–7. **MRI: Clinical State of the Art.** 24 Cat I Credits. *Oct 16–20.* **Five Day Emergency Medicine Board Review Course.** 38 Cat I Credits. *Oct 24, 26, 31, Nov 2.* **Review of Medical Mycology.** 12 Cat I Credits. NYU Medical Center. Contact: NYU Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

Oct 5–6. **12th Annual Current Concerns in Adolescent Medicine. A Practical Approach to Current Problems in Adolescents.** 12 Cat I Credits. The Grand Hyatt. Contact: Ann J. Boehme, Associate Director for Continuing Education, Schneider Children's Hospital of Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

Oct 10. **16th Arvilla Berger Lecture: Modern Medical Management of Myocardial Infarction.** 2 Cat I Credits. New York Helmsley Hotel. Contact: New York Cardiological Society, Inc, PO Box 668, FDR Station, New York, NY 10150. Tel: (212) 924-5346.

Oct 11–12. **American Board of Quality Assurance and Utilization Review Physicians 11th Annual Lecture Course and Certification Examination.** 8 Cat I Credits. The New York City Marriott Marquis. Contact: ABQAURP, Murdock Professional Center, 1777 Tamiami Trail, Box 12, Port Charlotte, FL 33948. Tel: (813) 743-2425.

Oct 11–14. **The 12th Annual Postgraduate Medicine Course.** 23½ Cat I Credits. Columbia University. Contact: Center for Continuing Education, College of Physicians & Surgeons of Columbia University, 630 W 168th St, New York, NY 10032. Tel: (212) 305-3682.

NEW HYDE PARK

Oct 14–15. **Pearls in Office Urology.** Teaching Center Auditorium, Long Island Jewish Medical Center. Contact: Ann J. Boehme, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

AROUND THE NATION

ALABAMA

Oct 13–15. **Applied Ultrasound: Accent on Doppler.** 18 Cat I Credits. Wynfrey Hotel Galleria Plaza, Birmingham. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077-1925. Tel: (404) 641-9773.

CALIFORNIA

Oct 2–5. **Update in Pediatric Imaging for the Practicing Radiologist.** 18 Cat I Credits. Ritz-Carlton Hotel, Laguna Niguel. *Oct 28–29.* **Advanced Imaging of the Musculoskeletal System.** Hotel del Coronado, Coronado. 9 Cat I Credits. *Oct 30–Nov 3.* **14th Annual San Diego Postgraduate Course.** 27 Cat I Credits. Hotel del Coronado, San Diego. Contact: Dawne Ryals, Ryals & Associates, PO Box 920113, Norcross, GA 30092-0113. Tel: (404) 641-9773.

Oct 2–6. **Clinical Magnetic Resonance Imaging.** *October 7.* **Pediatric Neuroradiology.** Hyatt Regency. San Francisco. Contact: Radiology Postgraduate Education, Registration Office, University of California—Room 569U, San Francisco, CA 94143. Tel: (415) 476-5731.

Oct 14–15. **The Skin from A to Z. Tips on Practical Therapy and Office Techniques.** 10 Cat I Credits. Laurel Heights Campus. San Francisco. Contact: Extended Programs in Medical Education, Room U-569, University of California School of Medicine, San Francisco, CA 94143-0742. Tel: (415) 476-4251.

COLORADO

Oct 2–6. **15th Annual Conference of the American Association of Pathologist's Assistants.** Denver Marriott, Southeast. Contact: Mary A. Latino, Department of Pathology, Nyack Hospital, N Midland Ave, Nyack, NY 10960. Tel: (914)358-6200, ext 2270.

HAWAII

Oct 14–21. **14th Annual International Body Imaging Conference.** Hyatt Regency Waikoloa Hotel, Waikoloa, Kona. Contact: Body Imaging Conference, 9800 D. Topanga Canyon Blvd, Suite 232, Chatsworth, CA 91311. Tel: (818) 700-9821.

MISSOURI

Oct 5–7. **Biomedical Image Processing with Microcomputers.** Mariott's Pavilion Hotel, St Louis. Contact: Loretta Giacoletto, Washington University School of Medicine, Office of Continuing Medical Education, 660 S Euclid, Box 8063, St Louis, MO 63110. Tel: (800) 325-9862.

Oct 31–Nov 5. **40th Annual Workshop and Scientific Program of the Society for Clinical and Experimental Hypnosis.** 43 Cat I Credits. Clarion Hotel, St

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COMMENTARIES

Idiopathic retroperitoneal fibrosis—past and present

> *The following 2 cases I deem worthy of presentation to you because not only are they unique in my experience but I have so far been unable to find any similar cases in the literature.*[1]

More than 40 years have passed since Dr John K. Ormond, a urologic surgeon at the Henry Ford Hospital in Detroit, Michigan, first presented two cases of retroperitoneal fibrosis (RF) to the American Association of Genito-Urinary Surgeons. He later published his findings as "Bilateral ureteral obstruction due to envelopment and compression by an inflammatory retroperitoneal process" in the *Journal of Urology*,[1] the first report in the English literature of the entity he described.

In this issue of the *Journal*, Buff and colleagues[2] present a timely and thoroughly comprehensive review of this baffling and not-so-rare disease. Though much has been learned, it is indeed impressive to note that so many of Ormond's initial observations remain valid. The current review allows reflection on the advances that have been made over four decades in the diagnosis and treatment of this process, as well as on the difficulties that remain.

> *We were much puzzled by the course of events in the first case . . . .As I look back on it, it seems that we were rather stupid not to recognize that there was ureteral obstruction of extra-ureteral origin and . . . explored the abdomen.*[1]

Ormond's confusion is easily understood, since he and his colleagues were dealing with an entity not previously described. Evaluation of the retroperitoneum in that era was limited to the conclusions drawn from an intravenous pyelogram and direct surgical exploration.

In the late 1980s, RF is readily recognized in most instances. Typical radiographic findings are well described, and preoperative evaluation of the retroperitoneum is now greatly enhanced by computerized tomography and ultrasonography. Still, definitive diagnosis of idiopathic RF remains dependent on surgical exploration with biopsy to exclude any specific cause of secondary fibrosis of the retroperitoneum (eg, occult malignancy).

> *Autopsy showed a fibrous retroperitoneal mass covering the promontory of the sacrum and extending up to the kidney. The ureters, which were structurally normal and showed no strictures, ran through the lateral margins of this mass, and were enveloped and compressed by it, but not invaded.*
>
> *I quote from the autopsy report . . . "Sections of this dense fibrous mass reveal that it is composed entirely of extremely dense bundles of collagen. This tissue is poorly nucleated, but the cells, where seen, are of typical fibrous type. This fibrous connective tissue surrounds without invading the large vessels, lymph nodes, nerves and ureters."*[1]

Even with a now vast collective experience, it is scarcely possible to improve on this first description of the pathologic findings in retroperitoneal fibrosis.

> *We have no theories to offer as to the genesis or development of these inflammatory masses or processes.*[1]

With several hundred cases reported there is still much speculation about the etiology (or etiologies) of idiopathic RF. In their review, Buff and colleagues well summarize the current literature.[2] Reports of some patients with fibrosis involving multiple sites and associated orbital pseudotumor suggest that RF may be but the most prominent manifestation of a systemic disorder. An intriguing and promising area of investigation concerns the suspected autoimmune nature of retroperitoneal fibrosis, although the evidence remains less than conclusive.

While the idiopathic disease predominates, much attention has focused on drug-related RF, with convincing evidence implicating only a single agent—methysergide (Sansert®), a serotonin antagonist with vasoconstrictive properties, used in the prophylaxis of vascular headaches. The mechanism by which the fibrosis is produced remains an open question. The occurrence of RF with methysergide is rare and generally reported only with prolonged, uninterrupted use. Furthermore, the fibrosis is usually reversible in the early stages if the drug is discontinued.[3] With the advent of other agents effective in the prevention of vascular headaches, the widespread use of methysergide has declined significantly. Still, the drug can be singularly effective in some patients, particularly those with cluster (Horton's) headache, and it may be used with relative safety, provided established guidelines are followed.[4]

The idiopathic and methysergide-related RF are virtually indistinguishable clinically and pathologically. Unfortunately, the mechanism of disease for both remains enigmatic.

> *The ureters were located below this mass, traced up, and found to pass through this tissue near its lateral margins. Both ureters were dissected entirely free from this enveloping tissue and seemed entirely normal. They were left free in the abdominal cavity . . . He was given deep x-ray therapy directed at the inflammatory mass . . .* [1]

Ureterolysis remains the logical mainstay of surgical therapy. It is interesting to note that Ormond utilized radiation therapy in his second patient, but its use in the treatment of RF never gained wide acceptance. Immunosuppressive therapy, although not effective in all patients, represents the singular significant advance in the nonsurgical treatment of idiopathic RF, giving credence to the proposed autoimmune etiology.

> [*The patient*] *is no longer anemic, has put on weight, is working regularly and feels the best he has felt in several years.*[1]

At the time of Ormond's initial report his second patient was well, with no evidence of recurrent ureteral obstruction. Even as late as 1960, when a second paper was published, and 14 years after diagnosis and surgery, the patient had no urinary symptoms or renal impairment.[5] Without surgical exploration and ureterolysis, the first patient died in uremia following a prolonged course.

Four decades after Ormond's original report, idiopathic retroperitoneal fibrosis remains an enigma, but physicians treating RF can anticipate a favorable prognosis in the vast majority of cases with appropriate surgical and medical therapy. Many questions persist, but for those in the healing art and their patients, an effective treatment is always more treasured than even the most detailed understanding of pathophysiology.

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Thyroid needle biopsy: The primary procedure

Needle biopsy of the thyroid has proven to be a valuable diagnostic tool for the evaluation of thyroid nodules. The evaluation of nodules using previously available technology such as thyroid scans, ultrasound, and suppression studies with exogenous thyroid hormone provided a specificity for cancer of only about 20%. Thus, 80% of patients would have had needless thyroid surgery. Thyroid needle biopsy can identify cancer in more than 96% of patients.[1]

The biopsy can identify the type of cancer, and thus both surgeon and patient can anticipate the extent of surgical treatment and postoperative plans. In this issue of the *Journal*, Bauman et al[2] report on a case of medullary carcinoma of the thyroid, in which the diagnosis was made preoperatively by needle biopsy. The patient was appropriately screened for a possible attendant pheochromocytoma. Approximately one third of patients with medullary carcinoma will have the multiple endocrine neoplasia syndrome type II (MEN-II) (medullary carcinoma of the thyroid, hyperparathyroidism, and pheochromocytoma). If pheochromocytoma had been discovered, the first operative procedure would have been to remove the adrenal tumor after appropriate catecholamine blockade. A possible adverse outcome might have resulted if the initial procedure had been a thyroidectomy. Also, family screening was performed in view of the fact that MEN-II is an autosomal dominant disease.

In the majority of situations, the interpretation and clinical decision based on the biopsy report are very straightforward. Patients found to have cancer are sent for appropriate surgery, and those with benign disease are followed or, in a case of Hashimoto's thyroiditis, placed on thyroid hormone. There are circumstances, however, in which additional considerations are necessary if the sensitivity and specificity of this procedure are to be maintained.

Obviously, adequate sampling of the nodule should be assured, and pathologists must refrain from making a diagnosis if inadequate tissue is presented. Critics of the procedure assert that differentiating follicular tumors, including Hürthle cell lesions, from carcinomas is impossible. While this is true in the majority of cases with these lesions, "follicular change" specimens comprise less than 10% of patient specimens. Assuming all these patients do have surgery, and since the incidence of follicular carcinoma is about 50% in these specimens, then only 5% or less of all patients will have had needless surgery. In some of these patients, the nodule will be found to be hot (extra nodular tissue suppressed) on thyroid scintiscans. Some will coincidentally be hyperthyroid or hypothyroid, and thus less likely to have follicular cancer of the thyroid. Observation or thyroid hormone suppression therapy may be indicated in some of these patients.

Another potential area for error is in the evaluation of the cystic thyroid nodule. About 20% of all patients with nodules have fluid-filled nodules, and while the incidence of cancer associated with these nodules is low, it is not zero. Surgical series have estimated the incidence of cancer in cystic nodules to be between 9% and 27%.[3] Clinicians therefore must resist the temptation to end the diagnostic workup with an ultrasound that shows a cystic nodule. Needle biopsy is the appropriate next test. It offers the advantage of providing not only a specific cause of the cyst, but also the drainage of the cyst during the same procedure, often providing psychological and cosmetic benefit to the patient.

Cystic nodules often present a challenge to the surgeon during the biopsy. While aspirating the cyst fluid is simple, assuring an adequate biopsy specimen is often difficult. Cyst fluid is usually acellular or contains necrotic material that is impossible to read cytologically. The operator must try to biopsy the cyst wall or perinodular areas to obtain sufficient material adequate for diagnosis. Often this is best accomplished by obtaining the biopsies before the cyst is collapsed and landmarks are lost. Patients are often alarmed when the cyst re-forms following successful aspiration. Generally, the larger the cyst, the more likely it is to re-form following aspiration. We have not found this phenomenon to be more peculiar to carcinomas, and we will sclerose these recurrent cysts with intralesional tetracycline if the initial biopsy is benign. Tetracycline sclerosis prevents cyst recurrence in over 75% of cases.[4]

Thyroid needle biopsy is painless, and some practitioners do not use local lidocaine anesthesia. To date, no significant adverse complications of the procedure have been reported. The procedure has also been shown to be cost-effective.[5] Indeed, in my own institution, the cost of the procedure and interpretation of slides rivals that of a thyroid scan. It is a wonder that more third party payers have not encouraged this procedure.

Excellent discrimination between benign and malignant thyroid nodules can be achieved by clinicians and pathologists dedicated to this procedure. While the techniques and interpretation of data are not esoteric, best results accrue to those with dedicated training and frequent experience.

One may ask what the relevant roles of thyroid scans and ultrasounds are in a decision tree analysis of thyroid nodules. There is no question that these procedures may be valuable in the evaluation or follow-up of patients, but I believe that there is also no question that needle biopsy of the thyroid has now assumed a primary role in this evaluation and should be the initial procedure after a patient is discovered to have a thyroid nodule.

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RESEARCH PAPER

Human immunodeficiency virus infection in a cohort of intravenous drug users in New York City

Demographic, behavioral, and clinical features

LAWRENCE S. BROWN, JR, MD, MPH; ALVIN CHU, MA; TOORU NEMOTO, PHD, MA;
DAVID AJULUCHUKWU, PHD, MPA; BENY J. PRIMM, MD

ABSTRACT. **In 1985, 454 intravenous drug users were recruited from among patients scheduled for physical examination in methadone treatment clinics in New York City. A questionnaire was administered, and serum was collected for human immunodeficiency virus (HIV) serology. The HIV seroinfection rate was 60.6%, with antibody and antigen detected in 58.4% and 4.3%, respectively, of the population. Nineteen percent of 307 subjects were in Group III or Group IV of the Centers for Disease Control (CDC) classification system for HIV-associated infections. Only behavioral factors ($p = 0.001$) and clinical indicators ($p = 0.003$) were found to distinguish the HIV-infected from the non-infected subjects. Frequent use of intravenous drugs ($p = 0.016$), duration of drug use ($p = 0.050$), and duration of drug treatment enrollment ($p = 0.038$) were significantly associated with HIV seroinfection status and CDC stage of HIV disease. These findings strongly support aggressive efforts to reduce parenteral drug use and enroll intravenous drug users into effective drug treatment programs.**

(NY State J Med 1989; 89:506–510)

Intravenous (IV) drug use continues to represent the second most frequent behavior associated with the prevalence of the acquired immunodeficiency syndrome (AIDS).[1] The prevalence of AIDS among women, children, and ethnic/racial minorities is also inextricably linked to the prevalence of IV drug use.[2–6] Nearly 70% of AIDS case reports, attributable to the Centers for Disease Control (CDC) heterosexual transmission category, are associated with a sex partner with a history of IV drug use.[7] Yet, despite the undeniable impact of IV drug use on HIV disease prevalence in the United States, significant gaps exist in knowledge about the behaviors of the intravenous drug user that lead to human immunodeficiency virus (HIV) transmission, the social setting in which IV drug use takes place, or the clinical picture of HIV infection in the IV drug using population. While there is growing awareness of the drug use patterns associated with HIV infection,[8–16] few studies explore the sexual practices of IV drug users that may also contribute to HIV transmission.[17,18] Even fewer reports are available on the clinical features of HIV disease in IV drug users prior to satisfying CDC criteria for AIDS. This study expands on this body of information by examining the prevalence of HIV infection and clinical HIV disease in association with demographic, socioeconomic, and behavioral factors in a population of IV drug users enrolled in a drug treatment program.

METHODS

Study Population. From June 1, 1985, to September 1, 1985, 454 study subjects (318 males and 136 females) were recruited from among patients scheduled for admission or annual physical examination in selected methadone maintenance clinics in the boroughs of Brooklyn and Manhattan of New York City. Minimal eligibility requirements for enrollment in these clinics were an age of 18 years and at least one year of opiate dependence. Pre-study information, counseling, and informed consent were obtained prior to the voluntary participation of each study subject, following a protocol approved by the institutional (human subject) review board.

The study subjects ranged in age from 19 to 62 years, with a mean age of 35 ± 0.35 (standard error of the mean, SEM) years and a median age of 33 years. The ethnic/racial composition of the study population was 48.9% black, 42.5% Hispanic, and 8.6% white. Forty-six percent of the study subjects were enrolled from Brooklyn clinics. The duration of drug use among this population ranged from one year to 38 years, with a mean and median of 12.3 ± 0.34 and 12 years, respectively. The duration of drug treatment enrollment ranged from 0.7 to 152 months, with a mean of 23 ± 1.4 months and a median of 13.8 months.

The study population represented 96% of the patients eligible for participation. Recruiting patients scheduled for admission or annual physical examination represented the most logistically practical method. This greatly limited the inconvenience to the patients or the interruption of drug treatment processes. Of those patients who were eligible but refused participation, the inconvenience of responding to the questionnaire was the major reason for refusal. Nonetheless, the study sample did not differ in any significant manner (based on demographic and drug use assessments) from those patients who refused participation, or the total

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From the Division of Evaluation and Medical Affairs, Addiction Research and Treatment Corporation, Brooklyn, NY; and the Department of Medicine, Harlem Hospital Center, College of Physicians and Surgeons, Columbia University (Dr Brown), New York, NY, and the Department of Health Education, School of Education, Health, Nursing, and Arts Professions, New York University (Dr Ajuluchukwu), New York, NY.

Address correspondence to Dr Brown, Division of Evaluation and Medical Affairs, Addiction Research and Treatment Corporation, 22 Chapel St, Brooklyn, NY 11201.

of 2,100 patients enrolled in these methadone maintenance clinics at the time of the study.

Questionnaire and Laboratory Measurements. A standardized questionnaire about demographic and socioeconomic status, drug use patterns, and sexual behaviors was administered by specially trained interviewers during one-hour face-to-face sessions. Drug use behaviors examined included types of drugs used, frequency of drug use, duration of drug use, etc. The current number of sexual partners, the frequency of sexual intercourse, and sexual preferences were among the sexual practices assessed with this questionnaire. Medical histories, physical examination, and phlebotomy were performed by medical staff.

Routine laboratory evaluation included a complete blood count, serum electrolytes, liver function tests, and urinalysis. Urine toxicology screening was performed by enzyme immunoassay and high performance thin layer chromatography, while hepatitis B serology was performed by the method of enzyme immunoassay (Abbott). Syphilis serology was assessed by the rapid plasma reagin method, and serum protein electrophoresis was performed by the microzone method. HIV-1 antibody was assessed by enzyme linked immunosorbent assay (ELISA) (Genetic Systems). Repeatedly positive ELISA specimens were confirmed by Western blot (Dupont). Sera with p24 and/or gp41 bands were considered positive. HIV antigen tests were performed as previously described by Allain and colleagues.[19] HIV seropositivity was defined as either a positive HIV antibody test confirmed by Western blot or a positive determination of HIV antigen.

Data Analysis. Unfortunately, limited resources prohibited analysis of data until recently. Using the SPSS/PC+ statistical package, univariate measures were used to analyze collected data. For 307 subjects, sufficient information was available to categorize them using a modified version of the CDC staging classification for HIV infections.[20] The one modification consisted of placing persons who were not found to have any clinical or serologic evidence of HIV infection in CDC Group 0.

RESULTS

Serologic evidence for HIV antibody and HIV antigen was found in 58.4% and 4.3%, respectively, of the 454 IV drug users participating in this study. When antibody and antigen results are combined, the HIV seroinfection rate was 60.6% for the entire study population. While the prevalence of HIV infection was greater among females, ethnic/racial minorities, subjects aged 25 to 30 years, and those who reside in Manhattan (Table I), these trends were not statistically significant. Subjects who had attained higher levels of education, who were married, who resided with their spouse and/or chil-

TABLE I. HIV Seroprevalence and Demographic Characteristics in Intravenous Drug Users in New York City, 1985

Characteristics	Number Tested	Seropositive No.	Seropositive Percent	Odds Ratio (95% Confidence Interval)
Sex				
Male	318	191	60	1.0
Female	136	84	62	1.1 (0.7–1.6)
Race				
White/other	39	21	54	1.0
Black	222	133	60	1.3 (0.6–2.5)
Hispanic	193	121	63	1.4 (0.7–2.9)
Age (yr)				
<25	26	14	54	1.0
25–30	104	68	65	1.6 (0.7–3.9)
31–35	156	93	60	1.3 (0.6–2.9)
36–40	92	59	64	1.5 (0.6–3.7)
>40	76	41	54	1.0 (0.4–2.5)
Marital status				
Married	173	91	53	1.0
Single	180	111	62	1.5 (0.9–2.2)
Divorced/separated/ widowed	101	73	72	2.3 (1.4–4.0)
Educational level				
Some college	53	29	55	1.0
High school graduate	109	64	59	1.2 (0.6–2.3)
Some high school	230	143	62	1.4 (0.7–2.5)
<8th grade	62	39	63	1.4 (0.7–3.0)
Borough of residence				
Brooklyn	209	118	56	1.0
Manhattan	245	157	64	1.4 (0.9–2.0)
Living status*				
With spouse and/or child	179	91	51	1.0
With self	98	59	60	1.5 (0.9–2.4)
With parent	61	40	65	1.8 (1.0–3.4)
Other	98	67	68	2.1 (1.2–3.5)
Source of income*				
Legitimate income	100	34	34	1.0
Illegitimate income	131	78	59	2.9 (1.7–4.9)
Public assistance	209	149	71	4.8 (2.9–8.0)
Employment status*				
Employed	100	55	55	1.0
Unemployed	332	198	60	1.2 (0.8–1.9)

* Missing information on some subjects.

TABLE II. HIV Seroprevalence and Behavioral Characteristics in Intravenous Drug Users in New York City, 1985

Clinical Features	Number Tested	Seropositive		Odds Ratio (95% Confidence Interval)
		No.	Percent	
IV route of administration most often used				
No	63	28	44	1.0
Yes	391	247	63	2.1 (1.3–3.7)
Drugs used				
Heroin only	20	8	40	1.0
Heroin and cocaine only	133	72	54	1.8 (0.7–1.5)
Heroin, cocaine, other	286	188	66	2.9 (1.1–7.3)
Other	14	7	50	1.5 (0.4–5.9)
Duration of drug use				
Less than 6 yrs	98	48	50	1.0
6 yr or more	356	227	64	1.8 (1.2–2.9)
Duration of drug treatment enrollment				
1 yr or more	259	146	56	1.0
Less than 1 yr	195	129	66	1.5 (1.0–2.2)
Ever had a homosexual relationship*				
No	396	234	59	1.0
Yes	56	39	70	1.6 (0.9–2.9)
Currently sexually active*				
Yes	413	248	60	1.0
No	40	26	65	1.2 (0.6–2.4)
Number of current steady sex partners*				
<2	369	223	60	1.0
2–4	77	47	61	1.0 (0.6–1.7)
>4	6	4	67	1.3 (0.2–7.3)

* Missing information on some subjects.

dren, and those who admitted to receiving income from legitimate employment were found to have lower rates of infection. As a group, the demographic and socioeconomic variables did not significantly differentiate seropositive subjects from those who were seronegative.

The HIV seroinfection distribution did not differ significantly by age of initiation or level of consumption of alcohol or tobacco. All subjects admitted to the use of intravenous drugs, and 92% admitted to polydrug use. However, those who responded that the IV route was their most frequent drug administration method were significantly ($p = 0.016$) more likely to be seropositive (Table II). HIV-infected subjects generally started the use of parenteral drugs at an earlier age, had a shorter cumulative period of drug treatment enrollment, and tended to be prescribed lower daily doses of methadone at the time of this study. Only the level of the daily methadone dose, the duration of drug use, and the cumulative duration of drug treatment enrollment reached statistical significance ($p = 0.001$, $p = 0.038$, and $p = 0.013$, respectively) in distinguishing seropositive from seronegative subjects. HIV-infected subjects were more likely to have had a homosexual relationship in the last five years and to admit to current sexual activity with a greater number of sex partners, although this was not statistically significant. Collectively, behavioral data significantly distinguished ($p = 0.007$) the HIV infected from the non-infected subjects.

A past medical history of tuberculosis, hepatitis, endocarditis, and oral candidiasis was uncovered in 7%, 26%, 4%, and 2% of the study subjects, respectively (Table III). The prevalence of HIV infection was not significantly associated with any of these diseases. HIV seropositive subjects were more likely (although without statistical significance) to volunteer symptoms of involuntary weight loss, frequent night sweats, persistent/chronic diarrhea, persistent fevers, malaise, anorexia, and dysphagia. HIV seropositive subjects, in contrast to seronegative subjects, had significantly lower total white blood cell counts ($p = 0.001$), hematocrits ($p = 0.006$), mean corpuscular volumes ($p = 0.018$), absolute platelet counts ($p = 0.018$), and lower serum albumin levels ($p = 0.001$). Serum globulin levels were significantly ($p = 0.001$) higher in HIV seropositive subjects. Results of liver function tests, hepatitis serology, and syphilis serology did not differ significantly between HIV seronegative and seropositive subjects.

Based on the clinical information available, 307 subjects were categorized according to the CDC classification system for HIV-associated infections.[20] However, no subject was classified in the Group I category. At the time of this investigation, the study design did not include the assessment of the prevalence of symptoms associated with acute HIV infection (the CDC Group I criteria). In comparison to their proportion of the study sample, female subjects were over-represented among those subjects in CDC Group III and Group IV (Table IV). Subjects in CDC Group III and Group IV were significantly more likely to use drugs parenterally as their most frequent route of drug administration ($p = 0.001$) and to admit to a homosexual relationship in the previous five years ($p = 0.003$). The mean daily methadone doses varied significantly ($p = 0.005$) among CDC HIV stages from 45.5 ± 2.3 mg (95% confidence interval of 40.9–50.1) for subjects in Group 0 to a mean dose of 34.3 ± 2.8 mg (95% confidence interval of 28.6–40.1) for subjects in Group IV. Similarly, the length of enrollment in the drug treatment program was significantly shorter among subjects in Group IV (mean of 11.7 ± 2.6 months) than among subjects classified in other CDC HIV categories.

Total white blood cell counts, serum hematocrits, absolute platelet counts, and serum albumin levels were significantly lower in Group III and Group IV subjects ($p = 0.002, 0.001, 0.018, 0.002$, respectively). Group 0 and Group II subjects had significantly lower serum globulin levels ($p = 0.001$). The prevalence of positive hepatitis or syphilis serology did not vary significantly between the subjects grouped by CDC classification for HIV infection status.

DISCUSSION

In this study population of IV drug users attending

TABLE III. HIV Seroprevalence and Clinical Features in Intravenous Drug Users in New York City, 1985

Clinical Features	Number Tested	Seropositive		Odds Ratio (95% Confidence Interval)
		No.	Percent	
Medical History of				
Tuberculosis				
No	421	255	60	1.0
Yes	33	20	61	1.0 (0.5–2.1)
Hepatitis				
No	335	200	60	1.0
Yes	119	75	63	1.2 (0.7–1.8)
Endocarditis				
Yes	18	10	55	1.0
No	436	265	61	1.2 (0.5–3.2)
HIV-associated symptoms*				
No	248	148	60	1.0
Yes	74	46	62	1.1 (0.7–1.9)
Lymphadenopathy†				
No	298	173	58	1.0
Yes	32	28	87	5.1 (1.7–14.8)

* One or more of involuntary weight loss, frequent or chronic diarrhea, or persistent fevers.

† Two or more noninguinal sites of lymph node enlargement by physical examination.

methadone maintenance clinics in New York City, the HIV seroinfection rate was found to be 60.6%. Admittedly, this population may not be entirely representative of the entire population of persons using intravenous drugs or of persons enrolled in various forms of drug treatment. Drug treatment programs serve a valuable role, and logistically represent the most practical points of access to sufficient numbers of IV drug users to make results from such investigations meaningful. Studies performed in New York City of IV drug users entering drug treatment demonstrated no appreciable difference in HIV infection as compared to those conducted among IV drug users outside of drug treatment.[21] Furthermore, in terms of demography and patterns of drug use, persons who participated were similar to those who refused participation. While other investigations have been conducted among IV drug users in drug treatment in New York City,[10-12,22] these current data are important in that they include serologic information about IV drug users in the borough of Brooklyn. In this study, IV drug users from Brooklyn experienced only a slightly lower HIV infection rate in comparison to the subjects in Manhattan.

Of the drug abuse patterns explored in this study, only the choice of the IV route as one's most frequent method of drug administration and longer cumulative periods of drug use were clear risks for HIV infection and an advanced stage of HIV disease. Previous investigations among IV drug using AIDS patients or among IV drug users in HIV serologic surveys have found needle-sharing,[8,9,12-16] frequency of injection,[8,9,12] duration of IV drug use,[21] injection setting,[23] and treatment modality[24] to be significant factors influencing HIV infection. Some of these factors were not addressed in the current study so that no conclusion can be reached about their association with HIV transmission in the current study population. Similarly, any additional differences between the findings of previously reported studies and the current investigation may well be explained by differences in study design.

The significant lower prevalence of HIV seroinfection among those subjects with longer periods of drug treatment enrollment supports previous reports[25,26] advancing drug treatment as at least an indirect method of reducing HIV transmission. In regard to methadone maintenance treatment, the association between lower doses of methadone and HIV seroinfection remains, even after time in drug treatment is statistically controlled. A clear therapeutic goal of methadone maintenance treatment is the achievement of a blocking dose of methadone necessary to thwart physiological and psychological signs of withdrawal. While the daily methadone dose necessary to accomplish this may vary from patient to patient, the prevailing view is that patients should be prescribed whatever dose is necessary to meet the therapeutic aim. Otherwise, inadequate low doses of methadone will be accompanied by illicit opiate use. To the extent to which lower methadone doses may not effectively discourage continued illicit drug use, rapid stabilization at higher methadone doses may be

TABLE IV. Gender and Racial Distribution by CDC HIV Classification* of New York City Intravenous Drug Users in 1985

CDC Stage	Gender		Race/Ethnicity			
	Male	Female	Black	Hispanic	White	Total
Group 0	55	21	37	31	8	76
Group II	121	50	77	80	14	171
Group III	12	11	14	8	1	23
Group IV	23	14	19	14	4	37

* Study design did not allow for the determination of subjects who satisfy CDC Group 1. Group 0 was added for subjects without any clinical or serologic evidence of HIV infection.

crucial if the full HIV transmission-associated benefits of drug treatment are to be gained.

As in previous studies, this study demonstrated a greater rate of HIV infectivity among black and Hispanic subjects. However, this finding did not reach statistical significance. Indeed, among demographic factors, race/ethnicity correlated least. The fact that black and Hispanic IV drug users in this study were over-represented among behavioral factors significantly associated with HIV seropositivity strongly suggests that environmental factors are more important than race/ethnicity regarding HIV transmission.

Interestingly, females in this study had a higher HIV infection rate than males. In comparison to their representation among those subjects for whom CDC staging information was available, the female IV drug users were disproportionately represented among persons with advanced stages of HIV disease. This gender-associated factor in HIV infection is consistent with the results of other studies of IV drug users in New York City showing either higher seroinfection rates among females or no significant differences between the sexes.[8,10,22] In another investigation conducted at this institution,[17] female IV drug users, as compared to their male counterparts, were more likely to become infected by two mechanisms: their own IV drug use, and sex with an IV drug user. This may, in part, be the mechanism underlying the role of any gender-related cofactor in HIV infection/disease among parenteral drug users.

While limited by its study design, this investigation added to the clinical information about HIV disease in IV drug users. Similar to studies among homosexual/bisexual men, this investigation demonstrated that HIV infection and advanced stages of HIV disease were significantly associated with leukopenia, anemia, thrombocytopenia, hypoalbuminemia, and hyperglobulinemia. Admittedly, it may be difficult to attribute these derangements entirely to HIV infection, as parenteral drug use has also been associated with many of these abnormalities long before the HIV epidemic began. Many more studies examining the clinical aspects of HIV infection in IV drug users are needed if we are to explain fully the shorter survival time experienced by IV drug users.[27] The categorization of subjects in this study using the CDC classification system for HIV infection[20] was particularly useful. The separate observations demonstrating associations between frequency of IV drug use and CDC clinical stage, and between duration of drug treatment enrollment and CDC clinical stage, may not necessarily suggest differences in ultimate HIV-associated mortality. Nonetheless, it is certainly prudent to advise IV drug users that continued parenteral drug use may increase their prospects for developing more advanced stages of HIV disease.

Finally, the inextricable correlation between intravenous drug use and HIV disease in New York City demands that continued research be directed at identifying cofactors for not only the acquisition of HIV infection but also its clinical progression. The continued success of health education efforts is particularly dependent on this evolving body of study. The public health significance of this direction of research is critical, as effective vaccine and therapeutic interventions are still in embryonic stages of development.

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Retroperitoneal fibrosis

A report of selected cases and a review of the literature

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Retroperitoneal fibrosis (RF) first gained acceptance as a clinical entity in 1948 when Ormond[1] described two patients with anemia, backache, and ureteral "compression by an inflammatory retroperitoneal process." Since then, more than 500 cases have appeared in the literature.[2] It has become clear that the term retroperitoneal fibrosis actually encompasses several different pathophysiologic entities that have as a common end result fibrosis in the retroperitoneal space. In approximately one third of patients, the etiology has now been established, but the majority of cases remain classified as idiopathic.[3] New imaging techniques have refined the diagnostic process, and immunosuppressive therapy has been accepted as both an adjunct and an alternative to surgery.

As the last comprehensive review of RF in the literature is now almost a decade old, we examined admissions to this community hospital to obtain a series of illustrative cases that could serve as a starting point for an updated review. These cases underscore the fact that retroperitoneal fibrosis is not an obscure disease encountered only in large university referral centers. As a potentially reversible cause of renal insufficiency, RF should be considered in the differential diagnosis of all patients who present with unexplained ureteral obstruction.

As most of the recent research has been on the idiopathic form of retroperitoneal fibrosis, this review will focus primarily on the etiologic, diagnostic, and therapeutic considerations of idiopathic RF.

Booth Memorial Medical Center is a university affiliated, community hospital to which there were approximately 160,000 admissions between 1978 and 1987. A chart review identified six cases of documented retroperitoneal fibrosis among these admissions. Table I summarizes the cases in this series. Below are three cases that introduce the concepts described in the review that follows.

Case 1. A 46-year-old man presented to another hospital in December 1985 for evaluation of abdominal pain radiating to both flanks and progressive renal failure. The patient had longstanding essential hypertension that was being treated with furosemide and clonidine, but he denied use of methysergide. Initial laboratory results were notable for a hemoglobin of 11 g/dL, a blood urea nitrogen of 88 mg/dL, and a creatinine of 7.4 mg/dL. Physical examination was normal. The patient's renal failure was felt to be secondary to hypertension, and he underwent dialysis. He was then referred to this hospital for further evaluation.

A renal ultrasound examination revealed right hydroureteronephrosis, and a retrograde pyelogram showed bilateral ureteral dilation, which was considered secondary to external compression (Fig 1). The patient underwent an abdominal computed tomographic (CT) scan that was notable for a periaortic soft tissue mass encasing both ureters. A laparotomy was performed with bilateral ureterolysis. Biopsy of the retroperitoneal mass was consistent with RF, and no evidence of malignancy was found. The patient's renal function improved after laparotomy; a serum creatinine of 1.7 mg/dL was obtained on discharge. He was placed on a short course of prednisone, 5 mg/day, and his condition remained stable. An 18-month follow-up uncovered no complications.

Case 2. A 58-year-old man presented in October 1987 with a one-month history of low back pain, weight loss, and malaise. He had had no previous medical problems, and his only medication was ibuprofen, prescribed because of back pain by an orthopedist who offered a consultation on the patient. Microscopic hematuria was noted on a routine urinalysis, and an intravenous pyelogram and cystoscopy performed as outpatient procedures were negative. On the patient's presentation to an internist, the only pertinent physical finding was a pulsatile abdominal mass. His laboratory results were normal except for persistent microscopic hematuria.

An abdominal CT scan revealed a 10-cm infrarenal aortic aneurysm extending beyond the iliac bifurcation inferiorly (Fig 2). A perianeurysmal soft tissue density was noted that appeared to surround and compress the left ureter. On laparotomy, the aneurysm was repaired, and left ureterolysis was performed. The patient had an uneventful postoperative course and remained well three months after discharge.

Case 3. A 62-year-old man presented in July 1987 with a history of several weeks of weight loss, malaise, and shortness of breath. He had a history of Type 1 diabetes

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TABLE I. Patients Diagnosed with Retroperitoneal Fibrosis, 1978–1987

Patient	Sex	Age (yr)	Etiology	Symptoms	Initial Serum Creatinine (mg/dL)	Therapy	Outcome
1	M	46	Idiopathic	Abdominal; flank pain	7.4	Steroids; surgery	Well 18 months after surgery
2	M	58	Aneurysm	Malaise; weight loss; back pain	1.2	Repair of aneurysm; ureterolysis	Well three months after surgery
3	M	62	Idiopathic	Malaise; weight loss; shortness of breath	16.6	Steroids	Well five months after diagnosis
4	M	59	Aneurysm	Malaise; weight loss; abdominal pain	22.0	Repair of aneurysm; ureterolysis; nephrectomy; steroids	Well seven years after surgery
5	M	73	Idiopathic	Dysuria; abdominal and flank pain	1.7	Ureterolysis	Unknown
6	M	83	Aneurysm	Abdominal and back pain	1.2	Repair of aneurysm; ureterolysis	Death from postoperative complications

mellitus and essential hypertension treated with insulin, prazocin, and hydrochlorothiazide. The patient denied a history of renal disease or methysergide use. Physical examination was normal, and routine hematologic tests were significant for a hemoglobin of 9.2 g/dL, a blood urea nitrogen of 83 mg/dL, a creatinine of 16.6 mg/dL, and an erythrocyte sedimentation rate of 122 mm/h.

Evaluation with renal ultrasound and retrograde pyelogram revealed bilateral hydronephrosis with medial deviation of the right ureter. Cystoscopy was performed, and bilateral ureteral catheters were placed without difficulty. Renal function gradually improved, and two weeks after admission serum creatinine was 1.6 mg/dL. An abdominal CT scan showed a well demarcated retroperitoneal lesion that enhanced with contrast (Fig 3). The patient declined biopsy or ureterolysis and was started on prednisone, 60 mg/day. His dose was slowly tapered to 20 mg/day, on which he was maintained for five months. The patient's systemic symptoms resolved, his renal function remained stable, and follow-up CT scan demonstrated that the mass had decreased in size (Fig 3).

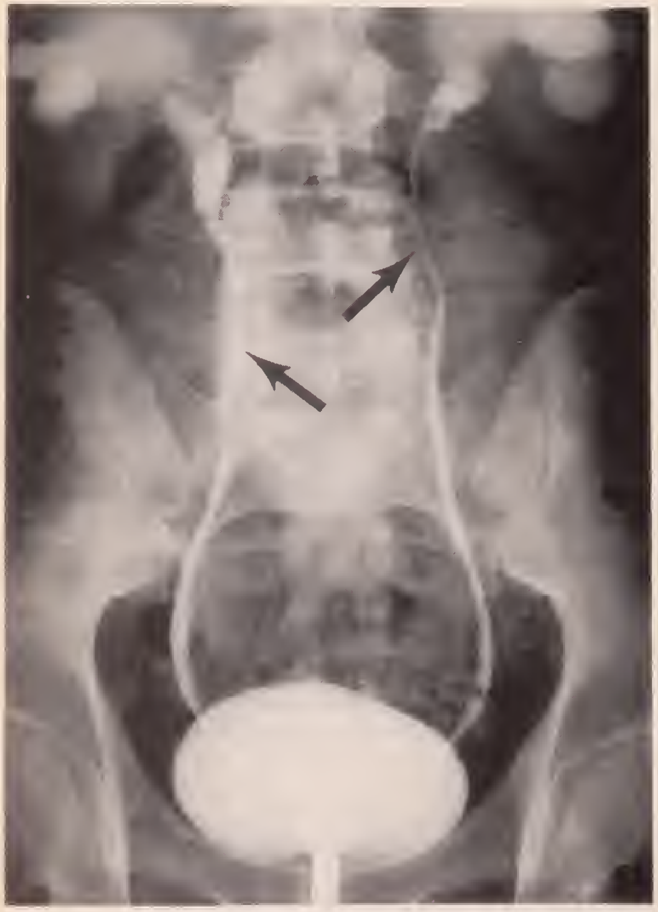

FIGURE 1. Retrograde pyelogram in a patient with idiopathic RF. Demonstrated is bilateral hydroureteronephrosis, with medial deviation and external compression of both proximal ureters (arrows).

REVIEW OF THE LITERATURE

Etiology. More than two thirds of patients with retroperitoneal fibrosis are considered to have idiopathic disease, as no certain etiologic factor can be identified.[3] Evidence has accumulated to support the concept that idiopathic RF is but one manifestation of a systemic inflammatory process. Between 8% and 15% of patients with RF have additional fibrosis outside of the retroperitoneum,[3,4] occasionally involving several organ systems.[5,6] Rare biopsy specimens reveal a systemic vasculitis,[7,8] and coexisting autoimmune disease with associated autoanti-

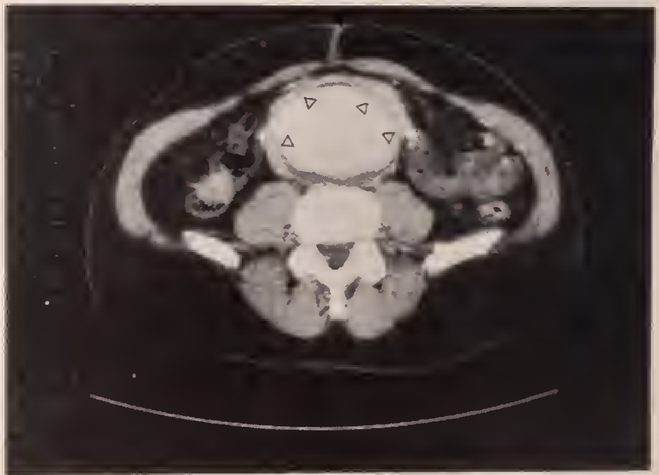

FIGURE 2. CT scan of the abdomen after intravenous contrast in a patient with an inflammatory aortic aneurysm. Demonstrated is an area of fibrosis surrounding the aneurysm (arrows) and compressing the right ureter.

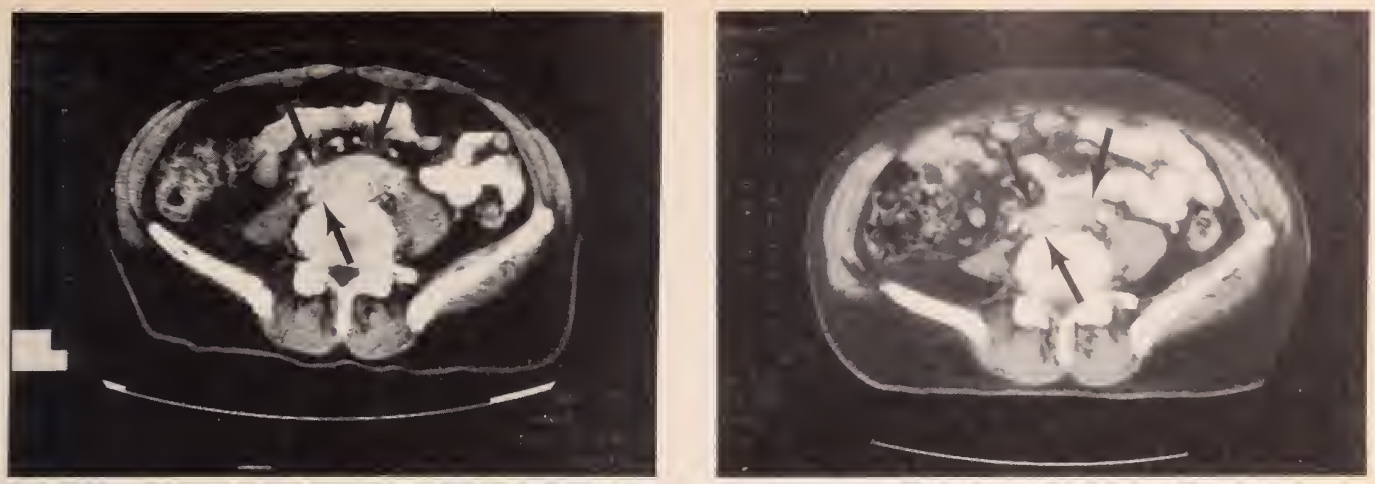

FIGURE 3. Left, CT scan of the abdomen after intravenous contrast in a patient with idiopathic RF prior to treatment. Demonstrated is a periaortic retroperitoneal mass obliterating the anterior margin of the aorta (arrows). The mass enhanced with contrast indicating fibrosis was in the active, cellular stage. Right, CT scan of the abdomen with contrast in the same patient after four months of corticosteroid therapy. Note that the fibrosis has decreased significantly in size (arrows).

bodies and circulating immune complexes have been reported.[9-16] A syndrome of retroperitoneal fibrosis, mediastinal fibrosis, sclerosing cholangitis, Reidel thyroiditis, and pseudotumor of the orbit has been described, termed familial multifocal fibrosclerosis.[4,17] But perhaps most suggestive of the inflammatory nature of this disease is the impressive response of the majority of patients to corticosteroid therapy.[18]

Recent observations by Mitchinson and his colleagues[19] in Great Britain have shed light on what may be the underlying immunologic mechanism for idiopathic RF. The fibrotic retroperitoneal mass usually surrounds the abdominal aorta, and fibrosis appears to occur only in areas where large atherosclerotic plaques have extended into an attenuated aortic media.[19] Antibodies to ceroid, a byproduct of lipoprotein oxidation that accumulates in plaques, have been isolated in patients with clinical and subclinical RF.[20] It has been proposed that when plaques breach the media, antigens such as ceroid are exposed to the circulation, antibodies are produced, and a periaortic inflammatory process is initiated.[21] Cause and effect between these antibodies and the development of RF has not yet been proven, but this theory could explain both the immunologic nature of idiopathic disease and the occurrence of "inflammatory" aortic aneurysms in many patients with RF. Between 5% and 23% of abdominal aortic aneurysms are associated with perianeurysmal fibrosis that is sometimes extensive enough to envelop local structures (Fig 1). These aneurysms can be associated with systemic signs and symptoms indistinguishable from those of idiopathic RF,[22,23] as in case 2 in this series.

Drug-induced disease occurs in 12% of patients with RF.[3] Several agents have been implicated (Table II), but firm data exist only for methysergide, an ergot preparation used in the treatment of migraine headache.[24] In 1964, Graham[25] discovered RF in two of 500 patients on methysergide, and more than 60 additional patients have since been described.[3] Dosages used are usually within the therapeutic range, and fibrosis has been diagnosed as soon as nine months after starting the drug.[26] In one study, 2.5% of asymptomatic patients on methysergide were noted to have abnormal screening intravenous pyelograms consistent with RF; when the drug was discontinued, repeat pyelograms were normal.[27]

The means by which methysergide causes fibrosis are not clear,[28] but the drug's vasospastic properties may be involved.[21] A recent dosage recommendation is to provide three- to four-week drug free intervals for every six months of methysergide therapy as a means of avoiding fibrotic complications.[24] This should result in fewer drug-induced cases of RF and may explain why there were no such cases in this series. Only isolated reports and anecdotal data are available for the other agents listed in Table II, and although suggestive, their widespread use might lead one to expect a much higher incidence of retroperitoneal fibrosis if they were indeed causative.

Malignancy has been identified in 8% of patients with RF.[3] No single form of cancer shows a strong association, and those implicated include solid tumors, Hodgkin disease, and lymphoma.[3,29,30] The remaining patients in whom a causative factor has been identified have had any one of a variety of disorders that can induce chronic retroperitoneal inflammation. These disorders include retroperitoneal hematomas from a bleeding diathesis[31] or trauma,[32] infection in the genitourinary tract or in the retroperitoneum, operations on the abdomen or pelvis,[3] and infusion into the retroperitoneum of sclerosing agents[33-35] or radiation.[32]

TABLE II. Drugs Implicated in the Development of Retroperitoneal Fibrosis

Ergot derivatives
  methysergide,[26] bromocriptine,[69] lysergic acid,[24] ergotamine[70]
Beta adrenergic blockers
  propranolol,[71] timolol,[72] sotalol,[73] oxyprenolol,[74] atenolol,[75] metoprolol,[76] pindolol[75]
Analgesics
  aspirin, phenacetin, codeine[77,78]
Antihypertensives
  alphamethyldopa,[79] hydralazine,[80] reserpine[81]
Miscellaneous
  haloperidol,[82] amphetamines[9,83]

Clinical Presentation. Patients with idiopathic retroperitoneal fibrosis usually present at mid-life, with 70% diagnosed between the ages of 31 and 60.[3,36] Overall, the incidence of idiopathic disease is more than two times higher in men than in women, while RF secondary to methysergide is more common in women.[3] The latter may be because migraine headache, the principal indication for the use of methysergide, is more common in women.[37]

The most frequent presenting complaint occurring in 90% of patients is dull, non-colicky pain localized to the back, flank, or abdomen. Other symptoms frequently encountered include weight loss, anorexia, nonspecific gastrointestinal complaints, and oliguria. Less common are edema (uni- or bilateral), gastrointestinal bleeding, malaise, and fever. The duration of symptoms prior to diagnosis is, on the average, four months.[3,36]

Physical signs referable to idiopathic RF are either uncommon or nonspecific. Abdominal fullness or a distinct mass are only found in 8% to 14% of patients,[36] and approximately 10% will have edema, hydrocele, or fever. Hypertension is frequently noted, and a recent review found that 71% of patients had a diastolic pressure of at least 90 mm Hg, while 53% had a diastolic pressure of at least 100 mm Hg.[38] If the fibrotic process is systemic, signs related to the involved sites may be present (ie, pericardial friction rub, thyroid enlargement).[3,36]

Routine laboratory tests may reveal any number of abnormalities. In most early reviews, 50% to 60% of patients had an elevated blood urea nitrogen level (greater than 30 mg/dL) on presentation,[39,40] but this appears to be less common today, as more patients are diagnosed before renal insufficiency develops.[3] Anemia may be present, and hematocrits below 33% are common. Patients may have an elevated erythrocyte sedimentation rate (ESR), elevated gamma globulin fraction, or positive autoantibody screen. Occasionally, pyuria or a positive urine culture may be noted, the significance of which is unclear.[3,36] If the biliary tree is involved, bilirubin and alkaline phosphatase levels may be elevated.[41,42] In the presence of mediastinal fibrosis or inflammatory aneurysms, routine radiographs may be abnormal.[43,44]

Diagnosis. Intravenous pyelography (IVP) has been the most common test utilized to confirm a clinical diagnosis of RF. The triad of medial deviation of the middle one third of the ureter, hydroureteronephrosis, and extrinsic ureteral compression has been considered virtually pathognomonic (Fig 2).[45,46] In major reviews, more than 90% of patients with RF have abnormal IVPs, with bilateral hydroureteronephrosis occurring in more than two thirds.[3,36] However, some patients will have normal or inconclusive IVP results, and contrast studies can be hazardous in RF due to renal insufficiency. Additionally, medial deviation of the ureter has now been shown to be a normal variant.[47] The diagnostic utility of pyelography has been questioned, and more sophisticated tests are now available.

With the advent of ultrasonography and computed tomographic scanning, much more detailed and reliable data can be obtained. Ultrasonography reveals a homogeneous, anechoic retroperitoneal lesion[45] and can demonstrate hydronephrosis if it is present. On CT scan, the mass appears smooth and clearly delineated, sometimes encasing the large vessels and ureters, and has a density similar to that of muscle (Fig 3).[47] Ongoing inflammation in the lesion may lead to contrast enhancement on CT,[48] as well as gallium uptake on nuclear scanning.[49] Despite the sophistication of these imaging techniques, it can still be difficult to differentiate benign RF from that secondary to malignant disease; displacement of the major vessels[50] and psoas muscle[39] may be suggestive of malignancy.

Once noninvasive tests are performed, laparotomy is required to establish the diagnosis, evaluate for the presence of malignant disease, and provide therapy.[51,52] Multiple deep biopsies should be taken, as malignancy can be difficult to localize[3,32] and has been discovered after operative procedures had indicated benign disease.[5,53,54] A possible exception to the need for biopsy is in those patients with methysergide-related RF. These patients can be followed closely while off the drug, with surgery necessary only if progression of the mass or persistent symptoms are noted.[2,26,55]

Pathology. On gross examination, the fibrotic mass appears grayish white and hard. It surrounds the lower abdominal aorta and commonly follows the course of the aorta beyond the common iliac bifurcation.[4,36] Fibrosis rarely extends below the pelvic rim inferiorly, but may extend superiorly into the mediastinum. Local structures can be encased in the mass, and bowel may be adherent.[4]

On microscopic analysis, the tissue appears as a spectrum of inflammation.[3] Specimens obtained early in the fibrotic process reveal collagen bundles with a large number of inflammatory cells, predominantly lymphocytes, plasma cells, and fibroblasts (Fig 4). Small blood vessels are numerous, as is mucopolysaccharide substrate. Specimens obtained later or after steroid therapy appear relatively acellular and avascular, with deposits of calcium within collagen bundles.[3,4] The histology of mediastinal and perianeurysmal fibrosis is similar in structure and cell type.[18,19] Because of the consistent histology and distribution of inflammation, "chronic periaortitis" has been proposed as a more accurate descriptive term for RF and its associated manifestations.[18]

As stated previously, all biopsy specimens must be ex-

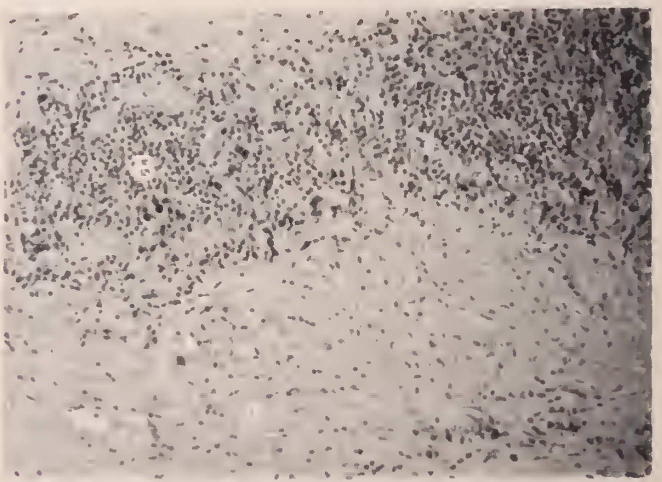

FIGURE 4. Biopsy specimen demonstrates dense fibrosis with bands of collagen (marked A) surrounding aggregates of inflammatory cells (marked B). The inflammatory cells are predominantly lymphocytes, with an occasional plasma cell (hematoxylin-eosin stain; original magnification X 300).

amined carefully and thoroughly for the presence of malignancy.[3,32]

Treatment. The therapeutic approach to retroperitoneal fibrosis includes the removal of any identifiable inciting agents, surgical relief of ureteral obstruction, and suppression of the inflammatory process. If a patient has been using methysergide, or possibly one of several other agents (Table II), the drug should be discontinued. The majority of these patients will do well, but some (eight of 19 in one series) will still require surgical intervention to treat obstructive complications.[26,55]

The goal of surgery is to make an accurate diagnosis and to protect retroperitoneal structures from fibrotic encasement. In the presence of significant ureteral obstruction, emergency decompression can be accomplished with either placement of ureteral catheters or percutaneous nephrostomy. When renal function is stable, therapeutic and diagnostic laparotomy is the recommended form of treatment. The surgical procedure involves freeing the ureters from entrapment in the fibrotic mass and placing them either inside the peritoneal cavity or away from the area of inflammation.[55] Some believe that the procedure of choice is to wrap the newly freed ureters in omentum, as this may prevent recurrence of obstruction.[56] Surgery successfully improves renal function in more than 90% of cases.[55-57] Relief of systemic signs and symptoms due to RF has occasionally been noted with surgical therapy alone, possibly because of stress-related increases in endogenous corticosteroid secretion.[36]

The rationale for immunosuppressive therapy in idiopathic RF is that while surgery treats a complication, it does not affect the underlying inflammatory nature of the disease. Recurrence of ureteral obstruction after surgery on the ipsilateral or contralateral side can occur in as many as one third of patients,[58] and other retroperitoneal structures that cannot be surgically displaced (ie, great vessels and nerves) are still at risk for entrapment.[13,51] Additionally, some patients are simply too ill to undergo a major intra-abdominal procedure, or the initial surgery may not be successful.

Ross and Tinkler[59] were the first to use corticosteroids as an adjunct to surgery, and experience accumulated since then has served to clarify the role of immunosuppression. Resolution of ureteral obstruction without surgery and recurrence of obstruction after surgery have been documented by intravenous pyelogram and computed tomography in patients treated with steroids.[2,60-62] Jones et al[63] treated six patients with steroids after surgery, noting no recurrences, a rise in hemoglobin, and a decrease in ESR. Elimination of autoantibodies has also been seen.[64] Azothioprine, administered either alone or in combination with corticosteroids, has been successful in two patients with RF and associated collagen vascular diseases.[11,65] It is important to note that not all patients will respond to steroid therapy, and immunosuppression appears to be most effective early on in the course of the disease, when inflammation is in the cellular stage.[55,60] Recommended situations in which the administration of corticosteroids can be considered include the following:

- postoperatively, especially if there is a good degree of retroperitoneal fibrosis, gastrointestinal involvement, or systemic disease.
- postoperatively, for ipsilateral or contralateral recurrences.
- preoperatively, in combination with ureteral stents or nephrostomy, for patients with non-malignancy-related RF and poor renal function (a group of patients in whom improvement of renal function after surgery is less likely); surgery can be performed later if renal function improves or the fibrotic mass does not respond.
- for patients who are too ill to undergo surgery, and in whom all non-invasive tests show no evidence of malignancy.[51,60,66]

The dose and duration of corticosteroids vary considerably and are empiric.[51] In general, high initial doses are used (40–100 mg daily of prednisone or its equivalent) and continued for a period of weeks to months, after which they are tapered to small maintenance doses or are discontinued.[6,51,60,63,64] Renal function, ESR, and imaging studies are followed, as the response to immunosuppression is variable,[60] and patients need to be observed closely for signs of infection. For those patients who undergo relapse after therapy is completed, isolated case reports indicate that immunosuppression can be reinstituted in the form of corticosteroids,[52] azothioprine,[13] or a combination of the two.[11]

Prognosis. With accurate diagnosis and aggressive intervention, the prognosis for idiopathic disease appears to be good. Data are limited and length of follow-up varies, but cumulative mortality rates of from 0% to 14% over several years have been reported.[2,3,67] The prognosis for disease secondary to methysergide is similarly good,[36,68] but case reports of malignancy-related RF indicate that most of these patients succumb within months of diagnosis.[6,53] Studies of patients with inflammatory aneurysms note for the most part a favorable outcome, with 80% surviving a mean of 28 months after surgery.[22]

CONCLUSIONS

Although retroperitoneal fibrosis is considered a rare cause of renal failure, it is occasionally encountered in clinical practice. We have reviewed six cases of RF diagnosed at a community hospital during the last ten years. Through awareness of its presentation and routine application of sophisticated imaging techniques, the diagnosis of RF should become more common and can be made at an earlier stage than in the past. Previous reviews as well as this series indicate that prompt recognition can provide a good prospect for full recovery. Recent developments have added to our understanding of the pathophysiology of retroperitoneal fibrosis and justify the use of corticosteroid therapy as an adjunct to surgery.

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Current challenges of AIDS in New York City

Stephen C. Joseph, MD, MPH

As the official responsible for acquired immunodeficiency syndrome (AIDS) public health policy and implementation, I am constantly reminded that the key to the future of the epidemic is the intravenous drug user, and that the future has already arrived in New York City. The urgency of the AIDS epidemic in New York City leaves us little room for taking comfort in abstraction, or refuge in speculation over what might have been, as we consider the epidemic's prognosis.

As of the end of July 1989, more than 21,100 cases of AIDS had been reported in New York City, with almost 12,000 cumulative deaths. Close to 2,700 women and 487 children had been diagnosed with AIDS. Blacks and Hispanics made up more than half of all people with AIDS in New York City, but 84% of women with the disease and over 90% of the children.

Some 200,000 New Yorkers are estimated to be infected with the human immunodeficiency virus (HIV). AIDS is the leading cause of death among New York men aged 25–44 years and women aged 25–34 years.

By 1991, we project that more than 43,000 people will have developed AIDS in New York City; 32,000 people will have died. If we counted the addicts who are sick and dying from tuberculosis and other diseases related to HIV infection but not officially classified as AIDS, projections would be as much as 150% higher for addicts.

Against this background, what are the important developments in the long term of the epidemic in New York City? Our success in meeting the challenges of AIDS depends upon our answers to this question. Let me suggest three.

EPIDEMIOLOGIC DEVELOPMENTS

First is the changing epidemiologic profile of the epidemic, marked by the increasingly important role of the IV drug user. Before the end of 1985, reported cases here were predominately among men who had sex with men (58%, in contrast to 29% of cases among IV drug users). From the beginning of 1986 on, the overall percentage of cases reported among men having sex with men has dropped to 47%, while the percentage of cases among IV drug users has risen to 37%. Among men, before January 1986, 64% of cases were among men who had sex with men, compared to 26% among male IV drug users; from January 1986 on, 55% of cases were among men who had sex with men, compared to 34% among male IV drug users. Women represented 9% of the earliest cases, and 14% of the most recent cases.[1]

While 56% of the people with AIDS reported prior to 1986 resided in Manhattan, 55% of the people reported with AIDS since the beginning of 1986 have lived in the other boroughs, primarily Brooklyn and the Bronx.[1]

In case reporting of Centers for Disease Control (CDC)-defined AIDS, the incidence of AIDS among IV drug users now approaches the same incidence as among men who have sex with men (Fig 1). Indeed, in the first three months of 1988, the number of AIDS cases reported among IV drug users in New York City surpassed the number of cases among gay men in quarter-by-quarter analysis for the first time.

The change in case definition of September 1, 1987, has had a greater impact on reported cases among IV drug users than any other group.[1] Thirty percent of cases among IV drug users are now identified using the new case definition. In addition, a large number of undercounted cases of HIV-related disease is responsible for an approximately 100% increase in severe morbidity among IV drug users.

The trend toward HIV infection among substance

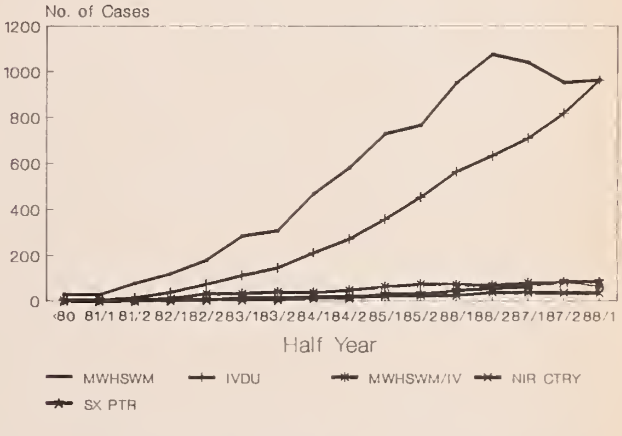

FIGURE 1. AIDS cases by risk by half year of diagnosis since 1980. Lag time between diagnosis and report to the Department of Health is usually less than six months, but may be as long as 24 months; therefore, the more recent data are less likely to be complete (from the NYC-DOH AIDS and HIV Surveillance Unit, 12/88).

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abusers is not confined to IV drug users. Crack and the hypersexuality associated with it has lead to increases in genital ulcer disease, HIV transmission, and heterosexual HIV infection. I scarcely need point out that we lack effectively proven mass-applicable treatment modalities for cocaine addiction.

While the gay community has made progress in reducing the spread of new infection, among IV drug users the virus continues to spread virtually unabated. This holds a number of implications for the epidemic's continuing, disproportionate impact upon minorities, and for heterosexual transmission. The epidemic of AIDS and other severe HIV-related morbidity and mortality is focusing most severely and dramatically upon IV drug users, their sex partners, their children, and the neighborhoods of heavy IV drug use, which are overwhelmingly poor and minority, with a large pool of HIV-infected people who may not be aware of their risk. The implications for heterosexual transmission are grim, in that black or Hispanic heterosexual men comprise the majority of IV drug users in this city. Neither minority communities nor we in public health can ignore the fact that AIDS is tracked straight at minorities.

CHANGES IN PREVENTIVE AND THERAPEUTIC RESPONSES

The second important development is the changing nature of preventive and therapeutic responses to the epidemic. The therapeutic horizon for clinical, preventive, and social services is rapidly broadening. The shift from the increasingly less relevant "AIDS" label to the broader spectrum of HIV infection puts more emphasis on the treatment of earlier forms of HIV illness and even asymptomatic infection.

Many major implications follow from this. A different, more complex mix of resource requirements has emerged over the past year. As more therapies become available for treating and preventing opportunistic infections, early diagnosis of HIV infection and early access and entry into treatment benefit the infected patient.

There are important implications of this for treatment services. There will be increased pressures on ambulatory care, increased need for acute care beds, and increased demand for associated social services.

This is altering the view of HIV antibody testing. We are now in a period when we should be actively case finding for HIV infection for prevention and prolongation of life. We must aggressively continue with counseling and testing, but with even more determined efforts to keep testing voluntary, confidential, anonymous, and counseling-based. The best defense against coercive testing and the enforced registration of seropositives is a program of voluntary testing at all clinical points: in hospitals, outpatient departments, physicians' offices, sexually transmitted disease and tuberculosis clinics, and family planning and abortion clinics. We must have the cooperation of medical practitioners, and we must have strong legal confidentiality and antidiscrimination protections.

Another implication of the shift from AIDS to HIV infection is an increase in the traditional public health approaches to prevention. This will mean greatly increased programs of partner notification. Again, our challenge will be to preserve our noncoercive stance.

POLITICS OF CONFLICT

The third development concerns the politics of conflict. We must not think that we have passed the era of bitter fights, such as the 1985 court battle over the rights of children with AIDS to attend school. New and more volatile issues lie ahead, based on the changing face of the epidemic.

For example, much unhappiness and sexual politics have influenced the reaction to our reduction in the estimates of HIV-infected New Yorkers, from 400,000 to 200,000. Though we revised estimates for several risk categories, the most substantial revision is the reduction of the estimate of the number of HIV-infected men who have sex with men. Some groups have overreacted to one or another set of data, but we have performed our epidemiology and research without responding to political influence of any sort, and we will continue to do so. It is of capital importance that we not do epidemiology by negotiation or according to public pleasure.

NEEDLE EXCHANGE PILOT

The pilot needle exchange program has also engendered a great deal of controversy. On one hand, public debate around this kind of issue is welcome, because it helps us define our direction as a society. On the other hand, we come under enormous pressure to let public perceptions, not science, form the underpinnings of public health. This makes the support of medical authorities all the more crucial for us.

The needle exchange pilot is part of a mix of educational, therapeutic, and law enforcement methods to break the link between IV drug use and AIDS. Certainly, greatly increased availability of drug treatment programs must be our first line of defense. Existing treating programs can accommodate only 35,000 of the city's 200,000 IV drug users, and waiting lists can be months long. I have called loudly and often for the rapid and long-overdue expansion of treatment programs. It will take federal and state money, and the willingness of communities to accept treatment programs in their midst.

But even if the capacity of treatment programs were tripled in the next few years to 100,000 (which is not likely to happen), and even if treatment were always successful (which, of course, it is not), there would still be some 100,000 addicts outside of treatment, whether due to lack of openings or unwillingness to enter treatment.

The inexorable progress of AIDS in our city and the all-too-familiar numbers, as well as the unacceptable impact of AIDS on minority women and children who are infected through drug use, forced us carefully to consider the merits, and honestly to weigh the risks, of a pilot needle exchange study.

Conducting a small, carefully controlled study involving one-tenth of one percent of the city's IV drug users, as we are doing, will have a negligible effect on the day-to-day dynamics of drug abuse here. Preliminary data from European needle exchange studies indicate that they have helped slow the spread of AIDS virus infection among IV drug users. Research does suggest that drug users would try to avoid getting infected by using clean needles if these were available.

As of the beginning of August 1989, more than 200 participants receive needles and syringes, with drug counseling and intensive education on how to change the sex and drug behavior that puts them at risk of getting or giving the AIDS virus. Those who take part must return their used injection equipment before they can receive more. Special efforts are being made to place participants into drug treatment programs.

Participants are IV drug users 18 years of age or older who are awaiting placement in drug rehabilitation programs throughout the city. The pilot program will determine whether providing clean needles and syringes, with constant reminders not to share drug injection equipment, can motivate addicts to stay on treatment waiting lists until an opening appears (currently, only half of all IV drug users on drug treatment waiting lists actually enter treatment; the rest lose motivation to quit injecting drugs before a treatment slot becomes available), and help addicts reduce the needle sharing and sexual behavior that spreads AIDS. Participants who do not regularly return the uniquely labeled, used needles that they were given at their previous visit are not allowed to receive new equipment and remain in the study.

Perhaps the most important piece of information that has emerged from the needle exchange program thus far is its proven effectiveness as a supportive bridge to treatment. We initially expected the linkage of clients to drug treatment programs to be a single, albeit important, element in a larger spectrum of services. We have found so far that the primary value of the pilot program for clients lies in drug counseling and referral to treatment. If the program's main effect is to speed IV drug users into treatment programs, then this will have been an important and successful demonstration.

Now that it has shown its value as a supportive bridge to treatment, our next priority must be to expand out of the single site, the Department of Health's administrative offices. We are currently exploring possible additional sites to increase the program's accessibility to addicts.

Two of the most often voiced concerns about this trial have been that a needle exchange trial would give addicts the message that the city encourages drug use, and that such a trial program could in some way be construed as racial genocide.

If anything, I believe the message that this program gives addicts is that New York City cares about reducing the spread of AIDS among all of its citizens. Research has not shown an increase in drug use in areas where needle exchange programs are in effect. Experts across the country have agreed that the time has come for a trial needle exchange study.

As to the second objection, that this pilot is racially biased, the answer must be, absolutely not. Like all attempts to stop or slow the spread of AIDS, this pilot study is directed against the *behavior* that spreads the virus, not against *people*. We must not confuse a public health research investigation aimed at a problem that disproportionately affects a particular population with an effort directed against that population.

No single strategy will be effective against the complex problem of HIV infection among IV drug users. Although the state is responsible for drug treatment programs, the city has taken a number of steps to increase drug treatment slots, and will continue to expand other approaches, including more and better education and direct street worker outreach to IV drug users and their sex partners in the neighborhoods and on the streets where drug use is rampant; frank television, print, and radio ads that bring the AIDS prevention message to drug abusers and their partners, heterosexuals, and adolescents; a network of voluntary, anonymous risk reduction counseling and counseling-based HIV antibody testing sites; and now a pilot needle exchange study.

We must reevaluate our concept of substance abuse treatment. Historically, substance abuse treatment programs have been isolated from other health care services. AIDS, the Great Educator, shows us that drug treatment must be a part of a primary care network that takes into account the connections between AIDS and HIV infection and a range of morbidity, including sexually transmitted diseases and tuberculosis, as well as the increasingly important reproductive health care for women at risk through IV drug use. These connections present us with treatment options and prevention opportunities that we must not lose, and underscore the importance of integrating drug treatment with clinical care programs and services.

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A short history of the quack's materia medica

JOHN S. HALLER, JR, PHD

From the 17th century through the early decades of the 20th, readers found it impossible to pick up a newspaper or periodical, either literary or scientific, artistic or religious, without finding considerable space advertising quack medicines. These advertisements were highly uniform: claiming some remarkable discovery, providing a long list of endorsements or testimonials from fictitious or influential persons with impressive degrees and credentials, promising unfailing success under all conditions, accusing medical orthodoxy of attempting to stifle sale or use, and selling under an attractive, euphonious, and original name. In addition, the ads almost always referred to the small quantity necessary to effect results, the saving of doctors' fees, and the great variety of diseases which the quack medicines cured.[1(p99),2(pp4-5)]

These products, known popularly as "secret medicines," "nostrums," or "patent" or "proprietary" medicines, were manufactured as elixirs, syrups, tablets, and pills. Manufacturers gave these medicines catchy names ending in "-ia," "-ine," "-ites," or other terminal syllables intended to impress upon buyers the idea that they were derived from the active principles of plants or chemicals. Reading that preparations such as Bromidia, Febriline, Maltine, Hypophosphites, Celerina, and Lactopeptine were endorsed by members of the medical profession, users found themselves justified in thinking they were saving the price of the doctor's visit.

ORIGINS

Since antiquity, manufacturers, chemists, and physicians have prepared and advertised secret preparations promising to cure man's ills, for a high price. From the *Terra Sigillata* tablets (Lemnian clay) to Epsom salts and ague drops, drug manufacturers exerted a steady influence on the practice of medicine and pharmacy. As this market grew, European governments stepped in to protect the rights and rewards of discovery and reap some of the revenues. By definition, *nostrums* were medicines whose composition was kept secret from the public. A *proprietary* medicine, such as Beecham's Pills, was one to which a proper possessive title was added. Finally, a *patent* medicine was sold under the protection of a Royal Letters Patent or Patent Letters, and derived from the time when the English monarch gave an individual the right to be the exclusive manufacturer or seller of a specific article of trade.[1(p96)]

Following the American Revolutionary War and the resulting financial strain to the British Crown, a coalition ministry of Fox and North, with Lord John Cavendish as Chancellor of the Exchequer, brought "quack medicines" under direct taxation. The Act (23 Geo. III, c. 62), passed in 1783 and amended (25 Geo. III, c. 79) in 1785, divided "quack medicines" into two categories: medicines sold under the king's patent and those sold as proprietary medicines or nostrums. The first Act granted the Crown the right to place a stamp duty upon all licenses to manufacture and sell medicines. Two years later, George III signed a second Act which stated:

> Any person whatsoever who has, or claims to have, any secret art or sole right of compounding preparations of drugs, and advertising and recommending the same as specifics for the cure or relief of any complaint or malady, shall affix a Government stamp to the vials, vessels, or inclosures containing them.[3(p80)]

These two acts, and those which followed, excluded from the tax those medicines prepared and sold by chemists following doctors' prescriptions.

By strict definition, a patented medicine could not be a secret medicine. Etymologically, "patent" derives from the Latin *patere*, meaning "to be open." Thus, a patented medicine was an open and nonsecret medicine; the inventor had to disclose both the ingredients and the process of manufacture, which became public property after a set period of time (17 years in the United States). Nevertheless, the etymology of the word and its actual use from the 17th century on suggest a substantially different meaning. In effect, the manufacturers of sarsaparillas, perunas, tonics, pain killers, blood purifiers, favorite prescriptions, and golden nervenes refused to disclose the ingredients of their patented medicines because of the limited period of protection. Instead, manufacturers chose to secure a more exclusive property right to their products by registering the names as trademarks. In this manner, the trademarked nostrum remained a monopoly in perpetuity rather than becoming public property.[4(pp1675-1676)]

In the United States, general patent acts were passed in 1790 and followed the pattern set by the British. These laws remained in force until 1874 when Congress rescinded the practice of copyrighting labels for patent medicines and provided instead for registering such labels in the Pa-

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tent Office.[3(pp80-81)] In 1883, the British Parliament passed an Act (46–47 Vict., c. 57) for the regulation of patents, designs and trademarks. This Act, built upon earlier Parliamentary restrictions, stated that for a medicine to be declared a patent preparation, it should be an original invention, with its complete description or secret made known. It should be obtainable by the public, and useful patents should cease after a specified number of years. Under the Adulteration of Food and Drugs Act (38–39 Vict., c. 65), manufacturers of drugs which were not prepared in this manner were liable to prosecution. As a result, there were actually few genuine patented medicines; instead, manufacturers continued to introduce a great number of secret or proprietary medicines erroneously called patent medicines.[1(p97)]

Of those medicines patented after 1812, most passed out of existence quickly—a strange fate for substances that promised to cure most of mankind's ills. Remedies such as the Friend to Man, Schults' Vegetable Acid Air, Ward's Liquid Sweat, Solomon's Balm of Gilead, and the Vital Balm seemed not to live up to their claims. By 1845, one critic of quack medicines guessed that some 20,000 different remedies had been manufactured and sold over a period of just 50 years. "And each one," he observed, "has been proclaimed by extravagant certificates, and pompous assertions of thousands cured, to be *the* infallible remedy for some disease, or more generally for *all* diseases." Ironically, "every one, although infallible, has disappeared from the light of *trial*, like gaseous meteors from the morning sun, in less than a dozen years from the time of its fabrication."[5(p9)]

By the 1890s, in a study of 100 consecutive sales by a large wholesale druggist, H. C. Wood, MD (1841-1920), of the University of Pennsylvania, discovered that 58% of the orders were for patented and proprietary articles, 6% for pharmaceutical preparations, 1% for packaged goods, and the remainder for crude drugs. A three-month purchase pattern by five druggists who bought supplies from one wholesale druggist indicated similar statistics. Nearly 64% of the purchases consisted of patented and proprietary drugs, 1% packaged goods, and 35% legitimate pharmaceutical preparations and crude drugs. For Wood, this indicated that only 40% of the sales of the retail druggist were connected with legitimate pharmacy. This figure was down nearly 80% from the earlier part of the century.[6(p908)]

Even more troublesome to Wood was his estimate that physicians were involved in 10% of the patent and proprietary trade—either by providing testimonials or through prescriptions. "We, as members of the medical profession, and especially those of us connected with the medical press," he wrote, "are accustomed to inveigh against newspapers, secular and religious, for accepting the advertisements of patent and proprietary medicines, whereas in truth, unless the pot be warranted in calling the kettle black, we ought to keep silence with shamefacedness." He found these doctors not so much corrupt as "disgustingly weak," having sold to some vendor a statement or certificate of support. "So far as I can remember," recalled Wood, "I never gave but one certificate concerning a drug or a commercial article, and so far as I know no greater scoundrel ever misused a certificate than did the one to whom I gave this lonely representative of personal weakness."[6(p909)]

FIGURE 1. Chromolithographed advertising card given out by druggists (189_?) for Lydia Pinkham's Vegetable Compound (from the National Library of Medicine, Bethesda, Md).

Interestingly, an 1899 study of advertising circulars confirmed that most of the certificates supporting these remedies came from physicians, professors, and graduates of so-called "mushroom" medical schools. None came from the University of Michigan, Harvard, Yale, University of Pennsylvania, or the Johns Hopkins University. The study also showed that 128 editorials supporting patent and proprietary articles were "found largely in corrupt medical journals."[7(pp169-170)]

Curiously, some of the most enthusiastic advocates of sectarian systems and patent medicines were clergy whose names graced published recommendations in religious newspapers, magazines, booklets, circulars, and handbills.[8] "I have often attended the conferences of the Southern Methodist Church of Kentucky," remarked S. J. Harris, MD, "and I have actually seen proprietary and patent medicines all around the steps, piled up to such an extent that one could hardly get in or go around without stepping on them."[9(p275)]

THE SYSTEM BUILDERS

Just as orthodox medicine faced a continuous assault from homeopaths, the botanico-medical Thomsonians, eclectics, and other sectarian system-builders, so, too, medicine faced a challenge from quackish claims to replace the materia medica with new "secret" formulae.

Perhaps the most famous of the early system nostrums originated with Mithridates VI, King of Pontus (132–63 BC), who reputedly compounded 54 substances into a universal antidote called mithridate. Brought to Rome, it was made popular by Galen of Pergamon who passed it on under the name theriaca, containing 64 ingredients beyond its opium base. Eventually produced in many different countries, theriaca remained a popular panacea well into the late 18th century. Based on the philosophical speculations of humoral pathology, the nostrum was formulated according to the body's affinities for the humors and elements.[10(pp39–43)]

The so-called Morisonian system of "vegetable universal medicines" was another case in point. James Morison of Bognie, Aberdeenshire, began his career as a broker of medical articles in Russia and the West Indies. Later, settling in Bordeaux, he proceeded to establish a therapeutic regime consisting mainly of cream of tartar and aloe which he sold on "puffs, . . . pamphlets, and advertisements." His popular #1 pills contained equal parts of aloe and cream of tartar made into a mass with either mucilage or syrup. His #2 pills contained colocynth, gamboge, aloe, and cream of tartar, also made into a mass with mucilage or syrup. Morison's Aperient Powders, made with equal parts of cream of tartar and sugar which he flavored with powdered cassia, was touted as "morning lemonade" to its users.[11(pp10,25)]

Morison based his vegetable universal medicines on a simple formula which he trumpeted in countless advertisements.

> All animals consist of *fluids* and *solids*.
> All embryo animals consist *entirely* of fluids.
> The chief fluid is the blood, from which all others are derived.
> Blood forms the body—*air* gives it life.
> All constitutions are radically the same.
> All diseases arise from impurity of the blood.
> All diseases arise from *one* source, and therefore require but *one* medicine.
> Proper purgation by vegetables is the only effectual way of curing *all* diseases.
> This vegetable purgative must be capable of being digested, and mixing with the blood, so as to rid the body of all superfluous humors (disease).
> This panacea was discovered by James Morison in the composition of his vegetable universal medicine.[11(p25)]

Morison was certainly not alone in his claims or his concoctions. Thomas Holloway's Pills and Ointment were advertised in seven different languages and Dr. Locock's Pulmonic Wafers promised to cure a cough in ten minutes and asthma or consumption in a "proportionately small space of time." Others included Dr. Locock's Female Wafers made of licorice and horehound candy, and a host of remedies whose chief ingredient was aloe—Dr. Baillie's Dinner Pills, Dixon's Antibilious Pills, Hooper's Female Pills, James's Analeptic Pills, Wyndham's Pills, Peter's Pills, Dr. Fothergill's Pills, Lady Crespigny's Pills, Lady Webster's Dinner Pills, and Fordyce's Pills.

Parr's Life Pills (after Thomas Parr, who reputedly lived to the age of 152!) consisted of aloe, rhubarb, jalap, extract of gentian, soft soap, liquorice powder, treacle, moist sugar, oil of cloves, and oil of caraway. The claims made by Parr tempted one critic to take license with the manufacturer's testimonials[11(p186)]:

> I hereby certify and swear to it, that at the age of fifteen years I had the misfortune to fall into the crater of Vesuvius, and was burned to a cinder; but on taking two of Parr's Life Pills, I completely recovered. At Waterloo I was blown to atoms by a Congreve rocket; but after taking one box and a half of the Pills I speedily got well, and with the exception of occasional *shooting* pains, which a single pill invariably relieves, I have since been a better man than ever. In 1828 I was cut in two by an engine and forty-five ballast waggons; but on taking one box of those life-renovating pills, I became one again. Last year, I fell from the Monument, and my head was driven in; but on taking three of thy pills, O Parr! a new head was observed springing up, and the old one sloughed off.
>
> Mit Hochachtung,
> Verbleibe ich u.s.w.
> Munchausen, Ph.D.
> Langerbogenschussprofessor

Soothing syrups containing opium or morphine, face preparations consisting of arsenic and oxide of zinc, stomach bitters made with whiskey, and health restorers containing narcotics were but a few of the products in the quack's materia medica.[3(p82)] One popular antineuralgic water contained inferior brandy soaked in tobacco and colored with sulphate of indigo. Similarly, specifics for rheumatism contained little more than red pepper, licorice, and sugar yet claimed to be charged "with electricity in combination with essential oils!"[12(p171)] Under the rubric of aperient, antibilious, and stomachic were the popular Abernethy's Pills, containing mercury and compound extract of colocynth; Bath's Digestive Pills, containing rhubarb, ipecac, capsicum, ginger, and gamboge; and Norton's Chamomile Pills, containing aloe, gentian, and oil of chamomile.[11(p406)]

Nostrums which sold as worm lozenges, worm cakes, soothing syrups, carminatives, cordials, and infants' preservatives were usually narcotics or mineral poisons in disguise. Ching's worm lozenges, containing a large quantity of mercury, combined with jalap or a similar purgative, were advertised as safe for children. Mrs. Winslow's Soothing Syrup promised a perfectly safe "natural sleep" and its manufacturer sold it as a harmless preparation for

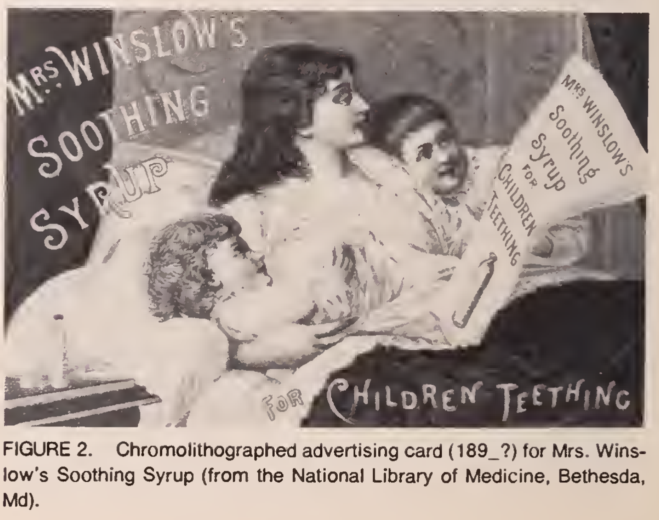

FIGURE 2. Chromolithographed advertising card (189_?) for Mrs. Winslow's Soothing Syrup (from the National Library of Medicine, Bethesda, Md).

children. However, if left standing uncorked, the preparation evaporated, leaving a dangerously concentrated narcotic residue of opium or morphine at the bottom of the bottle. The same was true of Godfrey's Cordial, Holt's Specific for Whooping Cough, Steedman's Teething Powders, and Atkinson's Infants' Preservative.[1(p107)]

Among the plasters were Baynton's Plaster, Mahy's American Plaster, Scott's Plaster, Sterry's Poor Man's Plaster, Ali Ahmed's Plaster, Roper's Royal Bath Plaster, and Berg's AntiRheumatic Plaster. Most of these nostrums contained a mixture of powdered white lead, olive oil, cantharides, black resin, and bees' wax.

In America, the highly touted half pint bottles of United States Sarsaparilla, United States Extract of Sarsaparilla, Mr. Moat's Sarsaparilla, and Dr. King's Sarsaparilla Pills flavored with guaiacum contained not a particle of sarsaparilla.[11(p227)] Repeated examinations of these nostrums showed that iodides and mercurial salts, generally chlorides, and alcohol, were the chief agents.[12(pp167–168)] Of the eleven sarsaparillas studied by the Colorado State Medical Society, the smallest amount of alcohol in any of them was 17%.[13(p246)]

Sexual problems were cured with nostrums sold as "silent friend," "cordial balm of syriacum," "concentrated detersive essence," "purifying specific pills," and "preventative lotions." Promising cure for "nervous patients," "barrenness," "deficiencies of natural strength," "female complaints," "weakness," and "turn of life," the Cordial Balm of Syriacum offered a ready and complete cure. Perry's Purifying Specific Pills offered an effectual remedy for gonorrhoea, gleets, strictures, irritation of the bladder, and other disorders of the urinary passages. Perry's Preventive Lotion guaranteed that its users could have "connexion with the fair but frail Cyprians who perambulate our streets, without any fear or reason to dread the consequences in the shape of the venereal disease." Similar promises existed for La'Mert's Self-Preservation, Curtis's Manhood, and Curtis's Anti-Venereal Lotion.[11(pp284,362)]

Many nostrums were concocted to restore men to "those duties which are among the most sacred to human nature." Advertisements warning of "premature decline," "decay," "early excesses," "youth chained by noxious indolence and complete imbecility," "ruinous practices," "ebbing energies," "losses of the system's most important fluid," "hereditary debility," and the "consequences of youthful indiscretion" fed the anxieties of Victorian men who feared impotence as a result of either masturbation or nocturnal emissions (spermatorrhoea). One such nostrum, known as Sir Astley Cooper's Vital Restorative, sold during the 1860s and guaranteed a cure for those "special ailments" peculiar to the male. Each drop of the Vital Restorative's concentrated powers pervaded the entire system, acting directly upon the brain and spinal cord, strengthening "the whole frame work of man, rendering it strong, and fitted to suit all his wants and desires." According to the manufacturer, its precious drops re-invigorated "God's favoured masterpiece." When taken in the prescribed quantities it resulted in "reviving nature's exhausted functions, and restoring health, nervous and muscular power, and manhood, under every circumstance, and at every age, nourishing, fertilizing, and developing every part of the human organization, thereby insuring to the mind and heart its fondest desires."[14(pp28,30–31)]

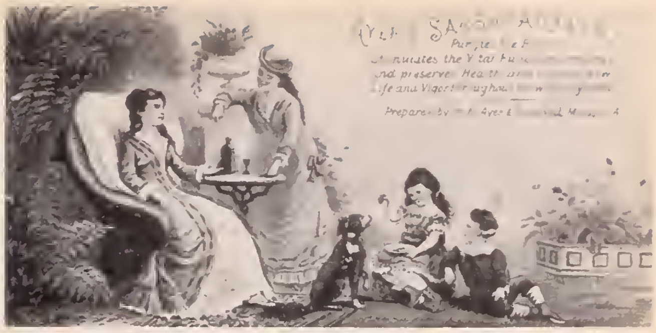

FIGURE 3. Chromolithographed advertising card (189_?) for Ayer's Sarsaparilla (from the National Library of Medicine, Bethesda, Md).

Another form of hucksterism focused on pregnancy. Without making any direct insinuation, advertisements for pills such as Sir James Clark's or Periodical Drops promised to correct "female irregularity;" they cautioned, however, that women in "a delicate situation" should not use them. In effect, manufacturers promised to restore women to their regular menses through the use of nostrums containing powdered savin, aloe, hellebore, ergot, and the solid extracts of tansy and rue. These became the main ingredients of 19th century abortifacients.[15]

Probably the most unethical of all the nostrums were those promising to relieve the horrors of opium addiction. Advertising for these remedies assured that cure was possible, and for a specified sum of money, payable in advance each month, the habitue could receive a vial of pre-

FIGURE 4. Lithograph, hand colored, 1820, by Caroline Naudet, 1775–1839.

cious cure. Without an examination of the patient and with no knowledge of the circumstances surrounding the addiction, the vendor provided an eight-ounce vial of liquid which, upon analysis, proved to be little more than a solution of morphine with coloring matter and essence to disguise the contents. All this for only five times the price of opium itself! Actually, this amounted to shrewd economy. In effect, vendors sold off the residue of the manufacture of morphine as a cure for the victims of the opium habit.[12(pp174–175)]

Typically, nostrum vendors concealed the alcohol content of their products. According to the Massachusetts State Board Analyst, the amount of alcohol by volume in the most popular nostrums was significant (Table I).[16]

OPPOSITION

Opponents of proprietary medicines argued that their popularity discouraged the practice of scientific pharmacy, defrauded the sick, and obscured the distinction between quack medicines and legitimate prescriptions. In addition, they gave quackery a degree of unwarranted respectability, degraded rational therapeutics, and demoralized the medical profession by tempting the retail druggist to prescribe for customers. Equally important, the marketing of patent medicines rendered scientific nomenclature problematic. After reading the claims of such inventive mixtures as Lactopeptine, Maltine, Vitalized-Phosphites, Celerina, Bromidia, Iodia, Petroleum syrup, Soluble Phenole, Malto-cocoa, Hydroleine, Listerine, Caulocorea, and Viburnum Compound in the advertising pages of journals and newspapers, it was difficult for both the medical profession and the public to discern their difference from prescription medicines. When prescriptive drugs could no longer be distinguished from the popular Jayne's Expectorant, Hop Bitters, and Swain's Panacea, the relations between the physician and pharmacist could only deteriorate. Curing dysmenorrhea with Hyden's Viburnum Compound which carried the testimonials of several prominent physicians and which women could purchase off the shelf seemed far easier and less expensive than a visit to the physician for an examination and prescription, and then another trip to the pharmacist for a prescribed cure.

TABLE I. Alcohol Content of Nostrums, 1902[16]

Name	Percent of Alcohol by Volume
Lydia Pinkham's Vegetable Compound	20.6
Paine's Celery Compound	21.0
Dr. Williams's Vegetable Jaundice Bitters	18.5
Whiskol, "a non-intoxicating stimulant"	28.2
Colden's Liquid Beef Tonic, "recommended for treatment of alcohol habit"	26.5
Ayer's Sarsaparilla	26.2
Thayer's Compound Extract of Sarsaparilla	21.5
Hood's Sarsaparilla	18.8
Allen's Sarsaparilla	13.5
Dana's Sarsaparilla	13.5
Brown's Sarsaparilla	13.5
Peruna	28.5
Vinol, Wine of Cod Liver oil	18.8
Dr. Peters' Kuriko	14.0
Carter's Physical Extract	22.0
Hooker's Wigwam Tonic	20.7
Hoofland's German Tonic	29.3
Howe's Arabian Tonic, "not a rum drink"	13.2
Jackson's Golden Seal Tonic	19.6
Mensmen's Peptonized Beef Tonic	16.5
Parker's Tonic, "purely vegetable"	41.6
Schneck's Seaweed Tonic, "entirely harmless"	19.5
Baxter's Mandrake Bitters	16.5
Boker's Stomach Bitters	42.6
Burdock Blood Bitters	25.2
Hoofland's German Bitters, "entirely vegetable"	25.6
Hop Bitters	12.0
Hostetter's Stomach Bitters	44.3
Kaufman's Sulfer Bitters, "contains no alcohol"	20.5
Puritana	22.0
Richardson's Concentrated Sherry Wine Bitters	47.5
Warner's Safe Tonic Bitters	35.7
Warren's Bilious Bitters	21.5
Faith Whitcomb's Nerve Bitters	20.3

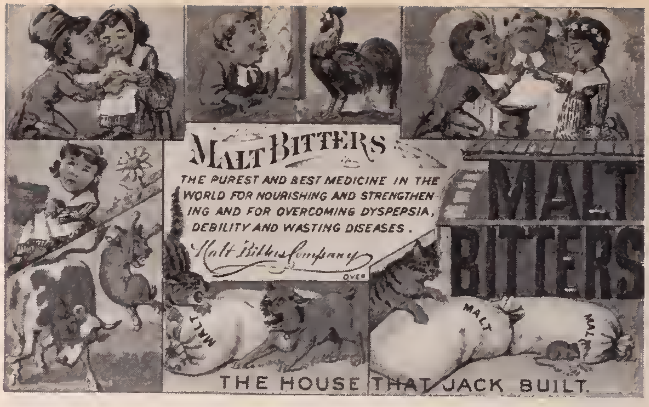
MALT BITTERS
THE PUREST AND BEST MEDICINE IN THE WORLD FOR NOURISHING AND STRENGTHENING AND FOR OVERCOMING DYSPEPSIA, DEBILITY AND WASTING DISEASES.
Malt Bitters Company
OVER
MALT BITTERS
MALT
MALT
MALT
THE HOUSE THAT JACK BUILT.

FIGURE 5. Chromolithographed advertising card (189_?) for Malt Bitters (from the National Library of Medicine, Bethesda, Md).

The medical profession loudly criticized druggists for defrauding them of their fees by prescribing medicines across the counter. In response, many doctors chose to dispense their own medicines, thus removing both the pharmacist and the temptation to self-dose. Naturally, pharmacists accused physicians who dispensed their own drugs of depriving them of their livelihood. As one physician admitted, the medical profession deliberately and without apology had defrauded the pharmacist of his function.

> We degrade his vocation to the business of a storekeeper, we compel him to pay the profits which are justly his to other manufacturing pharmacists of distant cities, and so enhance the cost of medicines to our patients. Having in this way hindered the cultivation of pharmacy as a science, injured our local pharmacist in his business, and increased the cost to our patients, it is not astonishing that we are still so blind to the impropriety of the whole thing that we cannot see that in doing so we have also provided the most direct, effectual, and surest means of injuring ourselves, and even opened up another serious source of danger to the public.[17(p6)]

Opposition to the patent medicine trade also contained elements of racism and nationalism. In *A Bomb in the Camp of the Enemy, Or an Exposure of Quackery and "Patent Medicines,"* published in 1845, J. P. Epperson took particular issue with the so-called Indian remedies. "What a wretched state of intelligence must that be," he wrote,

> which venerates and cherishes the traditions and crude notions of that absolutely ignorant and stupid race!—a race, which, in respect to all proper human characteristics, are but little elevated above the stupid beasts which they hunt and slay! What sort of intelligence is that which is captivated more by a remedy, or notion of Indian renown, than by the experiments and opinions of the most enlightened and scientific men? The venerators of Indian physic should be consistent, and go on to adopt Indian religion, Indian morals, and Indian government; or, more correctly speaking, Indian heathenism, brutism, and barbarism; or, in a word, Indian ignorance and stupidity.[5(pp4–5)]

Epperson, who lived in Pulaski, Tennessee, also blamed much of America's interest in nostrums on "Northern quacks with Southern pills." He attributed the Yankee fondness for money as a catalyst to the popularity of patent medicines in America. "It is equally well established," he wrote,

> that many of them [Yankees] are opposed to following ordinary and useful occupations to gain it. Hence they invent all sorts of notions. It is also certain that they are shrewd enough to guess that, as the people are generally captivated by pompous novelties from a distance, and the South is in a state of commercial dependency on the North, they can conveniently tack on a medical dependency likewise.

Given that northern cities were "overstocked with physicians and druggists," the "inferior members of both classes" chose willingly and deceptively to avoid "honorable competition" and engage in nostrum selling. For Epperson, northern nostrums contributed no small amount of the "inconsiderable channel by which Southern money is drained into Northern pockets."[5(pp17–18,22)]

CONCLUSION

As with many of America's reform efforts, legislation covering the use and abuse of nostrums did not occur until late in the 19th century. Initial attempts by city and state governments to register dispensers of drugs met with only limited success. Nevertheless, city governments implemented regulations as early as 1804 in New Orleans, 1851 in Louisville, 1866 in Williamsport, and between 1870 and 1876 in Baltimore, San Francisco, Philadelphia, St Louis, Cincinnati, and Milwaukee. These legislative initiatives to license pharmacists had the effect of reducing the number of individuals vending drugs. By 1870, some 25 states had passed statutes concerning the adulteration of drugs. Much of this legislation required the manufacturer to label the contents of dangerous medicines. Many of these same states had passed legislation prohibiting the sale of abortifacients and regulating the sale of alcohol.

In addition, the American Pharmaceutical Association, founded in 1852, presented a model law on the regulation of drugs and medicines at its meeting in Chicago in 1869. This model law became the basis of some 30 state laws adopted in the 1870s and 1880s. Although this legislation attempted to define the difference between pharmacists and merchants and in effect establish the professional identity of the pharmacist, it did little to curb the proliferation of patent and proprietary medicines. Eventually, the uneven nature of this social legislation resulted in a model food and drug act prepared by the National Board of Trade, and efforts by the Bureau of Chemistry in the US Department of Agriculture to address the problems of adulterated foods and drugs.

The frustration over the lack of consistent regulatory legislation across state lines and the increased level of social concern with drug adulteration lent impetus to "progressive" legislation at the turn of the century. The Federal Food and Drug Act of 1906, although initially responding to practices in the meat-packing industry, also affected patent medicines. This legislative act superseded efforts of self-regulation and resulted in a more comprehensive interstate control of food and drugs. Within a few years, most states had revised their own legislation to address intrastate violators in a manner consistent with the federal legislation. Despite the best intentions, however, patent and proprietary medicines continued to flourish within the pervasive language of the statutes and were not sufficiently regulated until the Harrison Narcotic Act of 1914, the Food, Drug and Cosmetic Act of 1938, and later amendments which insisted on more exact labeling of contents and directions.

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Student participation in mixed gender sports competition

Male and female post-pubescent youngsters should not participate in competition with each other in those sports in which the frequency and severity of collision forces is substantial. This is a medical position based on sound principles of anatomy and physiology. The concept is supported by the American Medical Association, the American Academy of Pediatrics, and the New York State Public High School Athletic Association.

Our legal system, in trying to conform with the principle of equal rights for the sexes, has disregarded the potential for injury in favor of a legal opinion. If this principle holds, certain considerations should be kept in mind when approving of the participation in sports of individuals of one sex with individuals of the opposite sex.

Physical Size. There are growth charts which allow us to determine into which percentile a given individual is placed. There are separate charts for males and females. The size of the individual in question should be evaluated using the gender of those on the team. Therefore if a girl wishes to play on a boys' team, her height and weight percentiles should be determined using the chart for boys. Only in this way can a determination be made as to how the individual compares with his or her teammates.

Physical Fitness. This is best determined using the New York State Physical Fitness Screening Test. Again, no matter the gender of the individual taking the test, he or she is compared with the gender of those individuals on the team. In this way one can determine if the applicant's ability is equal, inferior, or superior to that of his or her peers.

Past Activity and Experience. The motivation for participation by the student must be examined. Is this a capricious whim of the student or has the student actively participated in the sport in previous years? Is this a sincere endeavor leading to a continuing pursuit or a desire for sensationalism? Sincere interests and goals should be respected.

Adequate Locker Room Facilities. Is it possible to provide adequate locker room facilities for both male and female students, both at home and when playing at other locations? Facilities should be similar and private.

Attitude of the Coach. An athlete of the opposite sex from that of the other team members requires equivalent amounts of training and coaching. Playing time must be based on athletic ability and an assessment of the ways in which the athlete can contribute to the team's success. A coach who admits that there may be some difficulty in performing as described will consciously or subconsciously create an obstacle.

Attitude of the Team Members. Does the student wishing to join the team have the respect of the team members as an athlete and as an individual? For success there must be mutual acceptance.

Protective Equipment. We readily accept the differences in male and female clothing. Protective equipment must be specifically designed for the person it is to protect. Available equipment may not fit a person of the opposite gender. Some of the areas requiring protection may vary between the sexes. In any case, proper fitting, well designed, protective equipment should be available.

Documentation of Increased Risks. Whenever post-pubescent students of opposite sexes participate in contact sports there is an increased risk of injury. If a girl wishes to play on a boys' football team she is at risk. A boy playing on a girls' volleyball team places the team members at risk. If a boy and a girl are of equal size and weight, the girl has less lean body mass which produces a difference in the ability to create power. Generating a weaker force, females will absorb more of the force created by a collision. These risks must be explained to the individual or team and be documented in case of future medical-legal problems.

Increased Risks to the Opposing Team Members. A member of the opposite sex may influence or put at risk the participation of the opposing team members. Their views should be sought.

The preceding considerations are suggested as a guide in reaching approval or disapproval decisions concerning the participation of an individual of the opposite sex as a member of a team designated for a particular gender. There are many local variations and options. Each case must be considered individually.

These guidelines were prepared by the Committee on School Health and Sports Medicine and approved by the Council of the Medical Society of the State of New York.

The clinical value of fine needle aspiration biopsy in a patient with medullary thyroid carcinoma and pseudofollicular carcinoma

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Since the recognition of medullary thyroid carcinoma (MTC) as a distinct entity in 1959, it has been viewed as an integral part of multiple endocrine neoplasia (MEN)-2a (Sipple syndrome), which includes pheochromocytoma, usually bilateral, and hyperparathyroidism due to hyperplasia or adenoma, and MEN-2b, which includes additional abnormalities such as mucosal neuromas, hyperplastic corneal nerves, and an assortment of skeletal anomalies.[1-3] In the case reported here, a fine needle aspiration biopsy (FNAB) diagnosis of MTC required preoperative consideration of the syndrome of MEN-2 for the patient and her children, who were screened for elevated plasma calcitonin, an infallible but not the only neuropeptide marker for MTC.

Since its reintroduction in recent years,[4-8] fine needle aspiration biopsy of the thyroid has become widespread, and its usefulness has greatly increased. It is especially reliable in the diagnosis of benign diseases such as nodular goiter and Hashimoto thyroiditis, and malignant diseases such as papillary carcinoma, anaplastic carcinoma, and MTC. Multiple fine needle aspiration biopsy from different sites of the same lesion minimizes possible diagnostic errors. When follicular or Hürthle cells are identified in the aspirate, excisional biopsy with careful histologic study is required to differentiate a benign adenoma from carcinoma. Evacuation of benign thyroglossal duct, thyroid, and parathyroid cysts can be performed easily and without risk.

CASE REPORT

A 40-year-old woman was referred for evaluation of an enlarged thyroid gland. There were no signs or symptoms of hyperthyroidism or hypothyroidism, and no history of radiotherapy to the head or neck. A single firm, nontender 4 × 6-cm mass was felt in the left lobe of the thyroid gland. A radioiodide study disclosed an uptake of 39%—the nodule did not concentrate radioiodide. A fine needle aspiration biopsy was performed. The initial diagnosis was that of a mixed follicular carcinoma and MTC. With additional evaluation, the apparent follicular elements were more accurately characterized as pseudofollicular elements of the MTC. Further study disclosed an extremely cellular aspirate with sheets of polyhedral cells; some cells were spindle-shaped with large oval, elongated nuclei (Fig 1). Both types of cells possessed a distinctly granular cytoplasm. Some fragments of material suggested colloid production, and on restaining the tissue with Congo red, the characteristic apple green birefringence of amyloid was evident when examined by polarized light. Restaining of other slides by the immunoperoxidase technique confirmed the presence of calcitonin in the epithelial cells with a granular cytoplasm (Fig 2), confirming the diagnosis of MTC. Follicular elements of varying size with large and varied nuclei were also identified.

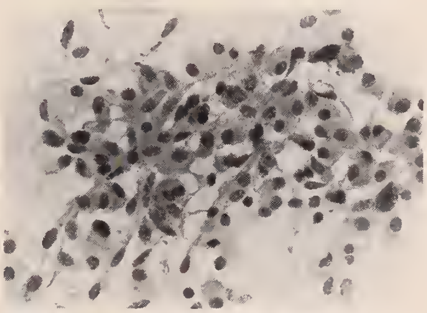

FIGURE 1. Fine needle aspiration biopsy of thyroid demonstrates both spindle and polyhedral cells with very fine cytoplasmic granules. Appearances are essentially diagnostic of medullary carcinoma of thyroid (Papanicolaou stain; original magnification × 1,600.

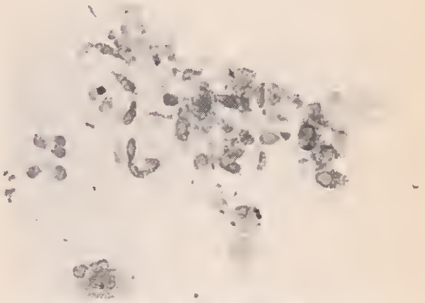

FIGURE 2. Fine needle aspiration biopsy of thyroid with immunoperoxidase staining confirms the presence of calcitonin in the cytoplasm of all the cells (original magnification × 1,600).

Following fine needle aspiration biopsy, the calcitonin level was found to be 26,400 pg/mL (normal, less than 400 pg/mL). The thyroxine level was 5.1 μg/dL (normal, 4.5 to 12.5 μg/dL). The hemogram, cholesterol, electrolytes, blood urea nitrogen, creatinine, lactate dehydrogenase, and serum glutamic-oxaloacetic transaminase levels were all within normal limits. The 24-hour excretion of metanephrines was 0.41 mg (normal, 0.3–0.9 mg), and the 24-hour excretion of vanillylmandelic acid was 3.71 mg (normal, 1–8 mg).

The patient's three children, ages 20, 19, and 16 years, had normal baseline levels of calcitonin. When pentagastrin was administered to the patient's 20-year-old daughter, her calcitonin level did not rise. On October 1, 1982, the patient underwent a total thyroidectomy. A well-encapsulated nodule was present in the lower part of the left lobe. No evidence of lymph node involvement was seen, and no other suspicious areas were identified within the rest of the gland.

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A well-circumscribed tumor 5.5 cm in diameter occupied the lower part of the left lobe of the thyroid, compressing the adjacent normal thyroid tissue.

On histologic evaluation, the tumor consisted of follicular and medullary components (Fig 3). There was no evidence of capsular invasion; one blood vessel contained tumor. No tumor was found in the rest of the thyroid gland. Also, no C-cell hyperplasia was found. Congo red staining confirmed amyloid stroma within the medullary areas. Electron microscopy demonstrated neurosecretory granules in the tumor cells and amyloid fibrils in the stroma (Fig 4). Immunoperoxidase staining confirmed the presence of calcitonin (Fig 2). Thyroglobulin was also present in the pseudofollicular and other cells (Fig 3).

The tumor concentration of adrenocorticotropic hormone (ACTH), insulin, and vasoactive intestinal peptides was determined by radioimmunoassay, according to methods established in our laboratory.[9-11] The calcitonin content of the tumor tissue was 255 μg/g; ACTH content, 2.4 ng/g; insulin content, less than 5 ng/g; and vasoactive intestinal peptide content, less than 7.5 ng/g. The ACTH immunoreactivity was totally removed from the water extract by incubation with Quso, suggesting the presence of an actual hormonal form rather than artifact in the assay system, even though the concentration noted was relatively low.

The postoperative course was uneventful. Within 87 hours, the calcitonin level returned to normal and has remained so (Table I). Pentagastrin did not induce a significant elevation of calcitonin in February 1984, 15 months after surgery.

The patient was placed on a regimen of 0.2 mg of levothyroxine daily and has since been completely well.

DISCUSSION

Identification of MTC requires serious consideration of possible MEN-2 in the patient and family, since these syndromes are inherited as autosomal dominant traits. No other clinical or laboratory data revealed skeletal or other anomalies, and no abnormal urinary catecholamine metabolites of hypercalcemia were found in this patient. No excess calcitonin was detected even after pentagastrin stimulation in her children. Thus, this appeared to be a case of sporadic MTC and not an MEN-2 syndrome.

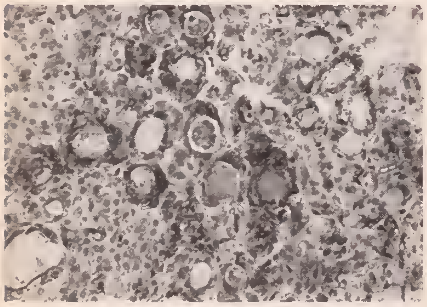
FIGURE 3. Immunoperoxidase staining for thyroglobulin showing both follicular or pseudofollicular elements. The dark areas confirm the presence of thyroglobulin throughout the tumor in both follicles and single cells or groups of cells (original magnification × 400).

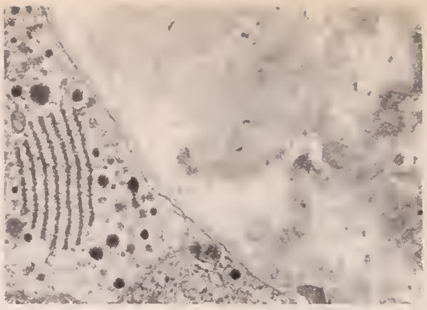
FIGURE 4. Electron micrograph revealing amyloid in the stroma. Intracellular neurosecretory granules and rough endoplasmic reticulum are also seen (original magnification × 47,500).

The diagnosis of MTC can be made based on a markedly elevated level of calcitonin in the serum or plasma, but calcitonin is not routinely assayed prior to surgery, with the exception of cases that are known to have a history of MEN-2. In nonfamilial cases of MTC, the diagnosis has usually been made after surgery. In the patient described here, the diagnosis of MTC was made before surgery, based on the results of fine needle aspiration biopsy, with Congo red staining and immunoperoxidase, and was confirmed by a very high concentration of serum calcitonin levels.

Serial calcitonin levels within 87 hours after surgery decreased to normal levels and have remained normal during a 44-month follow-up period (Table I). Since an extremely elevated calcitonin level (25,600 ng/mL) and blood vessel invasion were noted, it is surprising that there were no extrathyroidal metastases of the carcinoma.

Occasionally, when the plasma concentration of calcitonin is not elevated in small MTC or C-cell hyperplasia,[12] or after surgery in the presence of residual regional or metastatic disease, provocative tests using infusions of pentagastrin, calcium, or both[13,14] can be helpful. Testing family members is important, since about one fifth of patients without any familial history of MTC may actually be index cases for familial MTC, inherited as an autosomal dominant trait.[1-3]

Although bilateral MTC occurs in 90% of subjects with this disease, multicentricity is present in 20% of nonfamilial or sporadic cases.[1-3] In this patient, the calcitonin concentration in the tumor was significantly elevated at 255 μg/g. ACTH content, present in a relatively low concentration, was not clinically significant. No insulin or vasoactive intestinal peptide was found in the tumor.

Criteria for the diagnosis of MTC include stromal amyloid, the occurrence of cells in nests or solid trabeculae, and nuclei that are small and uniform with some atypia. Cystoplasmic secretory granules are positive for a variety of neuropeptides, polypeptides, and invariably calcitonin, but occasionally for a variety of substances, including ACTH, vasoactive intestinal peptide, neurotensin, somatostatin, dopadecarboxylase, histamine, histaminase, catecholamines, carcinoembryonic antigen, serotonin, prostaglandin, kallikrein,

TABLE I. Serum Calcitonin Level Before and After Surgery (Normal, <400 pg/mL)

Date	Time from Surgery	Serum Calcitonin (pg/mL)
5/18/82	Baseline/before	26,400*
10/1/82	5 h†	4,590
10/2/82	17 h	1,650
	26 h	1,150
10/3/82	40 h	755
10/3/82	48 h	660
10/4/82	64 h	415
10/5/82	87 h	348
7/19/83	9.5 mo	9‡
11/10/83	13 mo	11
2/2/84	16 mo	68§
3/18/85	30 mo	70
5/5/86	44 mo	47

* Upper limit of normal 400 pg/mL from 5/18/82–10/5/82.

† h = hours, mo = months.

‡ Upper limit of normal 100 pg/mL since 7/9/83.

§ Three minutes following intravenous injection of pentagastrin.

corticotropin releasing hormone, and other substances.[2,3]

Follicular carcinoma is characterized by the arrangement of thyroid epithelial cells and follicles of varying sizes, nuclei that may vary from uniform to atypia, varying ability to concentrate radioiodide, colloid, and the demonstration of intracellular thyroglobulin.

Ljunggren et al[15] reported that some of the tumor cells in one case contained both thyroglobulin and neurotensin. Thyroglobulin in the region of the medullary carcinomatous cells may merely represent spillage from the normal follicular cells or follicular carcinoma, or may be due to the incorporation of normal thyroid cells in the tumor tissue.[16] In the case presented here, the tumor contained follicular elements with colloid that were immunoperoxidase-positive for thyroglobulin; however, the distribution of follicles was scattered throughout the trabecular stroma so as to suggest that they were pseudofollicular and thus an integral part of the MTC.

There is controversy regarding the cellular origin of each of these tumor cells. If follicular carcinoma is derived from the thyroglossal duct, and MTC originates from the C-cells of the branchial pouches—ie, neuropeptide secretory cells migrating from neuroectoderm, as proposed by Pearse[17]—the occurrence of a mixed tumor would be coincidental. That neuropeptide producing cells along with follicular cells may arise from the endoderm has been suggested by the occurrence of the appendocarcinoid tumor of the appendix and colon,[18,19] and by finding mucus producing cells in a case of MTC.[20] Insulin has been produced in chick embryos from which the ectoderm has been removed prior to the differentiation of the neural crest,[21] suggesting an endodermal origin of neuropeptide secreting cells with a common precursor cell.

Ljungberg et al[22] presented a group of 14 cases with features of a solid tumor (MTC) and papillary and/or follicular patterns. All tumors contained thyroglobulin, and 12 of the 14 contained neuropeptide, calcitonin, somatostatin, and/or neurotensin. In the authors' view, there is a spectrum of neoplasms. At one end there are the solid tumors capable of producing identifiable neuropeptides and, at the other, pure follicular and/or papillary tumors that synthesize and secrete thyroglobulin. Between these two extremes, an assortment of tumors exists, in which the cells may produce thyroglobulin or neuropeptides, or neither.

These data also tend to support a common stem cell origin, further undermining the APUD-neuroectodermal origin for peptide-producing thyroid tumors.

This case illustrates that fine needle aspiration biopsy is a simple, inexpensive, and readily available technique that can provide valuable and accurate information promptly concerning a large variety of both benign and malignant thyroid tumors.

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Renal arteriovenous fistula in the setting of subacute bacterial endocarditis

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Mycotic aneurysms are an infrequent complication of subacute bacterial endocarditis. Occurrences in the cerebral, hepatic, splenic, mesenteric, hypogastric, profunda femoris, and renal arteries have been reported.[1,2] Development of an arteriovenous fistula secondary to erosion of a mycotic renal artery aneurysm into a renal vein is an extremely rare event. Such a case is the subject of this report.

CASE REPORT

A 65-year-old woman was admitted to the medical service with a one-month history of back pain. The patient had been in fairly good health, physically quite active, and living alone in her own home. This illness began four weeks prior to admission on a Caribbean vacation trip when she awoke in the morning with pain in the mid and upper back radiating bilaterally below the scapula. She saw a local physician who gave her some pain medications. Her condition improved, but pain developed in another location in the back.

Ten days after returning from vacation the patient was seen by her regular physician, who noted marked restriction of bending and pain on straight leg raising at 60 degrees bilaterally. Radiographs of the lumbosacral spine showed scoliosis toward the left and degenerative changes at L3-L4. She continued to have back pain and, on reexamination a week later, findings were unchanged. However, her hematocrit was noted to be 28.1%, and her white blood cell count was 9,500/mL. Blood urea nitrogen and creatinine levels were 18 mg/dL and 0.6 mg/dL, respectively. There was no history of bleeding. The stool was negative for occult blood. A repeat hematocrit three days later was 32%. The patient's symptoms showed slow improvement over the next five days, but in the 24 hours prior to admission, she experienced excruciating back pain and generalized weakness.

On admission, the patient's pulse was 90/min; blood pressure, 140/80 mm Hg; and temperature, 36.8°C orally. A grade 2/6 systolic ejection murmur was heard over the aortic area. Extremities showed 1+ edema of both lower legs below the knees. The white blood cell count was 12,400/mL; hematocrit, 30.7%; urinalysis showed 1–5 red blood cells per high power field (HPF) and 1–5 white blood cells per HPF with no casts; blood urea nitrogen, 12 mg/dL; creatinine, 1.0 mg/dL.

A computed tomographic (CT) examination of the L-S spine showed only central disc bulging at L4-5 and L5-S1. While the patient was in the hospital, a spiking fever to 38.6°C was noted. Her white blood cell count had increased to 15,300/mL. Blood cultures were positive for *Streptococcus viridans*. At this time, a history was elicited from the patient of having had her teeth cleaned and a dental filling performed the week before she went on vacation. Cardiac ultrasound subsequently revealed a bicuspid aortic valve with vegetations. Numerous splinter hemorrhages were noted in the nail beds bilaterally. The patient was treated with intravenous penicillin-G and gentamicin. The gentamicin was discontinued after 14 days. After the first week in the hospital the patient was noted to have increased signs of congestive heart failure. Lasix was started. Her condition continued to improve, and she had an uneventful course until the sudden onset of severe pain over the right flank on the 17th hospital day. A new loud systolic and diastolic bruit was heard over the whole abdomen, loudest over the right upper quadrant where a prominent thrill was palpable. Deep palpation produced some tenderness over the right upper quadrant. A voided urine at that time was amber with 1–2 red blood cells per high power field. The blood urea nitrogen was 16 mg/dL and creatinine, 1.0 mg/dL.

An intra-arterial digital subtraction angiography abdominal aortogram and right renal arteriogram (Fig 1) showed a large right renal artery with a 7-mm aneurysm, distally from which there was rapid filling of a renal vein dilated due to an aneurysm. Rapid flow was noted on the serial films, with contrast opacification of the dilated renal vein, inferior vena cava, and right atrium during the arterial phase of the injection. The mask images of the selective right renal arteriogram showed a grossly normal excretory pyelogram (Fig 1). This was confirmed by dynamic and static images during a Tc 99m DTPA renogram. The dynamic images from the renogram also showed the early accumulation of activity in the renal arteriovenous fistula during the vascular phase of the renogram. The patient underwent a right nephrectomy. The surgical specimen showed a 7-mm arteriovenous fistula with granulation tissues seen in the wall. There was no evidence of glomerulonephritis or other abnormality. No bacteria were cultured from the

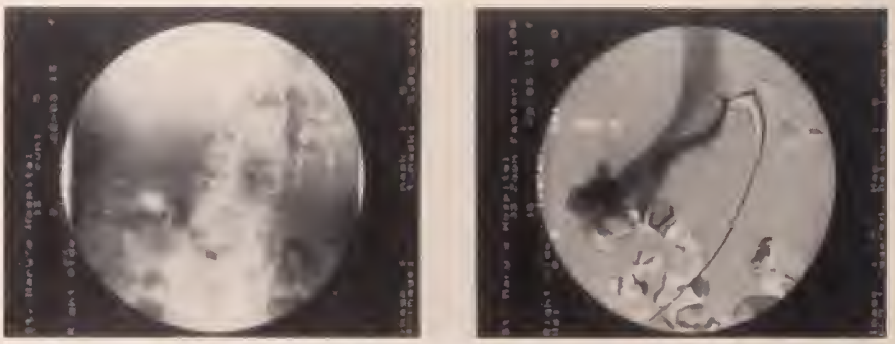

FIGURE 1. A selective right renal arteriogram shows a large right renal artery with a 7-mm aneurysm distally, from which there is rapid filling of a dilated renal vein. The mask image before selective injection but following the abdominal aortogram shows a grossly normal pyelogram (image on left).

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fistula site. Marked aneurysmal dilation of the adjacent renal vein was noted. The postoperative course was uneventful. However, a new diastolic aortic murmur was detected during an office follow-up visit after discharge from the hospital. Follow-up examination over two years has shown a stable aortic insufficiency which is not hemodynamically significant.

DISCUSSION

Despite effective antibiotic treatment of infective endocarditis, severe complications are not uncommon and are increasingly being recognized. This is probably related to the increased life span of these patients as compared to that of the pre-antibiotic era, allowing for more time for serious complications to develop.[3] In addition to major cardiac complications such as cardiac failure, valvular insufficiency, myocardial abscesses, and pericarditis, extracardiac complications can occur. These are usually felt to be related to direct arterial embolization to major organs, as evidenced by autopsy series,[4,5] but can also result from other mechanisms.

Renal involvement in bacterial endocarditis[6] can occur in several ways, including embolization to the renal vessels leading to infarction and/or abscess formation, an immune complex glomerulonephritis which is most commonly focal proliferative on histologic analysis, and acute renal failure, the causes of which can be multifactorial. Renal infarction is the most common renal lesion[7] and probably represents embolic occlusion of the renal vasculature. Infarcts usually occur early in the course of antibiotic therapy but can occur as a delayed event after the acute stage of the disease has been controlled.[8]

A relatively uncommon complication in the renal circulation is the development of a mycotic aneurysm. These aneurysms can result from direct bacterial invasion of the arterial wall with subseqent abscess formation and rupture, septic or bland embolic occlusion of the vasa vasorum, a direct extension from a contiguous infected focus, or trauma to an arterial wall with direct contamination.[9] While mycotic aneurysms can involve any artery, vessels involved in descending order of frequency include the aorta, cerebral circulation, visceral arteries, and peripheral vessels.[10] Involvement of multiple vessels is common and was found in 49 of 217 cases reviewed by Stengel and Wolferth.[10] These patients had a total of 382 mycotic aneurysms, of which only five were in the renal vasculature. Subsequent series describing patients with mycotic aneurysms have also rarely demonstrated renal involvement.

Recently DuBrow and Patel[11] described three cases of mycotic aneurysm of the renal artery, all of which showed evidence of focal nephrographic defects (renal infarcts) with normal visualization of the collecting system on intravenous pyelogram and small solitary aneurysms of a branch artery. All of these patients had urography and angiography done for reasons other than a primary suspicion of a renal artery aneurysm, highlighting the fact that these lesions are usually silent and may go unrecognized unless a complication develops.

The patient described here provided an unusual presentation of this complication, in that the sudden appearance of right flank pain associated with the development of a new systolic-diastolic bruit and thrill over the right upper quadrant possibly heralded the rupture of an aneurysm into the adjacent renal vein. The clinical manifestation of a high-pitched bruit, both in systole and diastole and accompanied by a thrill, suggested the presence of an underlying arteriovenous communication and tended to exclude other causes of bruit.[11] Other clinical manifestations of a vascular fistula including cardiomegaly, high output congestive heart failure, diastolic hypertension, and hematuria[12] were not present, probably because of the short duration in this case.

Renal arteriovenous communications can be congenital or acquired; the former are usually cirsoid in structure and represent a truly complex lesion comprising many arteriovenous connections. Acquired fistulas may occur spontaneously secondary to an arterial wall inflammation with the formation of a pseudoaneurysmal connection to the venous system.[13] In addition, more common causes of acquired renal arteriovenous fistulas include needle biopsies of the kidney, trauma, renal cell carcinoma, and renal surgery.[14] Most of these patients present with prominent abdominal bruits or evidence of hemodynamic compromise, but the underlying cause of these symptoms can go unrecognized.

Urography and arteriography are the mainstay of diagnosis. The urographic appearance may vary from that of a nonfunctioning kidney to that of irregular filling of the pelvis and calyces,[12] while arteriography demonstrates an early run-off of contrast from the arterial injection with prompt appearance in the venous circulation, as was seen in this patient. In addition, the Tc 99M DTPA renogram may be suggestive of the lesion, as in this patient. The presence of inflammatory changes in the renal arterial wall in the nephrectomy specimen suggests the possibility of a mycotic aneurysm as the primary lesion. The absence of a definite abscess at the fistula site may be related to the prolonged antibiotic treatment or a bland embolic occlusion of the vasa vasorum as the cause of the developing aneurysm and arteriovenous fistula. While anticoagulant therapy may decrease the incidence of such bland emboli, the increased risk of bleeding from mycotic aneurysm or arteriovenous fistula would seem to preclude their use. Other causes of renal artery inflammation, including polyarteritis nodosa and tuberculosis,[14,15] would be unlikely in this clinical setting. The presence of an aneurysmal dilation of the renal vein is also consistent with the finding of the arteriovenous fistula because of high arterial pressures in the venous circulation.

Although most mycotic aneurysms and arteriovenous fistulas do not require surgical treatment, the location of the fistula in this patient and the chance of further complications including rupture[16,17] prompted surgical intervention and nephrectomy. While noninfected arteriovenous fistulae and congenital arteriovenous malformations may be effectively treated by primary vascular repair, embolotherapy, or balloon occlusion, these procedures risk the possibility of a smoldering infection in a poorly vascularized area, for example in the case of mycotic aneurysm.

We have described a patient with an unusual complication of subacute *Streptococcus viridans* bacterial endocarditis, which resulted in the development of a renal arteriovenous fistula, possibly secondary to a ruptured mycotic aneurysm of the renal artery. Although involvement of the renal vasculature secondary to embolization in bacterial endocarditis is not uncommon, renal mycotic aneurysms are rare, and development of renal arteriovenous fistulas is distinctly uncommon.

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Ultrasound diagnosis of jugular venous ectasia

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Internal jugular venous ectasia is a rare entity that is generally not considered in the differential diagnosis of pediatric neck masses.[1] It is a benign vascular anomaly which presents as a lateral neck mass during episodes of laughing or crying, and once correctly diagnosed, requires no therapy or further evaluation. Recognition of the characteristic features of this abnormality will prevent confusion with more significant pathologic processes. The purpose of this communication is to present the sonographic findings in a case of internal jugular venous ectasia that was clinically unsuspected.

CASE REPORT

A six-year-old boy presented for evaluation of an asymptomatic left-sided neck mass. His parents noted that the mass appeared only when he cried, coughed, or laughed. He had no other medical problems and had a normal developmental history. There was no history of antecedent trauma. At rest, no mass was detectable. When he performed a Valsalva maneuver, a soft, diffuse bulge was noted deep to the inferior margin of the left sternocleidomastoid muscle (Fig 1). The cardiovascular examination was unremarkable. No other abnormalities were detected.

At rest, ultrasound demonstrated normal internal carotid arteries and internal jugular veins bilaterally. No mass was apparent on the left side (Fig 2) at rest. Following a Valsalva maneuver, a cystic mass measuring 3 × 2 cm became evident on the left side of the neck (Fig 3). The mass corresponded in location to the internal jugular vein and exhibited normal venous Doppler signal. The right side was unchanged following Valsalva maneuver.

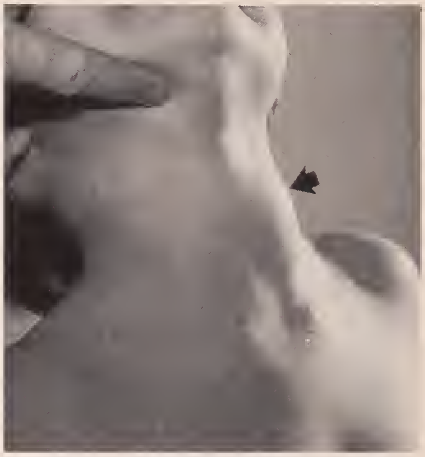
FIGURE 1. Photograph of patient with left-sided neck mass (arrow) which appears with a Valsalva maneuver.

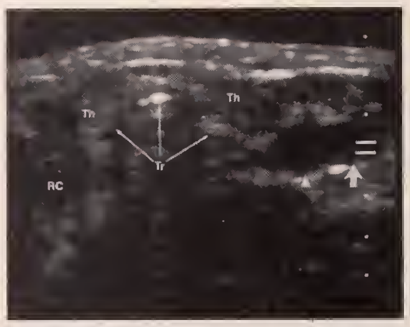

FIGURE 2. Transverse scan of the neck at rest showing left internal carotid artery (arrowhead) and normal caliber left internal jugular vein (arrow). The thyroid gland (Th), the tracheal air column (Tr), and the right internal carotid artery (RC) are indicated.

No further studies were done. Follow-up examination at one year showed no further change.

DISCUSSION

Jugular venous ectasia (also referred to as jugular venous aneurysm and jugular phlebectasia) usually presents in childhood[2-4] and appears to be a congenital anomaly,[5] unlike adult venous aneurysms, which are more often posttraumatic. The internal jugular vein, on either side, is by far the most commonly affected.[2-9] The external jugular[3] and the anterior jugular[7] veins have also

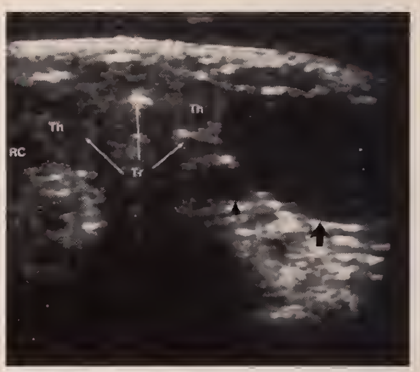

FIGURE 3. Transverse scan of the neck during Valsalva. Note marked distension of the left internal jugular vein (arrow), with no change in the size of the left internal carotid artery (arrowhead). The thyroid gland (Th), the tracheal air column (Tr), and the right internal carotid artery (RC) are indicated.

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been reported to be involved. While the mass is usually noted by a parent, children occasionally will complain of a "rumbling" sensation in the area.[2] A venous hum is sometimes noted on physical examination. Associated cardiovascular abnormalities have not been reported, and central venous pressure is normal. The abnormality is usually unilateral, although a case with bilateral ectasias has been reported.[6] The lesion may be resected in cases of relentless "rumbling" or thrombosis with possible embolic complications.

Surgical specimens[2,3,7,8] have had the gross appearance of dilated internal jugular veins. Histopathologic examination in one report showed focal loss of elastic fibers, which was felt to represent a primary vessel wall abnormality predisposing to subsequent dilation. Associated intimal thickening, connective tissue proliferation, and endothelial cell hyperplasia were attributed to the effects of longstanding turbulence.[2] Other reports have failed to duplicate this finding, showing normal venous wall anatomy[3,7] or uniform thinning.[8]

Neck masses are a common pediatric clinical problem. The determination must first be made as to whether or not the mass arises from the thyroid. Lateral neck masses that do not appear to be related to the thyroid may be inflammatory or noninflammatory, and the latter may be benign or malignant.[1] The differential diagnosis of neck masses that arise in the thyroid in children includes Hashimoto thyroiditis, goiter, abscess, cyst, and adenoma. Cervical adenitis and abscess present as extrathyroidal inflammatory neck masses. Noninflammatory extrathyroidal masses are generally benign, with the rare exception of lymphoma, metastatic neuroblastoma, and teratoma. These include fibromatosis coli and cystic masses such as thyroglossal duct cyst, branchial cleft cyst, dermoid cyst, enterogenous cyst, laryngocele, mediastinal cyst, cystic hygroma, lymphocele, hemangioma, and jugular venous ectasia. Of these, only a laryngocele, mediastinal cyst, or a jugular venous ectasia will distend with Valsalva maneuver and disappear completely at rest. Ultrasound examination with Doppler signal sampling rapidly distinguishes jugular venous ectasia by showing the identity of the mass with the internal jugular vein.

Previously, the diagnosis of jugular venous ectasia in children was established surgically[2,7,8] or angiographically.[3,7] The first published case of sonographic diagnosis demonstrated diagnostic accuracy by confirmation with angiography.[4] Since then, computed tomographic (CT) correlation has been obtained as well.[5,9] Ultrasonography with Doppler before, during, and after Valsalva maneuver is now the preferred method for diagnosing this entity. Awareness of this benign abnormality on the part of the clinician and radiologist may circumvent further unnecessary diagnostic evaluation.

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Malignant lymphoma presenting with severe hypercalcemia

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Hypercalcemia is a serious, sometimes life threatening complication of malignant disease.[1] Malignant lymphomas of the Hodgkin and non-Hodgkin type are not often associated with this potentially fatal complication. However, hypercalcemia associated with malignant lymphoma does occur and has been reported.[2-7] For example, Canellos[6] found only four of 217 patients (1.8%) with malignant lymphoma to have hypercalcemia, while in another series Stewart and coworkers[7] found that two of 50 patients with hypercalcemia of malignancy had lymphoma. The underlying pathogenesis of hypercalcemia in these disorders in the absence of osseous metastases is incompletely understood. There is strong experimental and clinical evidence that certain bone resorbing factors such as parathyroid hormone-like substances, prostaglandins, vitamin D metabolites, and osteoclast activating factor (OAF) may be selectively secreted by the neoplastic cells leading to the development of hypercalcemia.[2-4,8-10]

The purpose of the present communication is to report an unusual case of a patient with non-Hodgkin lymphoma presenting with severe hypercalcemia without evidence of osseous metastases. The absence of a demonstrable parathyroid hormone concentration or an elevated 1,25-dihydroxyvitamin D level suggests that humoral factors such as OAF or prostaglandins may have been responsible for bone resorption, causing the severe hypercalcemia in this patient.

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CASE REPORT

A 54-year-old man with a history of alcohol abuse and heavy tobacco use (2–3 packs/day) for many years was brought to the emergency room because of a five-day history of increasing confusion, disorientation, ataxia, anorexia, polydipsia, and polyuria. His last alcohol intake was approximately five days prior to presentation.

Physical examination disclosed a dehydrated, conscious but agitated male with stable vital signs. There was no palpable lymphadenopathy or splenomegaly, but the liver was enlarged. The patient was restless and disoriented, but no focal neurologic deficits could be demonstrated. Admission laboratory data revealed a white blood cell count of $12,000/mm^3$ and a normal differential cell count. The hemoglobin was 12.6 g/dL; hematocrit, 38.2%; platelet count, $134,000/mm^3$. Normal red cell and white cell morphology were observed. The erythrocyte sedimentation rate was 54 mm/h. Pertinent blood chemistry values on admission included the following: urea nitrogen, 61 mg/dL; creatinine, 3.3 mg/dL; total calcium, 19 mg/dL (normal, 8.8–10 mg/dL); phosphorus, 4.7 mg/dL (normal, 2.5–4.5 mg/dL); alkaline phosphatase, 84 IU/L (normal, 30–115 IU/L); lactate dehydrogenase, 910 U/L (normal, 100–225 U/L); glutamine-oxaloacetic transaminase, 66 U/L (normal, 7–50 U/L); and magnesium, 1.1 mg/dL (normal, 1.8–2.6 mg/dL). The chest film was normal, and an electrocardiogram showed sinus rhythm with a QT interval of 0.46 sec at a rate of 85 beats/min.

The patient's hospital course was complicated by refractory hypercalcemia unresponsive to vigorous intravenous saline rehydration and calcitonin administration. This required hemodialysis one day after admission. Concomitantly, the patient was given one dose of mithramycin (25 μ/kg of body weight). After the initial dialysis, the calcium decreased to 15.9 mg/dL. The patient underwent dialysis again on the second day of hospitalization with a further reduction in the calcium to 10.8 mg/dL. However, the patient experienced respiratory failure requiring intubation and mechanical ventilation.

Serum protein electrophoresis and serum immunoelectrophoresis revealed a relative increase in the gamma globulin fraction consistent with a benign polygammopathy. Thyroid function studies were unremarkable. An intact parathyroid hormone (PTH) level obtained on admission and measured using the Allegro™ Intact PTH Immunoassay system revealed an undetectable serum parathyroid hormone concentration. Additionally, a 1,25-dihydroxyvitamin D level drawn on the second day of the patient's hospital stay was 14.2 pg/mL (normal, 12.7 to 32.2 pg/mL). Human T-cell lymphotrophic virus Type 1 (HTLV-I) serology was also reported as negative (Dr Dugan, Health Science Center, Syracuse University, Syracuse, NY). A limited skeletal radiographic survey was unremarkable, and a bone scan showed only abnormal uptake in both lungs, kidneys, and stomach consistent with calcium deposition in soft tissue. Although some abnormal lymphocytes were identified on an initial bone marrow aspiration study, a definitive diagnosis could not be made. A computed tomographic (CT) scan of the abdomen revealed two hypodense areas in the right lobe of the liver. These lesions were biopsied under CT guidance and interpreted as non-Hodgkin lymphoma, mixed small and large cell type. A repeat bone marrow aspiration was performed, which confirmed the liver biopsy findings.

Control of the patient's hypercalcemia was maintained by periodic doses of mithramycin. This resulted in a brief period of clinical improvement, allowing for successful extubation. Subsequently, however, the patient's condition deteriorated, characterized by hypotension, respiratory distress, and *E coli* septicemia. This required re-intubation, the use of vasopressors to maintain an adequate blood pressure, and antibiotic therapy. Despite this therapy, his condition continued to worsen, and he died after 22 days of hospitalization.

At autopsy, multiple supradiaphragmatic lymph nodes were enlarged, measuring up to 3.5 cm and having a firm, white cut surface. Nodular and diffuse tumor involvement was found in the liver and spleen. Three parathyroid glands were examined, each one having a normal size and weight. No macroscopic involvement of bone or other organs was seen.

Microscopic examination of the lymph nodes revealed a diffuse malignant lymphoma, mixed small and large cell type,[11] which was found to involve extensively the liver, spleen, and bone marrow, and to a lesser degree, to involve the kidneys, adrenals, and lungs. Histologic bone examination showed increased numbers of osteoclasts with accelerated bone resorption. Soft tissue calcification was generalized and involved both vessel walls and the parenchyma of multiple organs. Immunophenotyping by flow cytometry of the bone marrow aspirate and light chain staining using the immunoperoxidase technique on paraffin embedded tissue indicated that the neoplasm was of B-cell origin.

DISCUSSION

The patient described in this case report appears to have had hypercalcemia due to the production of one or more humoral bone resorbing factors. His clinical course, laboratory data, and autopsy findings are consistent with this supposition.

The low concentration of immunoreactive parathyroid hormone (PTH) in the serum, the presence of hyperphosphatemia, and the lack of hyperchloremic acidosis[12] provide evidence that PTH or PTH-related peptides were not the mediators of hypercalcemia in this patient. The normal-sized parathyroid glands also suggest that the duration of hypercalcemia was relatively brief.

The biologically active metabolites of vitamin D, particularly 1,25-dihydroxycholecalciferol, have been implicated in the pathogenesis of hypercalcemia in patients with malignant lymphoma.[1,10] The low level of this sterol in the presence of marked hypercalcemia suggests that it was not causally related to the hypercalcemia. Therefore, an unanswered question remains as to which humoral factor(s) might have been responsible for the development of the severe hypercalcemia observed in this patient. Recent evidence suggests a role for other known stimulators of bone resorption such as prostaglandin E_2 or OAF.[1,2,4,9] For instance, Rossi et al[2] described nine cases of hypercalcemia and/or lytic bone lesions occurring in patients with B-cell malignancy, including non-Hodgkin lymphoma. Their findings clearly indicated that B-cell cancers other than multiple myeloma can produce significant bone resorbing activity in vivo as well as in primary tumor cell cultures established from the subjects.[2] Based on the features of these cases common to multiple myeloma, the authors emphasized the possible role of OAF as the humoral agent responsible for bone resorption. Although bone resorbing prostaglandins have not been as extensively investigated as OAF, their role as mediators of hypercalcemia in malignancy is suspected on the basis of results obtained in tissue culture[13] as well as in animal and human tumor studies.[9,14]

In summary, this case demonstrates that malignant lymphoma is a disease that belongs to the group of neoplasms associated with humorally induced hypercalcemia, and that the symptoms related to hypercalcemia may prompt the patient to seek medical attention. The case also points to the role of humoral factors other than PTH-like peptides and vitamin D metabolites in the pathogenesis of hypercalcemia associated with malignant lymphoma.

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FROM THE LIBRARY

MEDICAL EDUCATION IN THE MID-NINETEENTH CENTURY FROM A STUDENT'S POINT OF VIEW

From the Private Journal of the Affairs du Vie 1842–1852 by Henry Darwin Didama, M.D.

A Medical Career Begins. On a bright sunny day, Sept. 1st, 1842, in the village of Cazenovia, whose streets resound with the lively hum of business, I find myself seated in the office of Dr. Moore for the professed purpose of acquiring the art of healing some of the many diseases which "flesh is heir to." My name was entered just one month since but I did not commence my studies till the 22nd ult. I have read as far as Syndesmology in Wistar's Anatomy.

Nineteen long years have rolled their rounds over my head and never till now have I had a settled object at which to aim. I have dwelt in the regions of fancy . . . today forming hopes and resolutions which tomorrow would crash and break—existing only in the present and not daring to raise the curtain which veiled the future—passing my time in happy communion with kindred spirits and forming friendships and alliances which in future times are to be the guardian stars of my feeble bark on the ocean of life or the false lights which shall lure it onward only to shatter it on the rocks of disappointment—Now the scene is changed. I have left the abode of fancy and the home of romance and have entered my name as an actor in that great drama of reality—human life. The profession which I have made my choice and to qualify myself for which I am now striving is one requiring all the energy, all the perseverance which any mortal can possess. Three long years of severe mental labor will only give me a slight knowledge of the mechanism of the human system and if I succeed in obtaining a diploma I must expect to encounter the black waves of opposition—the bold and open attack of interested rivals and the cowardly and concealed, but nevertheless dangerous and often fatal, shafts of the most vile of all God's creatures—the insinuating slanderer.

Yet I hesitate not, I tremble not. With the motto *"Onward"* (which my friend Lansing suggests) raised to the breeze I push forward resolved to act my part manfully and if I then fail, if the black waters of adversity then roll over me I shall find some consolation in the thought that *I have done my best.*

COMPILED BY WILLIAM A. BRUMFIELD, JR, MD,
(NY State J Med 1957; 57:595-596)

LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Buck v Bell

TO THE EDITOR: It was with great interest that I read the reprinted excerpt of Lloyd Paul Stryker's 1927 review of the *Buck v Bell*[1] case in the March 1989 issue of the *Journal.*[2] Lloyd Paul Stryker was one of the most famous lawyers of his time. His review alone of this well-known case would be dramatic and interesting enough. But the United States Supreme Court was represented in this case by the great Justice Oliver Wendell Holmes, the son of an equally famous father, the well-known poet and physician of the same name. Justice Holmes himself waxed poetic when, in justifying a Virginia statute legalizing enforced sterilization, he wrote, "Three generations of imbeciles are enough."[1] Holmes was referring to Carrie Buck, a retarded state-institutionalized individual who was both the daughter of a retarded mother and the mother of a retarded daughter.

There is a certain ring to that line. It is always interesting to see the surprised look on the faces of lawyers who are interested in human rights and who feel secure in our democratic heritage and personal liberties when they read this case for the first time.

In *Constitutional Law,*[3] I covered this very interesting case under the title, "Equality of Treatment Within a Class." The general rule is that all persons affected by a particular classification must be treated equally. They are protected by the Equal Protection Clause of the 14th Amendment to the United States Constitution, which is applicable to state actions only.

In *Buck v Bell,*[1] a Virginia statute provided for the sterilization of feebleminded persons confined to state institutions. Feeble-minded people outside such institutions were not forced to be sterilized. Although there is an obvious distinction between persons confined to institutions and persons outside institutions, the Supreme Court nevertheless maintained that the state law did fulfill legally necessary requirements. These requirements dictated that all persons affected by a particular classification (here, state-institutionalized feebleminded persons) must be treated equally by indicating a policy and then applying the policy to all within its lines.

The year 1927 is the centerpiece here. This case arose before Hitler's laws came into effect as well as the tragedies and chaos that were to follow the dictator's insane course.

To appreciate *Buck v Bell* in its fuller historic and legal aspects, it is important to examine a case called *Skinner v Oklahoma,*[4] in which, in 1942, the Supreme Court held that a statute providing for the sterilization of habitual criminals was in violation of the Equal Protection Clause of the 14th Amendment of the United States Constitution since it discriminated against certain members of a class. For example, it provided for the sterilization of people convicted of grand larceny, but held immune persons who were convicted of embezzling. In *Skinner v Oklahoma* the Court held this to be invidious discrimination.

It is interesting to note that this World War II Court, while not specifically overruling *Buck v Bell,* apparently sought to legally forget it. In addition, Justice Jackson set the tone of the prevalent feeling by warning in a concurring opinion that "there are limits to the extent to which a legislatively represented majority may conduct biological experiments at the expense of the dignity and personality and natural powers of a minority." It is doubtful whether the Supreme Court would have held the same way if *Buck v Bell* had arisen after *Skinner v Oklahoma,* especially in light of the historic excesses perpetrated by the Nazis in their frenzied quest for their concept of racial purity.

Indeed, there remains one postscript to *Buck v Bell* that occurred in post-World War II years. During the Nuremberg trials of the Nazi war criminals, *Buck v Bell* was one of the cases cited by the defense attorneys as a defense and justification for some of the actions of Nazi war criminals and of the "laws" promulgated and passed by them. To this day, *Buck v Bell* is an interesting, intriguing, and well written case, thanks to the literary skill of Justice Holmes. It is further interesting to see it "reported" again in 1989 in a medical journal devoting an entire issue to human abuse. Hopefully *Buck v Bell* will now be seen in its proper historical perspective.

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Physician recredentialing

TO THE EDITOR: Drs Gellhorn and Cherkasky[1] attempt to lay out the conventional wisdom on recredentialing, but as in many other instances the conventional wisdom is wrong. Reassuring the public may be one achievable goal of recredentialing, but benefits limited to the public relations arena hardly justify such a large effort.

My experience during 30 years of daily hospital practice has led me to conclude that, in the absence of physical, psychiatric, or addictive problems, physician performance remains fairly constant over a working lifetime. The incompetents were that way when they left residency training (fully and freshly credentialed, of course). They often remain a burden to the delivery system and their patients until they retire. Training programs will have to develop a more consistent approach to deal with trainees unsuited for their chosen field by either temperament or poor motor skills. Senior faculty cite an unwillingness to create a gap in the rotation, fear of lawsuits, or the workload involved in a dismissal as inhibiting such action. Some programs defer operative experience for residents until the last year so that problems only come to light when it is too late to do anything. Polite but deficient residents are usually promoted while more talented but difficult residents may be at risk for dismissal. Residents whose specialty choice may be inappropriate need to be identified early and counseled intensively.

Physicians are correct to be negative about recredentialing while accepting licensing and board examinations. The licensing (or national board) examination documents appropriate preparation for initiating the residency training experience. Residency completion (or board certification) indicates enough preparation to continue learning without supervision. Cognitive tests are indeed hard to apply, especially for the most specialized, ie, who tests the testers? Who will test Dr David Axelrod, the Commissioner of Health of New York State, and on what will he be tested? Perhaps this explains the exemptions from licensing examinations that are sometimes granted to recognized teachers.

Recredentialing (and for that matter further training) has little to offer those whose original training did not achieve the desired results. Of course neither is it appropriate for a practitioner with physical, psychiatric, or substance abuse problems.

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In reply. Dr Atkinson feels that the only purpose of physician recredentialing is a public relations effort but then quickly describes the varieties of poor performance among physicians ranging from the physically, mentally, and drug addicted handicapped through the doctors who have made an inappropriate choice of specialty to those who were unsuited for their chosen field. This would seem to include a large number of physicians who should be identified for educational enhancement at the very least.

Contrary to Dr Atkinson's understanding, the specialty boards do not accept that certification provides a lifelong assurance of continued learning. This is why 16 of the 23 specialty boards have initiated plans for recertification.

It is well to emphasize to Dr Atkinson and other specialists that a majority of the specialty societies, including the American Society of Anesthesiologists, are developing standards of practice, applications of which will be one of the options to be used in peer review of specialists for recredentialing.

Finally, Dr Atkinson may rest assured that no one is to be exempted from physician recredentialing, including the Commissioner or the undersigned.

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Complications arising from the use of ophthalmologic medications in an intensive care unit patient

TO THE EDITOR: The critical care specialist is frequently confronted with deterioration of a patient's condition. Usually this can be ascribed to the exacerbation of an existing disease process or the onset of a new one. This should prompt a search for the inciting cause. An iatrogenic cause should always be considered. Because of the great number and variety of pharmacologic compounds used in the treatment of critically ill patients, drugs must be considered highly suspect as a cause for a patient's deterioration. The following case demonstrates the point.

Case Report. A 74-year-old woman was admitted to the neurosurgical intensive care unit with a subarachnoid hemorrhage. The diagnosis was based on a history of severe headache with change in mental status and was confirmed by a computed tomographic scan of the head. The patient's medical history was initially positive only for hypertension; there was no history of smoking or asthma. The physical examination was positive for mild confusion, aphasia, and hypertension with a blood pressure of 200/100 mm Hg. Serum chemistries, hemogram, and chest x-ray were normal. However, the electrocardiogram (ECG) revealed diffuse, nonspecific ST-T wave changes. Subsequent ECGs were normal and a myocardial infarction was ruled out.

The patient's hypertension was initially controlled with 5 mg intravenous doses of labetalol. Subsequently she was maintained on 200 mg of oral labetalol twice a day, with control of her blood pressure in the range of 120–150/80–90 mm Hg. Her other medications included intravenous epsilon amino caproic acid, lorazepam for sedation, and subsequently vancomycin for catheter related sepsis.

Approximately two weeks after admission the patient's family informed us that the patient was taking three preparations of eye drops for control of glaucoma: pilocarpine, propine, and ophthalmic timolol. That evening an ophthalmologic consultation was obtained and the patient was started on propine 1%, pilocarpine 2%, and betaxolol 0.5% one drop twice daily in both eyes, though her intraocular pressure was not elevated at the time. A chest x-ray taken the morning following initiation of her ophthalmologic medications revealed increased vascular markings. That evening tachypnea, tachycardia, rales, and hypoxemia developed. Congestive heart failure (CHF) was diagnosed and furosemide was administered intravenously, with resolution of symptoms after a 2.3 liter diuresis. Totals of input and output for the prior days revealed an excess of 2,500 cc over three days consistent with insensitive losses; a perfusion scan was negative, as was an ECG for evidence of ischemia or infarction. A review of the preceding events revealed that the only new intervention in an otherwise stable patient was the addition of the ophthalmologic preparations. A third dose of these medications was followed within a few hours by the abrupt onset of worsening confusion, wheezing, and hypoxemia, not long after the administration of the labetalol. This episode quickly resolved after the administration of nebulized albuterol.

In light of the prior episode of congestive heart failure, the question of recurrence was raised so a pulmonary artery catheter was inserted. It revealed a central venous pressure of 0 mm Hg, pulmonary artery systolic/pulmonary artery diastolic pressure of 20/2 mm Hg and pulmonary capillary wedge pressure of 1–3 mm Hg. Based on the clinical picture and the low filling pressures, congestive heart failure was ruled out and a diagnosis of bronchospasm was made. At this point the labetalol was changed to captopril, the ophthalmologist was reconsulted, and the betaxolol was discontinued. Subsequently there were no further episodes of CHF or bronchospasm and the patient's intraocular pressure remained controlled with levels in the mid to high teens.

Discussion. In a review article by Anad and Eschman,[1] the systemic effects of ophthalmic preparations were enumerated. Topically applied beta blockers can produce congestive heart failure and bronchospasm.[1–4] A small volume containing high concentrations of delivered medication is being instilled in a location where absorption may be great, causing development of significant blood levels of the given compound. These blood levels may be poorly tolerated by a patient and manifested as organ dysfunction as in this case, where bronchoconstriction and congestive heart failure were seen. This patient had no history of adverse effects from her glaucoma medication at home. However, she also was not receiving additional beta blockade at home. It would appear that the patient had insufficient beta blockade at her pulmonary and myocardial receptors to cause symptoms, but sufficient to make her susceptible to the added effects of the absorbed ophthalmic beta blocker. Additionally, the role of a cholinergic medication in the development of bronchospasm cannot be ignored.[1] Whether the discontinuation of one of the medications alone would have been sufficient is a possibility. However, her intraocular pressure did not require a third therapy and an alternative antihypertensive was chosen, so a repetition of this combination of medications would not occur upon discharge from the hospital.

This case is reported to increase awareness of the possibility that ophthalmologic medications can contribute to adverse effects of other medications and to underscore the need to consider these medications as a cause for sudden deterioration of a patient's condition.

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Clinical causes of death of elderly patients in a long term health care facility

TO THE EDITOR: The National Heart, Lung, and Blood Institute noted that in 1983, 51% of deaths in the United States were due to cardiovascular disease.[1] These deaths primarily occurred in patients 65 years or older. Coronary heart disease was the commonest cause of mortality due to cardiovascular disease. Rudman et al[2] reported that the most common immediate causes of death of 24 patients (average age 70 years) in a Veterans Administration nursing home were infections (67%) and cardiac disorders (25%). We performed a prospective study to investigate clinical causes of death of elderly patients in a long term health care facility. Follow-up data over a 45-month period are presented.

From November 1, 1984, through July 31, 1988, 1,040 patients (290 men and 750 women) between the ages of 62 and 103 years, mean age 82 ±8 years, resided at Hebrew Hospital for Chronic Sick. During this 45-month period, 521 of 1,040 patients (50%) died at Hebrew Hospital for Chronic Sick or at a general hospital to which they were transferred. The major clinical cause of death, including all pertinent data, was reviewed in all cases by the author.

Myocardial infarction was documented as previously described.[3] Primary ventricular fibrillation was defined as ventricular fibrillation documented by electrocardiogram in a clinically stable patient with cardiac disease unexpectedly having new cardiovascular symptoms. Sudden cardiac death was diagnosed when a patient with cardiac disease unexpectedly was found dead within one hour of being clinically stable.[4] Some patients with sudden cardiac death died within minutes after having new cardiovascular symptoms before an electrocardiogram could be taken. Chi-square analysis was used to analyze data.

TABLE I. Clinical Causes of Death of 521 Elderly Patients in a Long Term Health Care Facility, 1984-1988

Cause of Death	Number of Patients
Myocardial infarction	71 (14%)
Primary ventricular fibrillation	35 (7%)
Sudden cardiac death	99 (19%)
Refractory congestive heart failure	24 (5%)
Thromboembolic stroke	55 (11%)
Cerebral hemorrhage	10 (2%)
Pulmonary embolism	13 (2%)
Mesenteric vascular infarction at surgery	4 (1%)
All cardiovascular causes	311 (60%)
Pneumonia	75 (14%)
Urosepsis	34 (7%)
Bacterial endocarditis	7 (1%)
Sepsis from gangrene of lower extremity	1 (<1%)
Sepsis from adominal abscess	1 (<1%)
All infectious causes	118 (23%)
Cancer	49 (9%)
Renal failure	20 (4%)
Gastrointestinal or liver disease	9 (2%)
Chronic obstructive pulmonary disease	7 (1%)
Hematologic disorders	7 (1%)

One-hundred and fifty-three of 290 men (53%) and 368 of 750 women (49%) (difference not significant) died during the 45-month period. Table I presents the clinical causes of death of 521 elderly patients in a long term health care facility.

The major cause of death in the elderly population under study was cardiovascular disease. The prevalence of coronary heart disease,[5] systemic hypertension,[5] and valvular heart disease[6,7] was high in our elderly population. These cardiac disorders often coexisted. Primary ventricular fibrillation, sudden cardiac death, and refractory congestive heart failure in an individual patient may have been contributed to by coexisting coronary heart disease or cardiomyopathy, aortic and mitral valve disease, and systemic hypertension.

Infections were the second major cause of death in our patients. Pneumonia was the commonest infectious cause of death. Prior stroke contributed to aspiration pneumonia in some of these patients.

Bacterial endocarditis was documented as the major cause of death in seven of our patients. Physicians taking care of elderly patients should keep in mind the high prevalence of valvular heart disease in elderly patients[6,7] and use prophylactic antibiotics in patients with valvular heart disease according to American Heart Association guidelines.[8]

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FROM THE LIBRARY

THE FAMILY PHYSICIAN

It is the new, the strange, the unusual that attracts attention. The sun in its apparent ceaseless round with light and heat, giving life and growth, hours for activity and for rest, has scarcely a thought. Day and night, summer and winter come and pass as a matter of course and attract little attention. Let a wandering comet flash above the horizon and every telescope in the area of observation is pointed toward it. The papers discourse daily on its appearance, distance and velocity. For the time being, it is all there is of the celestial hemisphere in the minds of the people.

In the world of medical literature much the same state of affairs exists. A daring, novel surgical operation, successfully performed, the discovery of a new therapeutic agent, or a new application of one already known, fills the medical journals and the newspapers with columns of description, admiration and just praise. The time of our Society meetings is largely taken up by the reports and conclusions of specialists, while the duties, needs and achievements of the general practitioner—the family physician—receive scant attention.

This is the fact to such an extent that the bright, ambitious young man graduating in medicine to-day is caused to think that he must embrace and practice some specialty or be unknown to fame, poor, in fact a nobody. I would not detract one iota from the mead of just praise that is due to men, who like McDowell set a pattern in abdominal surgery, which being well followed and even improved upon, has given hope and life to thousands who were formerly despairing invalids; or to an observer like Don Lopez who brought into use Cinchona for the treatment of malarial poisoning; or to the bacteriologist, who like Koch, proves the germ cause of disease; or Laennec, who first used the stethoscope; or physiologists like Harvey and Malpighi who discovered and demonstrated the circulation of the blood; or Roentgen who brings under observation the covered and hidden organs of the interior of the body. These and hundreds of others who have followed one line of effort and investigation have accomplished results which are of inestimable value to the human race. Specialists are necessary. Human life is too short, the extent of human effort is too limited to allow one person, or one school, to thoroughly master all subjects pertaining to medical science. Fortunate is he who can accept the results and most effectively apply the precepts of those who spend their lives in perfecting themselves in one subject.

We cannot all be specialists. While sickness and suffering exist, as long as children are born, families reared, and men die; while respect for and confidence in others are engendered in the human mind, so long will the afflicted turn to some one, who by virtue of special training, is considered competent to advise, so long will there be need of, and a call for, the family physician. . . .

R.P. BUSH, MD
(*Trans Med Soc State of NY* 1905, pp 291–294)

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A public health perspective on alcohol abuse

The manner in which alcohol abuse is viewed affects the manner in which government and society are likely to respond. In New York State, as in many other states, the problem has not been widely or systematically viewed from the public health perspective, nor has the public health community been significantly involved in preventive efforts. The societal focus has been on the control of a limited number of behaviors which produce specific unacceptable outcomes. A prominent example of this focus is drunk driving behavior which leads to motor vehicle injuries and fatalities. When the problem is narrowly defined, the response is often singular in nature; closer attention is paid to DWI laws and their strict enforcement. These responses may be effective within a limited time frame and among a portion of the alcohol-consuming public. However, it is unlikely that these responses achieve a broad-based and long-term effect since fundamental factors that underlie the targeted unacceptable behavior are not addressed.

When alcohol has been viewed as a health issue, the response has largely been affected by a definition of the health concern as a substance abuse problem, with an emphasis on treatment and rehabilitation of those afflicted with the disease of alcoholism. Preventive interventions often deal with alcohol and illicit drugs jointly, blurring important distinctions between the two. Such an emphasis has also effectively separated the problem from the public health sector in the state.

The public health and health care communities, working incrementally to influence evolving public opinion, can help generate a climate in which targeted interventions not only react to the consequences of individual drinking behavior. An appropriate public health goal is to increase awareness of the importance of agent, host and environmental factors and to facilitate a reduction of the prevalence of the risk behavior, alcohol abuse.

Agent

The properties of alcohol are related to the health consequences of its abuse. In sufficient doses, the drug alcohol is intoxicating, addictive and toxic. Three typical alcoholic drinks consumed by a 160-pound person within a two-hour period are generally sufficient to result in legal impairment and a reduction in motor skills; five drinks commonly result in legal intoxication. Since alcohol acts as a dehydrating agent, the sensation of thirst increases as more is consumed, providing a physiological basis for facilitating drinking to intoxication. There is considerable interpersonal variation on behavior and motor function.

The use of alcohol can also be addictive, entailing habituation, tolerance, physical dependence and withdrawal. Susceptibility to alcohol dependence is not uniform throughout the population and the role of genetics is not yet well understood, although studies of separated twin children of alcoholics have shown them to have an increased risk of alcoholism. Long-term excessive alcohol consumption can also cause tissue and cellular damage, which may promote disease or interfere with normal organ functioning.

Alcohol has also been shown to be toxic to the developing fetus. One to three of every 1,000 newborns may suffer from fetal alcohol syndrome. A baby exposed in utero may weigh less at birth and suffer from mental retardation or chronic learning disabilities.

Beginning in November 1989, alcohol beverage containers will be required to display this warning label: “Government warning: 1) according to the Surgeon General, women should not drink alcoholic beverages during pregnancy because of the risk of birth defects; 2) consumption of alcoholic beverages impairs your ability to drive a car or operate machinery and may cause health problems.”

Alcoholic beverages are relatively inexpensive. The real cost to the consumer of beverage alcohol has actually declined during the past 20 years when indexed against inflationary trends. Federal excise taxes on alcohol have increased little since 1951, and reflect an unequal taxing of the beverage ethanol content, with beer taxed at one-fourth and wine at one-twentieth the rate of distilled spirits.

Host

Genetic susceptibility appears to be a factor in predisposing individuals to chronic alcohol abuse and to alcoholism. Notwithstanding the variability in genetic predisposition, however, the intrinsic properties of alcohol are such that all individuals who consume alcohol are potential hosts for an adverse alcohol-related health outcome. The near-term health consequences of intoxication can affect virtually all who consume to excess, not only those with a genetic predisposition to chronic dependence.

There is a consistent relationship between current heavy adult drinkers who drink heavily and the age at which drinking was initiated and, independently, the age at first intoxication. For example, 5% of New York State residents 35–49 years of age who started drinking at 21 years age or older are currently heavy drinkers. Of those who started drinking at age 16 or younger, 25% are now heavy drinkers. Beer and wine coolers are the alcoholic beverages of choice for adolescents and young adults. Nationally, young men under age 30 consume 70% of the beer. A recent state survey of wine cooler consumption indicated that nearly 40% of wine coolers are consumed by adolescents between the ages of 14 and 20.

Environment

Environmental influences that promote the consumption of alcohol pervade society. Advertising, especially television commercials, develops and reinforces positive images and attitudes about alcohol consumption. Advertisements associate drinking with good times, friendship, family, romance, manliness and a generally more exciting and rewarding life. Each year, more than 5,000 beer commercials are aired on network television, frequently featuring athletes, rock music stars and other popular personalities.

Alcohol is widely available. Virtually every grocery market sells beer and wine coolers, often late into the night. There are more than 3,300 licensed retail package stores and more than 24,000 bars, clubs and restaurants licensed to serve alcoholic beverages. Serving can continue to 4 AM, depending on local ordinance. Alcoholic beverages are heavily advertised, widely available and widely accepted as an intrinsic element of social interaction. House parties, club and professional organization gatherings and virtually every other form of adult interaction commonly employ alcohol as a social lubricant. "Social drinking" is the term used to describe varied patterns of use. The advertising message conveyed on television and within other media that alcohol is associated with social enjoyment is reinforced daily by peers, parents and organizations. This message has enjoyed a largely undisputed position within the social consciousness. It is the environment in which adolescents form their attitudes and behaviors.

Although consumption is widely condoned or encouraged by community attitudes and opportunities to drink abound, the consequences of abuse are generally deplored. For example, although there is considerable acceptance of driving after drinking (21% of adult male drivers surveyed in New York reported having driven while feeling the effects of alcohol during the past year), there is distress about the consequences of drunk driving, especially when the victims are not associated with the drinking episode.

Cancer incidence among Native Americans in New York State

The August 1988 issue of *Epidemiology Notes* highlighted cancer mortality among Native Americans in New York State. It is important to also consider the incidence of cancer when studying patterns of malignant disease among selected populations because all individuals who develop cancer do not die of the disease.

A retrospective cohort study design was used to investigate patterns of cancer incidence among the Seneca Nation of Indians (SNI) between 1965 and 1984. Incident cancers were identified through a computer match of Seneca Nation cohort members with data files maintained by the New York State Department of Health's Cancer Registry during each year of the study period. Sex-, age- and calendar-year specific cancer incidence rates, exclusive of New York City,[1,2] were used to yield expected numbers of incident malignancies during the study period. Sex and age standardized incidence ratios (SIRs) were calculated based on the ratio of observed to expected cancer cases. (If the observed number of cases was equal to the expected number, the SIR would be 100.) Published tables of significance values[3,4] were used to calculate 95% confidence intervals around each SIR. A point estimate of cancer incidence was considered to be significantly different from expected if its 95% confidence interval did not include 100.

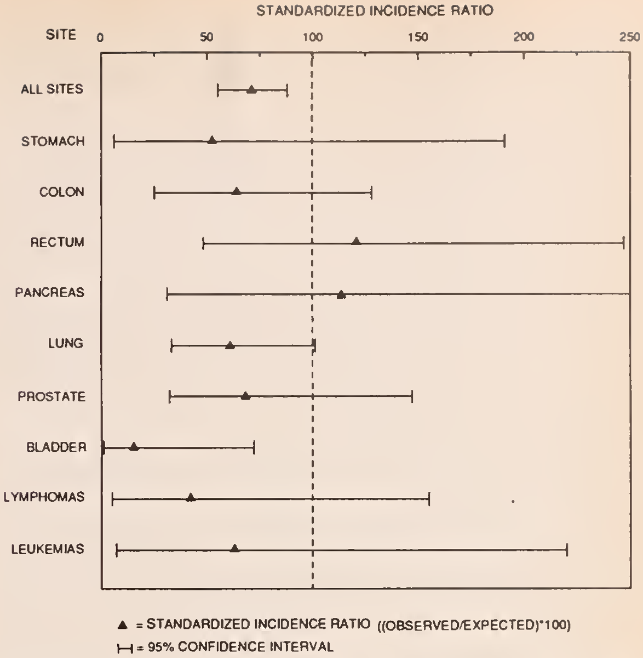

FIGURE 1. Cancer incidence among males, Seneca Nation of Indians, 1965–1984.

Figures 1 and 2 present SIRs for cancer among SNI males and females. Cancer incidence among SNI males between 1965 and 1984 was significantly reduced

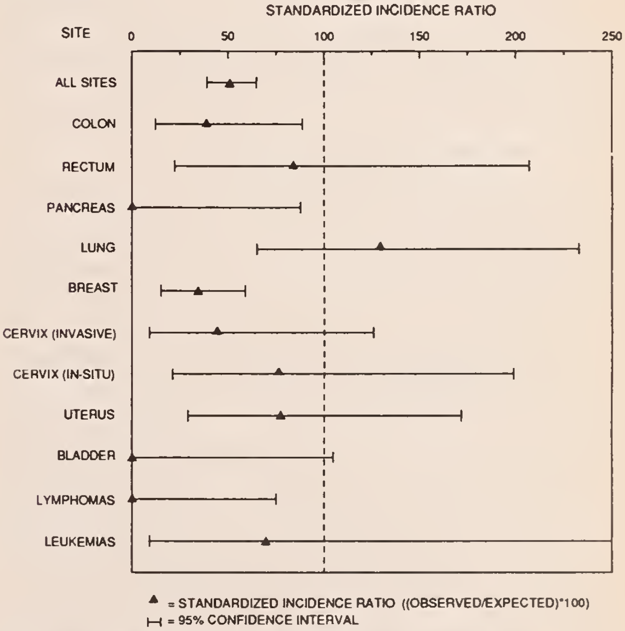

FIGURE 2. Cancer incidence among females, Seneca Nation of Indians, 1965–1984.

(SIR=70; 95% confidence interval (CI)=55–88). Among females, a significant deficit of cancer incidence was also observed for all sites combined (SIR=51; CI=39–65). Deficits in overall cancer incidence have been previously reported among both Native males[5–8] and Native females.[5,7,8] No cancer site demonstrated significantly increased incidence. As illustrated in Figures 1 and 2, point estimates of cancer incidence were consistently low among males and females for most cancer sites.

SNI females demonstrated a significant deficit in incidence for cancer of the pancreas between 1965 and 1984 (SIR=0; CI=7–88). A significant deficit in pancreatic cancer incidence has been previously reported only among Native males in Canada.[5]

Breast cancer incidence among SNI females was observed to be one third of the expected magnitude between 1965 and 1984 (SIR=32; CI=15–59). Other Native groups have exhibited a low incidence of breast cancer.[5–9] Indian mothers are twice as likely as white mothers to give birth before 20 years of age.[10] Furthermore, Indian females demonstrate higher fertility than white females.[10] Up until age 30 years, an inverse relationship exists between age at first pregnancy and a woman's risk of developing breast cancer.[11]

SNI females exhibited a low incidence of colon cancer (SIR=38; CI=12–89), however, colon cancer among SNI males (SIR=62; CI=25–128) was unremarkable. Observations of decreased colon cancer among the SNI are consistent with decreased colon cancer risks among Natives in New Mexico[7,8] and in Canada.[5,6] Possible explanations for this decreased risk include dietary factors, however, dietary data for the SNI are unavailable.

Between 1965 and 1984, a deficit of bladder cancer was noted among SNI males (SIR=13; CI=1–72), while bladder cancer incidence among females was not significantly different from expected (SIR=0; CI=0–105). Decreased bladder cancer incidence has been noted among other Native populations.[5,8,9,12] While no employment histories were available for SNI males, the significant deficit of bladder cancers among males may be due to a lack of exposure to etiologic agents in an occupational setting. Cigarette smoking is probably the most prevalent risk factor for bladder cancer among the SNI, however, information on smoking rates was not available.

While excess invasive and *in situ* cervical cancer incidence has been reported among Native American females in New Mexico,[7,8] the incidence of invasive cervical cancer (SIR=43; CI=9–126) as well as *in situ* cervical cancer (SIR=78; CI=21–199) among SNI females did not differ from expected.

Previous studies have suggested a deficit of incident leukemia cases among Native groups.[5,7,8,13] As shown in Figures 1 and 2, the incidence of leukemias among SNI males (SIR=61; CI=7–220) and females (SIR=71; CI=9–256) did not differ from expected. A deficit of incident lymphoma cases was noted only among SNI females (SIR=0; CI=0–75) over the 20-year study period. This is consistent with observations reported among Indian females in Manitoba, Canada[5] and in the United States.[7,9,13]

Since results of this study do not agree entirely with epidemiologic studies of cancer among Native Americans in other areas, this research raises additional questions regarding the interaction of genetics, diet and environment on cancer incidence among Native American populations. All Native groups are thought to have originated from Asiatic peoples who migrated across the Bering land strait which formerly connected Asia and North America. However, the present Native American population is heterogenous, even among tribes living in close proximity. While Native Americans often share similar living environments through residence on rural reservations, the interaction of genetic and lifestyle factors in observed patterns of cancer incidence remains unclear.

Decreased cancer incidence for all sites combined was observed among the SNI, with males exhibiting 76% of expected incidence and females 51%. Deficits of cancer mortality among Native American populations in the state are probably not solely the result of competing causes of death in this population and instead relate, in part, to lower cancer incidence in this population. Results from this investigation of cancer incidence will be valuable to the Seneca Nation to direct cancer control programs and services. For further information on this topic contact Dr Martin C. Mahoney, Director, Cancer Surveillance Program, Bureau of Cancer Epidemiology at (518) 473-7817.

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Designated care programs for patients with AIDS and HIV related illnesses in designated care centers

In January 1986, the New York State Hospital Review and Planning Council adopted emergency regulations to designate acute care centers for the care of individuals with AIDS and HIV related illnesses. The intent of the regulations was to develop centers of expertise in the clinical care and case management of AIDS and HIV related illnesses, and to increase access to essential services for people with AIDS. As of January 1989, the following hospitals have been designated as AIDS centers: Albany Medical Center; Beth Israel Medical Center; Bronx Lebanon Hospital Center; Montefiore Medical Center; Nassau County Medical Center; New York Hospital; Presbyterian Hospital; St Clare's Hospital and Health Center; St Luke's/Roosevelt Hospital Center; Strong Memorial Hospital; St Vincent's Hospital and Medical Center; University Hospital; Upstate Medical Center and North Shore University Hospital. Eight of the designated AIDS hospitals are in the New York City metropolitan area, three are on Long Island and three are in upstate New York. Thirteen AIDS centers are voluntary hospitals; one is a public facility. Designated AIDS hospitals accounted for 32.5% of 1987 statewide HIV related discharges and 30.7% of 1987 HIV related days.

To date, 14 additional facilities throughout the state have applied for designation. Twelve of these are in the New York City metropolitan area and two are in upstate New York. Three of these applicants are voluntary hospitals; 11 are public hospitals. Designated AIDS hospital applicants account for 29.9% of statewide 1987 HIV related discharges and 34.5% of 1987 HIV related days. A total of 25–30 hospitals are expected to be designated AIDS hospitals in 1989, providing approximately 60% of New York's hospital care for patients with AIDS and HIV related illnesses.

Each designated AIDS center must meet 13 program standards which address services provided during inpatient stays and use of ambulatory and community services from the time of the patient's entry into the AIDS center system, until withdrawal or death. This expectation represents a radical change in hospital care, as it requires AIDS centers to monitor patients and assure services after discharge.

Inpatient Care

AIDS centers have designated discrete inpatient units or scatter beds for the care of people with AIDS and HIV related illnesses in order to concentrate staff expertise and hospital resources. Specialized diagnostic and therapeutic services such as radiology and dialysis are provided or arranged when needed. The U.S. Centers for Disease Control's current recommendations concerning infection control have been integrated into each center's infection control procedures. To assure quality inpatient services, AIDS specific quality assurance programs were developed in addition to inservice education programs delivered to all hospital staff who care for people with AIDS and HIV related illnesses. Emergency services for the treatment of patients with AIDS and HIV related illnesses are available 24 hours per day.

AIDS centers must be linked with state of the art clinical treatments and therapies. AIDS centers currently participate in clinical research programs as approved by the institutional review board of each center. In addition, health services research is also underway at the designated AIDS hospitals.

Outpatient and Community Services

An important objective of the AIDS center program is to shift the primary emphasis of AIDS care from the inpatient to the outpatient service, as individuals with HIV infection can be identified early in the course of the disease and successfully maintained in the community. AIDS specific ambulatory or outpatient services for early diagnosis and treatment have been developed at each AIDS center. In addition, centers provide or arrange for the provision of a full range of home health services as well as participate in efforts to expand residential options such as skilled nursing and health related care.

AIDS centers serve their local communities by providing information and educational programs to the public regarding the prevention of HIV infection and treatment of AIDS. Many AIDS centers have established strong relationships with community-based organizations involved in HIV prevention efforts. In addition, AIDS centers provide or coordinate crisis intervention and peer counseling services for people with AIDS or HIV related illnesses, their families and friends.

Reimbursement

Designated AIDS centers may choose to bill Medicaid, when appropriate, under the standard New York State diagnosis related groups (DRGs) for AIDS and HIV related illnesses, and the accompanying alternate level of care (ALC) rate, or under an exempt unit AIDS center per diem and the associated ALC rate. The discrete reimbursement rates are designated to support enhanced inpatient, outpatient and case management services for people with AIDS and HIV related illnesses.

In addition, the Department of Health has developed a seven tier outpatient reimbursement system for AIDS centers that have eliminated the $60 maximum Medicaid reimbursement for outpatient care. AIDS centers are reimbursed for seven types of visits. Each visit type represents a different range and intensity of clinical services. Rates for AIDS center outpatient visits range from $264.14 for an initial visit, which includes a comprehensive history and physical examination, billable only once in a patient's lifetime, to $33.54 for a therapeutic visit, at which previously determined clinical information is explained to the patient. Other visit types are the comprehensive visit, which includes the yearly physical examination and update of medical history, the intermediate visit, at which new problems or marked changes in existing problems are addressed, and routine visits, where established patients are seen for follow-up. The final two visit types involve infusion of blood or chemotherapy.

Current analysis suggests that AIDS center models of care may be associated with reductions in length of stay and other positive patient outcomes. Continued monitoring of quality of care and analysis of the effectiveness of AIDS care models will be critical as the number of AIDS centers increase to an expected 25–30 centers in 1989, providing approximately 60% of New York's hospital care for patients with AIDS and HIV related illnesses.

SELLING OR BUYING A MEDICAL PRACTICE

By Gary R. Shaub, MBA. 250 pp, illustrated. Oradell, NJ, Medical Economics Books, 1988. $31.95 (paperback)

Although recent surveys from a variety of sources fail to document a massive repudiation of medicine by seasoned practitioners, one might be forgiven for believing that doctors approaching the end of their professional careers might be more inclined to get out while the getting is good than formerly. Perhaps this is what motivated the book, *Selling or Buying a Medical Practice*, by Gary R. Schaub. Schaub is a health care management consultant, who has appraised and/or sold over $25,000,000 worth of practices, by his own estimate. He has written the classic "how-to" book, which is obviously intended less as a "do-it-yourself" manual than a cautionary tale to convince would-be practice-sellers not to be dumb and try to do it themselves. After reading this step-by-step consideration of the selling process, from gleam in the eye to golf course in Florida, one is tempted to arrive at that very conclusion. The pitfalls are numerous, the stakes high, and the details tedious and full of bear traps.

Although repetitive, over-long, a bit paternalistically preachy, and oversimplified, the book is worth reading. The author has a sense of humor, organizes his book well, formats it in that inimitable *Medical Economics* style of publishing, and delivers what he promises. His words of wisdom are sincere, make sense, and do not challenge the credibility of either party. He adequately documents the paradox of confidentiality, in which, if the physician blabs his intent, he ruins his practice, but without which he may not sell to a likely community prospect. He reasonably points out that many physicians foolishly close up their practices, making no effort to sell, taking for granted much that they have worked years to build, and squandering resources never fully reckoned with. He gives tips for building a practice, rather than letting it decline as the golden years approach—the better to enhance its value to a newcomer. He talks about the emotional reasons hampering sensible decisions by physicians who should be thinking about these things at least two years in advance of sale. The book would have been enhanced by weightier considerations of the emerging note of managed care when practices are bought and sold. It also lacks a discussion of the ethical problems and procedures around transfer of confidential material. Finally, no information is given on how to find the appraisers and brokers the author recommends (except, of course, himself). These are easily found in the classified advertising of selected reputable journals. All in all, however, this is a useful, helpful, quick-read book for any practitioner over 50. It's also the sort of book one would wish to pass on to a friend in need, rather than retain on the bookshelves with other more clinical reading material.

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INTERVENTIONAL RADIOLOGY

Edited by Wilfrido R. Castaneda-Zuniga, MD, and S. Murthy Tadavarthy, MD. 873 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1988. $149.95 (hardcover)

This book represents the newest addition to the excellent Golden's Diagnostic Radiology series. It covers the entire gamut of interventional radiology procedures with the exclusion of neuroradiology. The text has more than 50 authors but is primarily written by interventional radiologists at the University of Minnesota. As a result, there is very little of the redundancy commonly seen in multiauthored texts.

The book begins with an overview of interventional radiology and then radiation protection. Additional topics described include embolotherapy, the diagnosis and management of gastrointestinal bleeding, transluminal angioplasty, management of vascular access, fibrinolysis, intravascular foreign body removal, inferior vena caval filters, percutaneous uroradiological and biliary procedures, management of enteric strictures, interventional radiology procedures in the chest, percutaneous biopsies, and drainage of abdominal abscesses.

Each chapter is well organized with excellent illustrations. The reference lists are extensive. In summary, this book should become *the* reference for interventional radiology. No other text is as up to date and comprehensive. It should be of value to all radiologists performing interventional procedures, as well as urologists, gastroenterologists, and surgeons who commonly refer their patients for these procedures. Interventional radiology is a rapidly evolving field. This text represents the best summary of the field as it stands today.

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RENEWING THE PROMISE: MEDICARE AND ITS REFORM

Edited by David Blumenthal, Mark Schlesinger, and Pamela Brown Drumheller. 215 pp. New York, Oxford University Press, 1988. $29.95 (hardcover)

In 1912 Theodore Roosevelt was campaigning for President on the Progressive Party ticket. His political party platform proposed the adoption of a comprehensive national health insurance program. That action marked the earliest known precursor of the Medicare program in this country. In an anomaly of history the American Medical Association actually supported the concept of a national health insurance plan in 1916 and 1917.

On July 30, 1965, after a turbulent 53 years of ups and downs, Title XVIII (Medicare) was legislatively mandated and signed into law by President Lyndon Johnson. President Johnson flew to Independence, Missouri, to sign the law and to present ex-President Harry Truman with the first Medicare card.

A critical history of the inception, development, and future of the Medicare program is presented in eight of the nine chapters of this edited book about renewing the sanguine promise held out to the elderly by the lofty goals of the Medicare legislation. These chapters explore the beginnings of Medicare, the record to date, beneficiary cost sharing, coverage provided, hospital payments, physician reimbursement, managed health care plans, and the government's role in the funding of the program. In the last chapter all the recommendations and options are tied together in a consideration of a comprehensive, long term approach to the future of Medicare over the next ten to 15 years. Appendix 9-A estimates the costs and savings from the proposed reform recommendations cited in chapter nine. An index helps to locate specific individuals and entities for those concerned with particular events in the political and social history of Medicare.

This book evolved from a two-year study of the program by the Harvard Medicare Project. Interestingly, the authors avoided an overwhelming emphasis on cost containment in their individual analyses. Instead, all the contributors aimed to discover how to improve the Medicare program for the benefit of the elderly. There was a focus on potential improvements that required few, if any, additional expenditures. Additionally, the contributors were able to avoid the temptation of piecemeal patching of the Medicare program and to take a comprehensive view of the ultimate effort. Participants in the project and contributing authors are identified at the back of the book.

Each of the initial eight chapters provides the historic background as well as insights into the specific subject matter being discussed. There are abundant references at the end of each chapter as well as explanatory notes when required. Furthermore, each chapter discusses the advantages and disadvantages of the programmatic aspects of Medicare administration, the potential for reform, and possible reform options.

In its conclusion, the Harvard Medicare Project opted for providing for more options within the existing Medicare program and for encouraging public and private sectors to take a cooperative approach. Within this framework the government must strive to achieve three goals; an essential containment of Medicare expenditures; provision of fairness to the elderly, to health care providers, and to others affected by the program; and administrative simplification of Medicare so that the elderly and the providers can understand it.

Reforms directly affecting Medicare beneficiaries included a decrease in copayments, an increase in premiums, adjusting the cost burden according to ability to pay, a public supplementary insurance policy to meet gaps in Medicare, expanded coverage of long term care and chronic illness, improved primary care, and administrative simplification.

Reforms in the payment of providers encompassed methods for paying physicians, methods for reimbursing hospitals, and the utilization of prepaid health care groups such as health maintenance organizations (HMOs).

Implementation activities are developed in three phases of one to five years; five to ten years; and ten to 15 years. Each phased movement projects changes in each reform area: cost sharing; service coverage; simplification; physician payment; hospital payment; prepaid health plans; and financing of the program. For example, physician payment begins with mandatory assignment of inpatient charges to the adoption of a relative value scale to mandatory assignment of all charges.

In 1985 dollars the Harvard Medicare Project anticipates that by the year 2000, their reform proposals could range from a net savings of several billion dollars to a net loss of about $3 billion.

In view of the prominent sponsorship of the Harvard Medicare Project and the obvious critical issue of providing comprehensive health care for a graying America, this book is highly recommended. In one location readers can find the oftimes insightful history of a health care movement as well as the specifics of operational activities.

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CHINESE MEDICINE: ITS HISTORY. PHILOSOPHY & PRACTICE, & WHY IT MAY ONE DAY DOMINATE THE MEDICINE OF THE WEST

By Manfred Porkert, MD, with Dr Christian Ullmann. 318 pp. New York, William Morrow & Co, 1988. $18.95 (cloth)

Modern medicine is characterized by specialization and subspecialization. Specialization is often organ based and provides effective care for specific organ diseases through high technology. However, specialization increases the cost of medical care and often results in the fragmentation of health services. In addition, most health problems encountered in the community do not require highly specialized care and many patients (30–40%) visiting physicians' offices have problems that are of functional origin without specific organ pathology. Chinese medicine, based on the functional principle, offers an alternative health care approach to the specialization of modern Western medicine. Its emphasis on health maintenance, humanism, and holistic integration is complementary to specialization. The authors cite acupuncture as an example to illustrate the usefulness and effectiveness of Chinese medicine in successfully managing common health problems in the community. The success of acupuncture therapy has attracted many Western physicians and scholars to study Chinese medicine in recent years. Many Western physicians find that Chinese medicine offers different approaches to identifying clinical problems and managing them in its own highly systematized fashion, often as effectively as modern medical science.

By having studied Chinese medicine for more than 20 years, the author, Manfred Porkert, MD, recognizes the difficulties for Western physicians in studying Chinese medicine. Many original articles are written in classical Chinese and even Chinese intellectuals find them difficult to understand. Dr Christian Ullmann, the co-author of the book, observes that Chinese medicine is deeply rooted in Chinese cultural heritage through its 2,000 years of history. Chinese medicine is a reflection of Chinese philosophy, which is interwoven with the daily life of the Chinese people. Chinese medicine adopts the Yin-Yang theory from Chinese philosophy as its fundamental principle. Health is viewed as a state of equilibrium between the two opposite yet complementary Yin-Yang forces. This Yin-Yang theory then governs the diagnostic and treatment approaches in Chinese medicine, which certainly is difficult for Western physicians or scholars to comprehend. However, as experienced by the authors, once these theoretical backgrounds are understood, the study of Chinese medicine soon becomes a fascinating, intellectual challenge and a rewarding exercise.

The authors introduce the interesting concept of the language barrier which may in fact prevent many Western scholars from studying Chinese medicine. They observe that language, like a binding force, holds us captive to our own cultural heritage. Consequently, in contrast to their Western counterparts, practitioners of Chinese medicine, by using different language, arrive at somewhat different approaches through observations of similar symptoms and signs in their patients. To help Western scholars and physicians in overcoming language barriers, cultural differences, and historical misunderstandings, the authors lucidly present the theoretical fundamentals, diagnostic approaches, and treatment modalities of Chinese medicine. New terminology is introduced. Many new terms may puzzle veteran readers who are already accustomed to the established older terms, but these may find greater acceptability with readers who are just starting a career in Chinese medicine. The authors claim that Chinese medicine is a full-fledged scientific discipline and its future integration into modern medicine is in order. To do this, further control studies are needed to support the theoretical claims which are developed largely through observation, experience, induction, and synthesis.

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COLOUR ATLAS OF SEXUALLY TRANSMITTED DISEASES

By Professor L. K. Bhutani. 160 pp, illustrated. St Louis, Mo, Ishiyaku EuroAmerica, Inc, Publishers, 1988. $42.50 (hardcover)

The purpose of this book is to provide the clinical practitioner of venereology with an authoritative collection of excellent photographs to ease the task of identifying and diagnosing sexually transmitted diseases. The author is Professor and Head of the Department of Dermatology and Venereology at the All India Institute of Medical Sciences in New Delhi, India. Atlases of venereology are not often published, and few are easily available. Consequently, any new addition in this area is definitely welcome.

Included in this atlas are syphilis (all stages), gonorrhea, nongonococcal urethritis, and nonspecific genital infections, such as chancroid, donovanosis, lymphogranuloma venereum, herpes progenitalis, genital warts, pediculosis, scabies, and acquired immunodeficiency syndrome. A special section is devoted to nonvenereal genital conditions, such as lichen sclerosis and atrophicus. Unfortunately, this section also includes molloscum contagiosum, candidiasis, and tinea versicolor, which are often, if not usually, sexually transmitted in adults.

Most of the photographs in this atlas are of good quality and representative of what the clinician is likely to see in actual practice. Some, however, suffer from poor resolution and definition (photo 12, of macular syphilis, photo 40, of a palatal gumma, photo 51 of gonococeal balanitis, photo 53, of gonococcal epididymo-orchitis, and others), and are consequently of little value.

An even more serious problem for the American venereologist stems from the treatment regimens recommended by the authors. For a book published in 1988 to vary so markedly in its therapeutic approach to sexually transmitted diseases from the recommendations of the Centers for Disease Control is most distressing, and likely to be a major source of confusion to its readers. For example, although spectinomycin is mentioned as a treatment for infections "refractory to penicillin," the phrase "penicillinase producing *Neisseria gonorrheae*" is not even mentioned, nor is the place of cephalosporin therapy in resistant gonorrhea. Erythromycin, the drug of choice for the treatment of chancroid in the United States, is not even mentioned in the chapter on chancroid. Instead, sulfonamides are recommended, with tetracycline, streptomycin, and metronidazole as alternatives.

The section on nonvenereal diseases of the genitals is uneven and poorly selected, with common problems such as psoriasis and eczema omitted. As most of the photos are excellent, however, this book can definitely be recommended for use as an atlas.

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DRUG INTERACTIONS INDEX

Second Edition. By Robert T. Weibert, PHARM D, and William A. Norcross, MD. 374 pp. Oradell, NJ, Medical Economics Co, Inc, 1988. $27.95 (softcover)

With the continuing increase in the number of prescription and nonprescription medications, it is difficult for a clinician to keep up with potential interactions and practical approaches to managing them. In the second edition of *Drug Interactions Index*, the authors attempt to provide a "quick, practical guide to significant adverse interactions and their clinical implications," with the "emphasis on essential information that is most useful for actual clinical practice." A limited number of significant food-drug interactions are also included.

The book contains four sections: a name index, a number index, the interaction descriptions, and an appendix of generic names by number. These sections contain 3,980 names, 9,580 interaction pairs, and 434 interaction monographs. Using the book requires a series of steps. The practitioner must first look up the drugs in question in the name index to identify their three-digit code number (an arbitrarily assigned even number). He must then locate one of the code numbers in the number index, and scan the list following that number for the number of the second drug. If the second number is there, then the index will refer to an interaction number (an arbitrarily assigned odd number preceded by an "I," eg, I-195), and the reader can then locate the interaction in the interaction description section. In the descriptions, or monographs, the action of the drug listed on the left side of the title is altered by the drug(s) listed on the right side. The monograph also lists the code numbers of all the drugs that react as described; to identify the drug names, the appendix must be used. Included in the monograph are a brief description of the interaction, suggestions for treating, minimizing, and/or preventing adverse effects, and one to three literature citations.

The preface proclaims that the "computer-like precision" of the "unique numbering system" will lead the practitioner to any specific interaction (drug-drug or food-drug) *in seconds*. In actual use this unique numbering system is very cumbersome; locating an interaction in this book generally takes longer than locating the same information in other similar references. The numbering system in this book takes up a significant portion of the 375 pages: the name index is 104 pages, the number index 68 pages, the interaction section 184 pages, and the appendix 18 pages.

The positive aspect of the name index is its extensiveness. It lists a large number of prescription and nonprescription medications, and it also includes the reacting ingredients in combination products. A potential drawback is the inclusion of products that have not been available in the US for several years (eg, Coco-Quinine™, Unitensin™, PAC™ [phenacetin, aspirin, and caffeine], and Quinamm™ [ingredients listed as quinine and aminophylline]). There are also several editing errors. The most obvious one is the incorrect food-drug interaction number listed for *every* theophylline and aminophylline product.

The number index contains only code numbers: to identify the drug associated with a particular code number, the appendix must be used. In using the number index it should not matter which of the two (or more) code numbers is located first, the other number should be listed after the first number if there is an interaction description. In a few of the cases checked, it did matter which number was located first. With almost 9,600 interacting pairs in the number index, it was not feasible to verify each pair; therefore, the extent of this problem is not clear.

Although the interaction information occupies only half of the book, it is the most important section. The concept of providing practical information to practitioners for managing drug interactions is a good one; however, there are a number of problems (some minor, some major) that limit the authors' achievement of this goal. For instance, the authors have included the interactions that in their "best judgment" are of the "greatest significance for the practitioner." Unlike other drug interaction books, which provide a rating scale or definition of clinical significance, this book does not provide this information.

Another potential problem is the over-generalization of some management suggestions, particularly the overuse of "avoid this combination." While this advice is appropriate for some interactions, it is not always feasible. The authors could have included additional practical suggestions for monitoring or dosage adjustments. Furthermore, some monographs are not as complete as they should be, and therefore could be misleading. For example, the monograph on aminoglycosides and inhalation anesthetics mentions neuromuscular blockade, but not nephrotoxic effects; the monograph on nondepolarizing muscle relaxants and aminoglycosides suggests observing the patient for respiratory depression, but does not discuss management of the respiratory depression.

There are a number of inconsistencies. The term "calcium blockers" is used, but so is "calcium channel blockers." Likewise, "beta blockers" is used, but so is "beta-adrenergic blockers." Potential alternative agents are not consistently suggested. For example, ranitidine and famotidine are suggested as alternatives to cimetidine for patients taking oral anticoagulants, but neither agent is suggested as a possible alternative for patients taking benzodiazepines. Also, the reference cited is not always the most appropriate or significant article dealing with a particular interaction.

Conceptually, this book would be useful for practitioners who need general practical information on drug interactions without detailed discussions of the pharmacology or mechanism. Other currently available drug interaction references (eg, *Drug Interactions*, 5th ed; *Evaluations of Drug Interactions; Drug Interaction Facts*) could then be consulted as needed for more detailed information. It should also be pointed out that the more detailed books also suggest practical ways to manage potential drug interactions. Unfortunately for *Drug Interactions Index*, the cumbersome numbering system, the various inconsistencies, and over-generalizations minimize its usefulness.

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WORSE THAN THE DISEASE. PITFALLS OF MEDICAL PROGRESS

By Diana B. Dutton. 528 pp. New York, Cambridge University Press, 1988. $29.95 (hardcover)

Diana Dutton's book is interesting, well researched, and very well written. Although not at all complimentary to physicians, its warning message is important and worthy of documentation. Using four graphic examples, she tells how lax some research laboratories, pharmaceutical manufacturers, and regulatory agencies can be.

The diethylstilbestrol (DES) story is well known but still shocking to read in such detail. The artificial heart experiment, the swine flu fiasco, and the dangers inherent in genetic engineering are strong arguments in favor of her proposal for patient and public involvement in critical medical and ethical decisions on new products and techniques. The reader will be impressed by Dutton's candor, and although slanted against physicians, the facts are difficult to refute and the conclusions are fair and realistic. She demonstrates an excellent understanding of the complexity of the medical dilemmas described.

Physicians whose careers began in the 1940s lived through the period covered in this book. At that time, for example, penicillin was acclaimed as the new "panacea." It was distributed in vials in a wax-like vehicle. One had to heat the substance by rolling the vial in the palms of the hands in order to be able to inject it intramuscularly. It worked miraculously on streptococcal infections as well as gonorrhea and syphilis. It was not a cure-all but well worth the lumpy buttocks it caused. Some of us saw and heard of young women with peculiar vaginal cancers. They had been delivered by mothers who had been exposed to DES. We were witnesses to the thalidomide horror, the dramatic but short-lived experiment with the artificial heart, the swine flu immunization campaign, the premature heart transplants, and the debate over genetic engineering. The latter is a current issue. Some see it as the creation of clones. Others yearn for the golden opportunity to eliminate congenital defects and inherited diseases.

What is the competent and ethical investigator to do? The voices against animal experimentation are loud and persistent yet dampened by the clamor for action and results as, for example, with the acquired immunodeficiency syndrome. Weigh if you will the pressure of one against the other. Patients with incurable diseases are willing to try anything. They complain that government agencies are too slow. Others feel that pharmaceutical companies are too fast. Some believe that double blind experiments are unfair to the placebo group when the results are good, but insist that the procedure be followed when the results are bad.

When knowledge is available but ignored for profit, it is unconscionable and deserving of severe penalties. However, findings are frequently neither black nor white and thousands of patients may be exposed before unpredictable adverse reactions occur. Scientifically controlled experiments on animals with prolonged periods of observation using adequate numbers outweighs by far the rush to publish, the profit motive, and the pressures mounted by an anxious public on behalf of the sick.

Investigators, physicians, regulatory agents and students should read this controversial book. It will certainly stimulate your thought processes.

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FDA expands testing of anti-AIDS drugs

In preliminary testing, dideoxycytidine (ddC) has shown promise in combatting the progression of AIDS. The apparent antiviral activity of ddC at doses low enough to avoid toxic side effects has prompted the Food and Drug Administration (FDA) to expand clinical trials in several patient populations.

Proposed studies include comparing ddC to zidovudine (AZT) in patients with AIDS or AIDS-related complex (ARC), evaluating ddC in patients with AIDS or advanced ARC who cannot tolerate AZT, and judging the effectiveness of ddC in persons with AIDS dementia. Possible future studies of ddC would focus on babies and young children with AIDS and on asymptomatic carriers of HIV at high risk of progressing to AIDS.

The National Institute of Allergy and Infectious Diseases (NIAID) is cosponsoring trials with Hoffmann-La Roche, which holds exclusive worldwide rights to ddC, to determine the safety and activity of alternating therapy with ddC and AZT. Combination regimens of ddC with AZT and ddC with Roferon®-A (recombinant interferon alpha-2a) are in the planning stages.

These expanded trials will provide more information on the association between ddC and peripheral neuropathy, an adverse effect of ddC therapy recognized in earlier studies. Both the FDA (800-874-2572) and Roche (collect at 201-235-2355) can supply details on all current and prospective trials.

Dideoxyinosine (ddI; Bristol Myers Co.) is another promising experimental AIDS drug. Phase I clinical trials to determine the safety of ddI have been completed, and phase II studies of the drug's efficacy will follow. Because of the immediate need for effective antiviral drugs to halt the progression of AIDS in infected individuals, the federal government is considering making ddI available to AIDS patients before the completion of clinical testing.

Pending the recommendations of an FDA advisory committee concerning the structure, management, liability, and other issues of a parallel track distribution program (in which a drug is available for use after phase I safety trials), the Department of Health and Human Services, together with the FDA, National Institute of Allergy and Infectious Diseases, and the drug manufacturer, will determine whether ddI will be available under a parallel track program or under an Investigational New Drug Program (IND; with distribution possible after phase II efficacy trials).

Prophylactic dentistry can help reduce oral complications of cancer therapy

Cancer therapies, including radiation, high-dose chemotherapy, and bone marrow transplantation, can produce complications of the oral cavity such as mucosal inflammation, chronic dry mouth, disintegration of bone, and septicemia (*Dental Products Report*, July/August 1989, p 12). Prophylactic dentistry can substantially reduce the incidence of these complications, which currently affect about 400,000 patients undergoing cancer therapy each year.

A National Institutes of Health consensus panel has recommended screening of all cancer patients to evaluate the need for root planing and scaling to clear away bacteria that could play a role in local or systemic infection. Patients should allow at least three weeks between any needed tooth extractions and the initiation of radiation therapy.

Oral complications are most common among patients receiving radiation therapy for head and neck cancers or those undergoing high-dose chemotherapy for leukemia, which can produce immunosuppression and myelosuppression.

Children and sports: What's healthy, what's not

As another school year begins, students will renew their participation in regular school physical education classes, and many students will opt to participate on school sports teams as well as extracurricular sports activities. Sports programs offer benefits such as physical activity and conditioning, the opportunity to compete, and experience in working with others as a team. All sports, and contact sports in particular, carry the risk of injury to the athlete.

To aid physicians in advising pediatric patients and their parents on issues of pediatric sports medicine, the American Academy of Pediatrics (AAP) has, over the years, formulated policy statements regarding such aspects of sports medicine as infant exercise programs, anabolic steroids and the adolescent athlete, sports and the child with epilepsy, and risks in long distance running for children.

Paul G. Dyment, MD, discusses some of the controversies in pediatric sports medicine in light of AAP viewpoints in a recent issue of *The Physician and Sportsmedicine* (1989; 17:57–71). The "industry" of infant exercise programs has mushroomed recently to include a growing number and variety of exercise classes and even home exercise equipment specifically for infants. Much of the commercialization of the infant exercise market has focused on extolling the benefits of exercise products and classes for an infant's physical fitness and rate of motor development. Dyment notes that most infant exercise programs consist of passive exercises, including stretching and massage, that "do not increase muscle strength or endurance" and are unlikely to affect the rate of motor development. Unless shown to incorporate potentially injurious practices, infant exercise programs should not be discouraged, because they can contribute to increased parent-child interaction and positive emotional consequences.

The AAP does not recommend infant swim classes because of the risks of infectious disease transmission and hyponatremia-induced convulsions that can occur when a child swallows excessive amounts of water. Infant swim classes should adhere to YWCA safety guidelines, which prohibit total submersion of the infant. Group swimming

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NEWS BRIEFS are compiled and written by Vicki Glaser.

lessons are best begun when a child is at least three years old.

The increasing number of catastrophic neck injuries associated with use of a trampoline led the AAP in 1977 to recommend against trampoline use in competitive sports and physical education classes—and most schools heeded the recommendation.

Although the causes of amenorrhea and other menstrual irregularities among some female athletes are not known, these disorders appear to be caused by the physiologic stress associated with sports such as gymnastics, long distance running, rowing, and ballet. Nutrition counseling and increased calorie intake may prove beneficial. Menstrual dysfunction attributed to intense sports activity does not appear to affect the female athlete's fertility once she reduces her level of physical activity.

For many years, it was the AAP's view that "there was no reason to segregate prepubertal boys and girls during athletic competition, but that after the onset of puberty sports should be gender specific because the greater size and strength of the average boy made open competition unfair to girls." Title IX of the 1972 Education Act mandated that schools provide girls and boys with equal athletic opportunities. If a school has one football team, for example, both girls and boys should be allowed to try out for a spot on the team, and, when tested, this right has held up in court. Dyment asserts that, "There is no evidence that a girl is any more susceptible to injury than a male teammate of similar weight, despite her relatively smaller bones and muscle mass." However, because boys in general have greater strength and endurance, they would have an advantage, particularly when competing for positions in contact sports. Therefore, he supports the AAP position of equal support but separate teams for adolescents.

Growing controversy on safety of implants

Medical devices and materials implanted into the human body were once thought of as inert or biocompatible. However, there is now a growing body of evidence to suggest that many implants "can produce chronic and potentially harmful effects on human tissue in some patients" (*New York Times*, July 25, 1989, pp C1,8).

Although the level of risk associated with implants is not yet known, the benefits of implants for medical purposes—primarily prolonging and improving the quality of life—would appear to outweigh any potential, long term risks. Persons who receive implants strictly for cosmetic purposes, particularly at an early age, should consider the medical problems they may face in the future. These include chronic inflammation, infection, blood clots, bone loss, connective tissue disorders, and, rarely, cancer.

Although the findings are still speculative, researchers are expressing concern that implant materials such as silicone, cobalt, and titanium may be associated with tumor development. Metals cobalt and titanium, commonly used in joint implants, can corrode and release small particles of metal into the bloodstream that can then travel to other parts of the body.

The American Academy of Orthopedic Surgeons recently addressed these controversies, documenting only eight reported instances of tumors near the site of joint implants from among the approximately three million joint replacements performed during the past ten years. They conclude that "some concern is warranted," however "the risk appears to be minimal."

The long term risks of implanted materials are of greater concern for younger recipients. "Infants are now being treated with teflon implants for urinary tract disorders, and the average age of women receiving breast implants is 32 years." Complications associated with silicone breast implants include the formation of hard lumps, leaking of the silicone, difficulty in detecting breast cancer, and development of connective tissue diseases.

Perhaps the least troublesome implants are eye lenses, because some of the immune system components responsible for inflammation are not present in the eye.

Little known hazard of drinking beer

For one 36-year-old man, a bottle of beer to be consumed while watching a baseball game led instead to a visit to the emergency room (*N Engl J Med* 1989; 321:121–122). The man's attempt to remove the bottle cap with his teeth led to esophageal obstruction when the compressed gas in the bottle forced the cap down his oropharynx.

Radiographs depicted the cap in the upper third of his esophagus. Use of a rigid esophagoscope was unsuccessful because the serrations were lodged in the mucosa. "The patient subsequently underwent a right thoracotomy, during which the bottle cap, measuring 2.7 cm in diameter and bearing the words 'Miller High Life,' was found at the level of the T4 vertebra just above the azygous vein; it was removed by esophagotomy."

The authors stress the danger of removing bottle caps from carbonated beverages with one's teeth.

Misuse of disposable contact lenses increases risk of corneal infections

The development of disposable contact lenses offered lens wearers the convenience of extended wear without the need for periodic cleaning of their lenses. However, ophthalmologists have warned that exceeding the recommended lifetime of disposable contact lenses will increase the wearer's risk of serious corneal infections (*JAMA* 1989; 261:3549–3550).

Robert M. Kershner, MD, described a patient with a corneal ulcer infected with *Pseudomonas* sp who wore disposable contact lenses for 2½ weeks at a time. The patient had been wearing disposable lenses comfortably for ten months, at which time he began gradually to increase the wearing time. He sought treatment for symptoms of redness, pain, and loss of vision.

Examination revealed a 1-mm deep corneal ulcer, and culture of the lesion grew *Pseudomonas* sp. Topical aminoglycoside therapy gradually led to healing. The patient was left with a small, central scar, and his vision was not correctable to 20/20.

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Francis A. Agrest, MD, Buchanan. Died June 1, 1989; age 81. Dr Agrest was a 1934 graduate of the State University of New York Health Science Center at Brooklyn. He was a Diplomate of the American Board of Internal Medicine. He was a member of the New York Academy of Medicine, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Leonard J. Barron, MD, Binghamton. Died June 4, 1989; age 76. Dr Barron was a 1937 graduate of the University of Toronto Faculty of Medicine. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Orthopedic Surgery. His memberships included the American Academy of Orthopaedic Surgeons, the Broome County Medical Society, and the Medical Society of the State of New York.

Hugh D. Black, MD, Naples, Florida. Died May 8, 1989; age 69. Dr Black was a 1945 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a Fellow of the American College of Anesthesiologists. He was a member of the Medical Society of the County of Chenango and the Medical Society of the State of New York.

Arnold G. Blumberg, MD, Glen Oaks. Died June 18, 1989; age 69. Dr Blumberg was a 1945 graduate of the New York University School of Medicine. He was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Samuel Herbert Bumble, MD, Brooklyn. Died February 15, 1989; age 89. Dr Bumble was a 1928 graduate of the New York University College of Medicine. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Remo Cancellieri, MD, Bronx. Died June 11, 1989; age 82. Dr Cancellieri was a 1932 graduate of the New York University School of Medicine. He was a Fellow of the American College of Angiology and a Diplomate of the American Board of Internal Medicine. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Armando H. Cucchiarella, MD, Scarsdale. Died May 25, 1989; age 65. Dr Cucchiarella was a 1954 graduate of the Columbia University College of Physicians and Surgeons. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Nabil D. El Hassan, MD, Syracuse. Died April 17, 1989; age 46. Dr El Hassan was a 1970 graduate of the Medical School, American University of Beirut, Beirut, Lebanon. He was a Fellow of the American College of Obstetricians and Gynecologists. He was a member of the American Fertility Society, the Onondaga County Medical Society, and the Medical Society of the State of New York.

Gerald Jay Epps, MD, Brooklyn. Died June 15, 1989; age 80. Dr Epps was a 1938 graduate of the Leopold Franzens Universitaet Medizinische Fakultaet, Innsbruck, Germany. His memberships included the American Academy of Family Physicians, the American Geriatrics Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Leo Fahnrich, MD, Valley Stream. Died June 16, 1989; age 75. Dr Fahnrich was a 1943 graduate of the Middlesex University School of Medicine, Waltham, Massachusetts. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Abraham H. Friedman, MD, New York. Died August 27, 1988; age 85. Dr Friedman was a 1930 graduate of the New York University School of Medicine. He was a member of the Clinical Society of the New York Diabetes Association, the New York Cardiological Society, the American Geriatrics Society, the New York County Medical Society, and the Medical Society of the State of New York.

Sawnie Renard Gaston, MD, Piermont. Died April 15, 1989; age 80. Dr Gaston was a 1935 graduate of Yale University School of Medicine. His memberships included the New York Society for Clinical Ophthalmology, the New York County Medical Society, and the Medical Society of the State of New York.

H. Charles Gerard, MD, New York. Died October 26, 1988; age 92. Dr Gerard was a 1922 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a member of the New York Society for Clinical Ophthalmology, the New York County Medical Society, and the Medical Society of the State of New York.

Barry David Gingell, MD, New York. Died April 29, 1989; age 35. Dr Gingell was a 1980 graduate of the New York University School of Medicine. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Charles Glassman, MD, Southbury, Connecticut. Died June 8, 1989; age 86. Dr Glassman was a 1934 graduate of the Faculty of Medicine University of Aberdeen, Scotland. He was a member of the American Academy of Family Physicians, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Louis Greenstein, MD, Newton, Connecticut. Died April 19, 1989; age 89. Dr Greenstein was a 1924 graduate of the Columbia University College of Physicians and Surgeons. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the New York Neurological Society, the American Academy of Neurology, the American Psychiatric Association, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Louis E. Itzkowitz, MD, Brooklyn. Died June 10, 1989; age 79. Dr Itzkowitz was a 1937 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the American Academy of Family Physicians, the Kings County Medical Society, and the Medical Society of the State of New York.

Leon Kahan, MD, Flushing. Died June 18, 1989; age 82. Dr Kahan was a 1933 graduate of the New York Medical College. He was a member of the American Academy of Family Physicians, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Frances Kardons, MD, New York. Died May 17, 1989; age 91. Dr Kardons was a 1921 graduate of the New York Medical College. She was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Charles Klepetar, MD, Delhi. Died June 22, 1989; age 79. Dr Klepetar was a 1934 graduate of the Charles Univerzita Fakulta of Ped-Gen Medicine, Praha, Czechoslovakia. He was a member of the Delaware County Medical Society and the Medical Society of the State of New York.

Swen L. Larson, MD, Elmira. Died June 19, 1989; age 86. Dr Larson was a 1930 graduate of the Georgetown University School of Medicine, Washington, DC. He was a Fellow of the American College of Chest Physicians and the American College of Surgeons. He was a member of the Medical Society of the County of Chemung and the Medical Society of the State of New York.

Samuel Kantor Levy, MD, Suffern. Died June 11, 1989; age 95. Dr Levy was a 1916 graduate of the State University of New York Health Science Center at Brooklyn. He was a Diplomate of the American Board of Pediatrics. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Frederick M. Margaretten, MD, Elizabeth, New Jersey. Died May 4, 1989; age 93. Dr Margaretten was a 1921 graduate of the New York Medical College. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Arthur R. Poole, MD, Far Rockaway. Died April 5, 1989; age 59. Dr Poole was a 1959 graduate of the Facolta di Medicina e Chirurgia dell'Universita di Roma, Rome, Italy. He was a Fellow of the American College of Anesthesiologists and a Fellow of the American College of Chest Physicians. His memberships included the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the New York County Medical Society, and the Medical Society of the State of New York.

Sylvester H. Pratt, MD, Lockport. Died May 4, 1989; age 61. Dr Pratt was a 1957 graduate of the Indiana University School of Medicine, Indianapolis. He was a member of the American Academy of Occupational Medicine and the American Academy of Family Physicians. He was a member of the Medical Society of the County of Niagara and the Medical Society of the State of New York.

Victor H. Raisman, MD, Kew Gardens. Died May 29, 1989; age 84. Dr Raisman was a 1929 graduate of the New York University School of Medicine. He was a Diplomate of the American Board of Orthopedic Surgery and a Qualified Fellow of the International College of Surgeons. His memberships included the American Academy of Orthopaedic Surgeons, the Pan American Medical Association, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Arthur Reich, MD, Crawfordville, Florida. Died July 23, 1988; age 77. He was a Diplomate of the American Board of Anesthesiology and a Fellow of the American College of Anesthesiologists. His memberships included the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the Suffolk County Medical Society, and the Medical Society of the State of New York.

Ernest Welton Saward, MD, San Jose, California. Died June 5, 1989; age 75. Dr Saward was a 1939 graduate of the University of Rochester School of Medicine-Dentistry. He was a Diplomate of the American Board of Internal Medicine and a Fellow of the American College of Preventive Medicine. He was a member of the Medical Society of the County of Monroe and the Medical Society of the State of New York.

Francis G. Scheider, MD, North Syracuse. Died May 19, 1989; age 58. Dr Scheider was a 1958 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a Diplomate of the American Board of Pediatrics. He was a member of the Onondaga County Medical Society and the Medical Society of the State of New York.

Harry Schwippert, MD, Eden. Died May 13, 1989; age 67. Dr Schwippert was a 1948 graduate of the University of Rochester School of Medicine-Dentistry. He was a Diplomate of the American Board of Surgery and the American Board of Thoracic Surgery and a Fellow of the American College of Surgeons and the American Board of Surgery. His memberships included the American Occupational Medical Association, the American Academy of Occupational Medicine, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Michael Slovak, MD, Schenectady. Died May 8, 1989; age 86. Dr Slovak was a 1929 graduate of the Albany Medical College of Union University. He was a Fellow of the American College of Surgeons. He was a member of the Medical Society of the County of Schenectady and the Medical Society of the State of New York.

Abraham Weber, MD, Middletown, New Jersey. Died May 28, 1989; age 89. Dr Weber was a 1923 graduate of the State University of New York Health Science Center at Brooklyn. He was a Fellow of the American College of Angiology. His memberships included the New York Cardiological Society, the Clinical Society of the New York Diabetes Association, the New York State Society of Internal Medicine, the American Geriatrics Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Saul Weinstein, MD, Ft Lauderdale, Florida. Died October 8, 1988; age 80. Dr Weinstein was a 1932 graduate of the University of Colorado School of Medicine, Denver. He was a member of the American Academy of Family Physicians, the Bronx County Medical Society, and the Medical Society of the State of New York.

Irving G. Werner, MD, Binghamton.

Died May 26, 1989; age 85. Dr Werner was a 1929 graduate of the University of Vermont College of Medicine, Burlington. He was a Diplomate of the American Board of Obstetrics and Gynecology and a Fellow of the American College of Surgeons and the American College of Obstetricians and Gynecologists. He was a member of the New York Academy of Medicine, the Broome County Medical Society, and the Medical Society of the State of New York.

Jane V. Williams, MD, Yonkers. Died April 2, 1989; age 65. Dr Williams was a 1949 graduate of Howard University College of Medicine, Washington, DC. She was a Diplomate of the American Board of Preventive Medicine, Inc, and a Fellow of the American College of Preventive Medicine. She was a member of the American Public Health Association, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Stanley L. Wallace, MD, FACP
1923–1989

Dr Stanley L. Wallace, Professor of Medicine and Chief of the Division of Internal Medicine at the State University of New York Health Science Center at Brooklyn, died June 20, 1989. He was 65 years old.

Dr Wallace was born in Jersey City, New Jersey, on November 21, 1923. In 1943 he was graduated magna cum laude from Duke University, where he was elected to Phi Beta Kappa. He then received his medical education at Bowman Gray Medical College. That was followed by an internal medicine residency at Metropolitan Hospital, Goldwater Memorial Hospital, Montefiore Hospital, and Maimonides Hospital. After completing a fellowship in rheumatology at Columbia University College of Physicians and Surgeons and two years as a captain in the United States Air Force, Dr Wallace embarked on a flourishing academic career at the State University of New York Health Science Center at Brooklyn, where he became Professor of Medicine in 1968. He held a number of leadership positions and, at the time of his death, was Chief of the Division of Internal Medicine, Chief of the Division of Rheumatology, and Director of Medicine.

Dr Wallace was a highly regarded teacher, clinician, and research scientist. He achieved eminence in his chosen specialty of rheumatology, and published more than 100 papers on gout, purine metabolism, rheumatoid arthritis, and the rheumatic diseases generally. He chaired many committees of the American Rheumatism Association and was President of the New York Rheumatism Association.

Dr Wallace's contributions to the American College of Physicians (ACP) included 30 years of participation in advisory committees and postgraduate courses. He was a founder of the New York State Chapter in 1982 and was its President in 1986–1987. He served as Governor for the New York Downstate II Region of the ACP from 1984 to 1988. In March 1989, Dr Wallace was honored at the Annual Meeting of the New York State Chapter where he was given its award for Service to Medicine.

Throughout his years as an academic leader, Dr Wallace remained devoted to patients and students. He is remembered by many as a skilled, wise, and humane physician who enjoyed encouraging young people to enter and advance in the profession he loved so much.

Dr Wallace is survived by his wife, Eleanor; three sons, Jonathan, Richard, and Joseph; and a grandson.

MARTIN LIEBOWITZ, MD, FACP
Vice Chair, Department of Medicine
Health Sciences Center, SUNY at Stony Brook

Governor, Downstate Region II
New York Chapter, American College of Physicians

(*continued from p 5A*)

Louis. Contact: Marion Kenn, Administrative Director, SCEH, 128A Kings Park Dr, Liverpool, NY 13090. Tel: (315) 652-7299.

NEBRASKA

Oct 2–7. **Emergency Medicine Review.** Omaha. *Oct 20–21.* **Etiology of Breast Cancer.** Peter Kiewit Conference Center, Omaha. Contact: Brenda C. Ram, Continuing Education, University of Nebraska Medical Center, 42nd & Dewey Ave, Omaha, NE 68105. Tel: (402) 559-4152.

SOUTH CAROLINA

Oct 26–28. **Fall Update in Geriatrics.** 24 Cat I Credits. Sheraton Hotel, Charleston. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077-1925.

TENNESSEE

Oct 12–15. **Flexible Fiberoptic Sigmoidoscopy and Naso-pharyngo-laryngoscopy in the Primary Care Physician's Office.** Opryland Hotel, Nashville. Contact: Seminars & Symposia, Inc, 132 E 70th St, New York, NY 10021. Tel: (212) 517-7520.

TEXAS

Oct 15–18. **Medical Oncology: A Comprehensive Review.** Houston. Contact: Richard Pazdur, MD, PO Box 78, 1515 Holcombe Blvd, Houston, TX 77030. Tel: (713) 792-2828.

AROUND THE WORLD

ITALY

Oct 18–27. **Allergy Abroad—Italy.** Padua, Florence, & Rome. Contact: Washington University School of Medicine, Office of Continuing Education, 660 S Euclid, Box 8063, St Louis, MO 63110. Tel: (800) 325-9862. □

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Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

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Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

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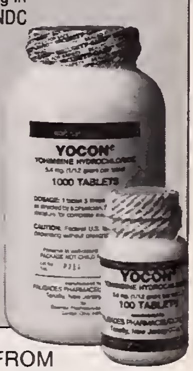
Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.^{1,3,4} 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.^{3}

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:
1. A. Morales et al., New England Journal of Medicine: 1221, November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

Rev. 1/85



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Please see adjacent page for brief summary of prescribing information.

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INDICATIONS AND USAGE: For the relief of moderate to moderately severe pain.

CONTRAINDICATIONS: Hypersensitivity to acetaminophen or hydrocodone.

WARNINGS:

Allergic-Type Reactions: VICODIN/VICODIN ES Tablets contain sodium metabisulfite, a sulfite that may cause allergic-type reactions including anaphylactic symptoms and life-threatening or less severe asthmatic episodes in certain susceptible people.

Respiratory Depression: At high doses or in sensitive patients, hydrocodone may produce dose-related respiratory depression.

Head Injury and Increased Intracranial Pressure: The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a preexisting increase in intracranial pressure. Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries.

Acute Abdominal Conditions: The administration of narcotics may obscure the diagnosis or clinical course of patients with acute abdominal conditions.

PRECAUTIONS:

Special Risk Patients: VICODIN/VICODIN ES Tablets should be used with caution in elderly or debilitated patients and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture.

Cough Reflex: Hydrocodone suppresses the cough reflex; as with all narcotics, caution should be exercised when VICODIN/VICODIN ES Tablets are used postoperatively and in patients with pulmonary disease.

Drug Interactions: Patients receiving other narcotic analgesics, antipsychotics, antianxiety agents, or other CNS depressants (including alcohol) concomitantly with VICODIN/VICODIN ES Tablets may exhibit an additive CNS depression. The use of MAO inhibitors or tricyclic antidepressants with hydrocodone preparations may increase the effect of either the antidepressant or hydrocodone. The concurrent use of anticholinergics with hydrocodone may produce paralytic ileus.

Usage in Pregnancy:

Teratogenic Effects: Pregnancy Category C. Hydrocodone has been shown to be teratogenic in hamsters when given in doses 700 times the human dose. There are no adequate and well-controlled studies in pregnant women. VICODIN/ VICODIN ES Tablets should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nonteratogenic effects: Babies born to mothers who have been taking opioids regularly prior to delivery will be physically dependent. The withdrawal signs include irritability and excessive crying, tremors, hyperactive reflexes, increased respiratory rate, increased stools, sneezing, yawning, vomiting, and fever.

Labor and Delivery: Administration of VICODIN/VICODIN ES Tablets to the mother shortly before delivery may result in some degree of respiratory depression in the newborn, especially if higher doses are used.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk and because of the potential for serious adverse reactions in nursing infants from VICODIN/VICODIN ES Tablets, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS:

The most frequently observed adverse reactions include light-headedness, dizziness, sedation, nausea and vomiting. These effects seem to be more prominent in ambulatory than in nonambulatory patients and some of these adverse reactions may be alleviated if the patient lies down. Other adverse reactions include:

Central Nervous System: Drowsiness, mental clouding, lethargy, impairment of mental and physical performance, anxiety, fear, dysphoria, psychic dependence and mood changes.

Gastrointestinal System: The antiemetic phenothiazines are useful in suppressing the nausea and vomiting which may occur (see above); however, some phenothiazine derivatives seem to be antianalgesic and to increase the amount of narcotic required to produce pain relief, while other phenothiazines reduce the amount of narcotic required to produce a given level of analgesia. Prolonged administration of VICODIN/VICODIN ES Tablets may produce constipation.

Genitourinary System: Ureteral spasm, spasm of vesical sphincters and urinary retention have been reported.

Respiratory Depression: Hydrocodone bitartrate may produce dose-related respiratory depression by acting directly on the brain stem respiratory center. Hydrocodone also affects the center that controls respiratory rhythm, and may produce irregular and periodic breathing. If significant respiratory depression occurs, it may be antagonized by the use of naloxone hydrochloride. Apply other supportive measures when indicated.

DRUG ABUSE AND DEPENDENCE:

VICODIN/VICODIN ES Tablets are subject to the Federal Controlled Substance Act (Schedule III). Psychic dependence, physical dependence, and tolerance may develop upon repeated administration of narcotics; therefore, VICODIN/ VICODIN ES Tablets should be prescribed and administered with caution.

OVERDOSAGE:

Acetaminophen Signs and Symptoms: In acute acetaminophen overdosage, dose-dependent, potentially fatal hepatic necrosis is the most serious adverse effect. Renal tubular necrosis, hypoglycemic coma, and thrombocytopenia may also occur. Early symptoms following a potentially hepatotoxic overdose may include: nausea, vomiting, diaphoresis and general malaise. Clinical and laboratory evidence of hepatic toxicity may not be apparent until 48 to 72 hours post-ingestion.

Hydrocodone Signs and Symptoms: Serious overdose with hydrocodone is characterized by respiratory depression (a decrease in respiratory rate and/or tidal volume, Cheyne-Stokes respiration, (cyanosis), extreme somnolence progressing to stupor or coma, skeletal muscle flaccidity, cold and clammy skin, and sometimes bradycardia and hypotension. In severe overdosage, apnea, circulatory collapse, cardiac arrest and death may occur.

Revised June 1989

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Classified Advertising 6A, 8A, 12A, 13A, 14A
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Knoll Pharmaceuticals 10A, 11A, 12A
Eli Lilly and Company . . . . 2nd Cover
Palisades Pharmaceuticals 9A
Roche Laboratories . . . . 3rd & 4th Cover
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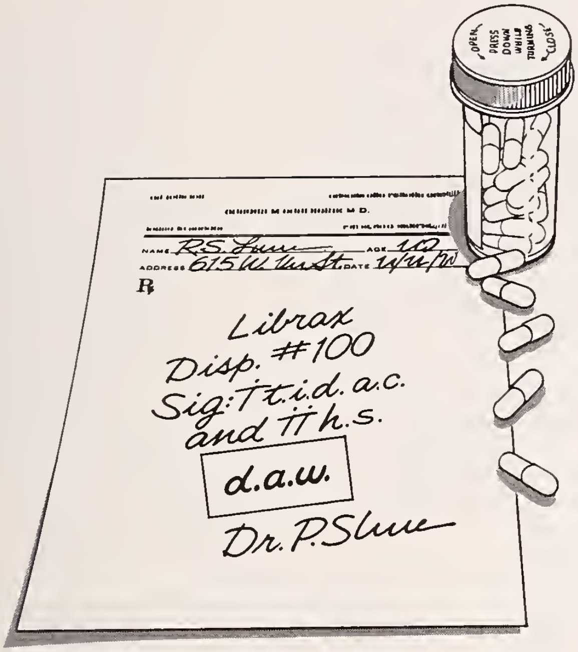

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Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur.

Withdrawal symptoms of the barbiturate type have occurred after discontinuation of benzodiazepines (see Drug Abuse and Dependence).

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established. Inform patients to consult physician before increasing dose or abruptly discontinuing this drug.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

Drug Abuse and Dependence: Withdrawal symptoms similar to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of chlordiazepoxide; more severe seen after excessive doses over extended periods; milder after taking continuously at therapeutic levels for several months. After extended therapy, avoid abrupt discontinuation and taper dosage. Carefully supervise addiction-prone individuals because of predisposition to habituation and dependence.

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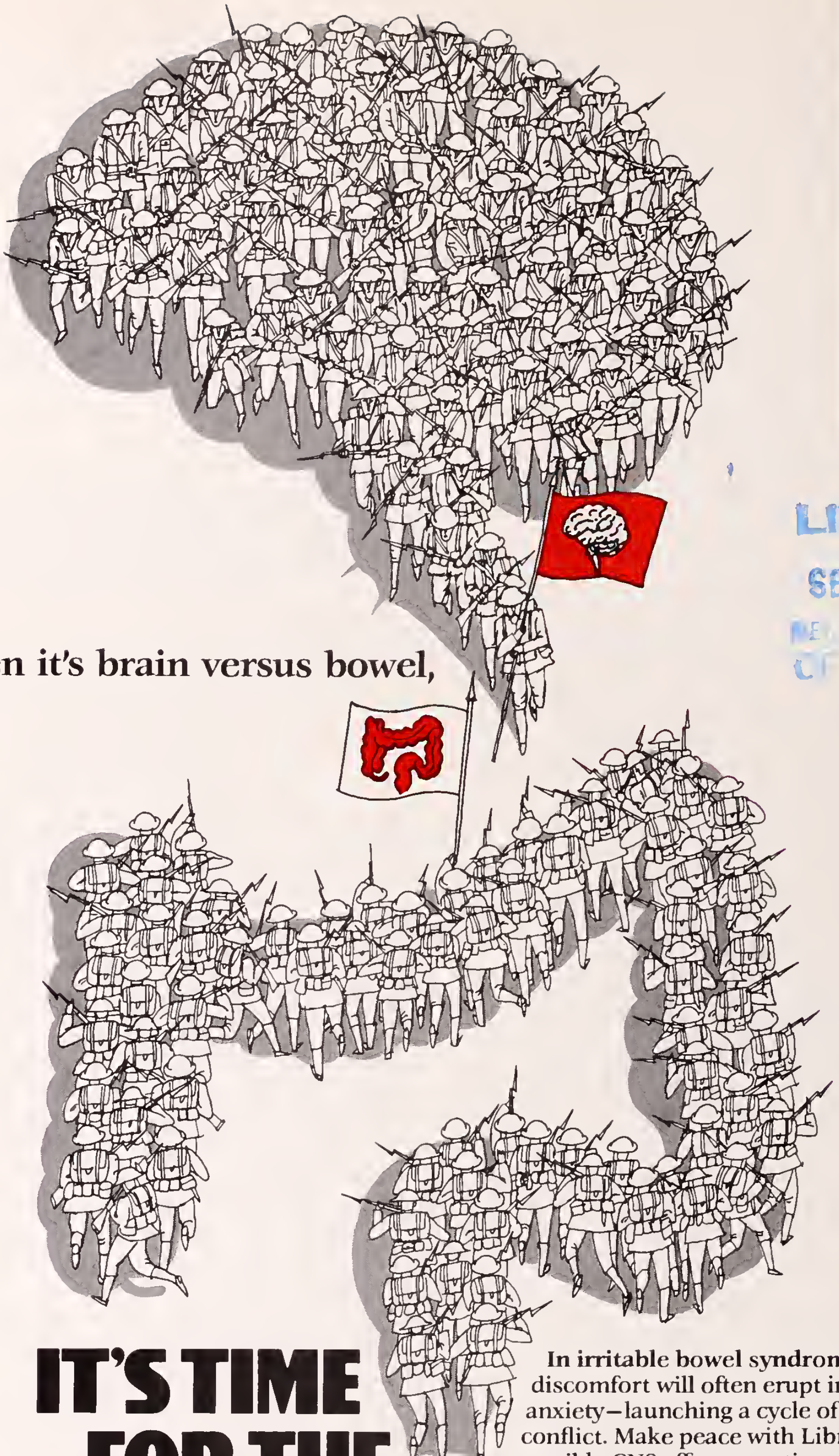

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OCTOBER 1989 • Volume 89, Number 10

Contents

COMMENTARIES

Current challenges to the practice of medicine 553
DONALD F. FOY

The medical malpractice mediation panel 554
RICHARD A. YOUNG, LLB

RESEARCH PAPERS

Mortality patterns among a Native American population in New York State 557
ARTHUR M. MICHALEK, PHD; MARTIN C. MAHONEY, PHD; K. MICHAEL CUMMINGS, PHD, MPH; JOHN HANLEY, MPH, MPS; RAE SNYDER, BS

The awarding of honors to medical clerks: Results of a national survey 562
FRED ROSNER, MD; BERNARD P. SHAGAN, MD; ELEANOR Z. WALLACE, MD

REVIEW ARTICLE

Lyme disease: A review of its clinical features 566
ARTHUR WEINSTEIN, MD, DANUTA I. BUJAK, MSN

SPECIAL REPORT

The confidential New York State Department of Health Universal New York Health Care (UNY*Care) proposal: Medical Society of the State of New York staff observations 572
DONALD F. FOY, MARIO V. MENGHINI

SPECIAL ARTICLE

A 91-year-old daughter's memoir of her physician father, Dr Vacil D. Bozovsky (1865–1933) 576
LOUISE BOZOVSKY DAGGETT

CASE REPORTS

Sarcoidosis of the thyroid clinically mimicking malignancy 578
IRENE A. WEISS, MD; ANJALI LIMAYE, MD; VICTOR TCHERTKOFF, MD; JACOB L. BRENER, MD

Pulsed Doppler-ultrasonic diagnosis of carotid body tumor 580
DENNIS P. TIHANSKY, MD, PHD; PAUL S. PORTER, RDMS

Late onset post-pneumonectomy empyema 582
KAUSHIK A. SHASTRI, MD; MONICA B. SPAULDING, MD

Scrotal ischemia after intravenous vasopressin therapy for hemorrhagic esophageal varices 583
FULL-YOUNG CHANG, MD; JIUNN-TARNG CHENG, MD; KWOK-HUNG LAI, MD; SHOU-DONG LEE, MD; YANG-TE TSAI, MD

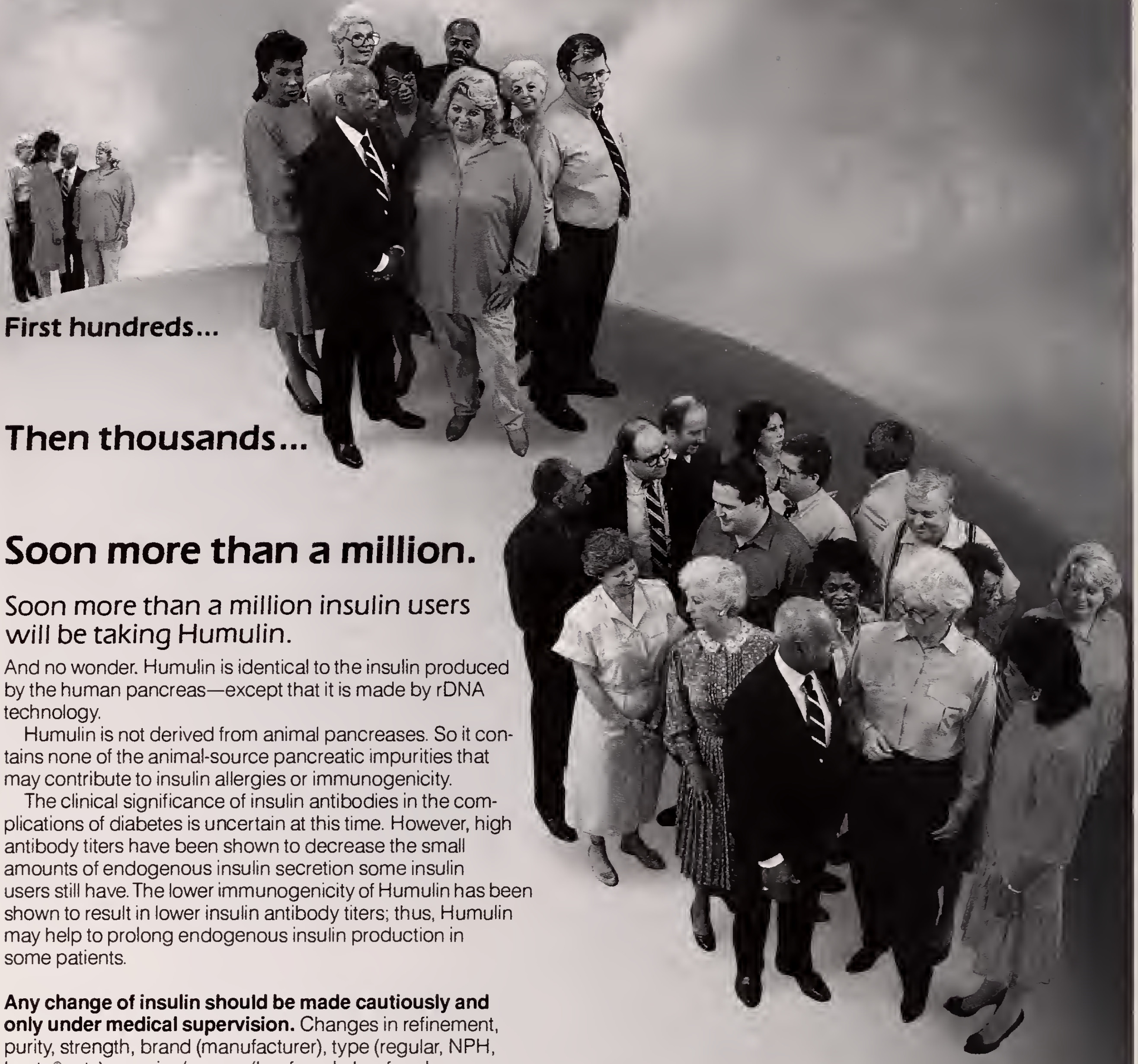
LETTERS TO THE EDITOR

Confidential Tay-Sachs carrier screening 585
FRED ROSNER, MD

Kashin-Beck disease 585
ALLAN J. SOBIN, MD
TSUNG O. CHENG, MD

Medical malpractice 586
JOSEPH R. FONTANETTA, MD

LEADS FROM EPIDEMIOLOGY NOTES 588
BOOK REVIEWS 592
NEWS BRIEFS 597
OBITUARIES 599
MEDICAL MEETINGS AND LECTURES 8A



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If excessive hypotension occurs, the patient should be placed in supine position and, if necessary, receive an intravenous infusion of normal saline. A transient hypotensive response is not a contraindication to further doses of VASOTEC, which usually can be given without difficulty once the blood pressure has stabilized. If symptomatic hypotension develops, a dose reduction or discontinuation of VASOTEC or concomitant diuretic may be necessary.

Neutropenia/Agranulocytosis: Another ACE inhibitor, captopril, has been shown to cause agranulocytosis and bone marrow depression, rarely in uncomplicated patients but more frequently in patients with renal impairment, especially if they also have a collagen vascular disease. Available data from clinical trials of enalapril are insufficient to show that enalapril does not cause agranulocytosis at similar rates. Foreign marketing experience has revealed several cases of neutropenia or agranulocytosis in which a causal relationship to enalapril cannot be excluded. Periodic monitoring of white blood cell counts in patients with collagen vascular disease and renal disease should be considered.

Precautions: *General: Impaired Renal Function:* As a consequence of inhibiting the renin-angiotensin-aldosterone system, changes in renal function may be anticipated in susceptible individuals. In patients with severe heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with ACE inhibitors, including VASOTEC, may be associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death.

In clinical studies in hypertensive patients with unilateral or bilateral renal artery stenosis, increases in blood urea nitrogen and serum creatinine were observed in 20% of patients. These increases were almost always reversible upon discontinuation of enalapril and/or diuretic therapy. In such patients, renal function should be monitored during the first few weeks of therapy.

Some patients with hypertension or heart failure with no apparent preexisting renal vascular disease have developed increases in blood urea and serum creatinine, usually minor and transient, especially when VASOTEC has been given concomitantly with a diuretic. This is more likely to occur in patients with preexisting renal impairment. Dosage reduction and/or discontinuation of the diuretic and/or VASOTEC may be required.

Evaluation of patients with hypertension or heart failure should always include assessment of renal function. (See DOSAGE AND ADMINISTRATION.)

Hyperkalemia: Elevated serum potassium (> 5.7 mEq/L) was observed in approximately 1% of hypertensive patients in clinical trials. In most cases these were isolated values which resolved despite continued therapy. Hyperkalemia was a cause of discontinuation of therapy in 0.28% of hypertensive patients. In clinical trials in heart failure, hyperkalemia was observed in 3.8% of patients, but was not a cause for discontinuation.

Risk factors for the development of hyperkalemia include renal insufficiency, diabetes mellitus, and the concomitant use of potassium-sparing diuretics, potassium supplements, and/or potassium-containing salt substitutes, which should be used cautiously, if at all, with VASOTEC. (See *Drug Interactions*.)

Surgery/Anesthesia: In patients undergoing major surgery or during anesthesia with agents that produce hypotension, enalapril may block angiotensin II formation secondary to compensatory renin release. If hypotension occurs and is considered to be due to this mechanism, it can be corrected by volume expansion.

Information for Patients:

Angioedema: Angioedema, including laryngeal edema, may occur especially following the first dose of enalapril. Patients should be so advised and told to report immediately any signs or symptoms suggesting angioedema (swelling of face, extremities, eyes, lips, tongue, difficulty in swallowing or breathing) and to take no more drug until they have consulted with the prescribing physician.

Hypotension: Patients should be cautioned to report lightheadedness especially during the first few days of therapy. If actual syncope occurs, the patients should be told to discontinue the drug until they have consulted with the prescribing physician.

All patients should be cautioned that excessive perspiration and dehydration may lead to an excessive fall in blood pressure because of reduction in fluid volume. Other causes of volume depletion such as vomiting or diarrhea may also lead to a fall in blood pressure; patients should be advised to consult with the physician.

Hyperkalemia: Patients should be told not to use salt substitutes containing potassium without consulting their physician.

Neutropenia: Patients should be told to report promptly any indication of infection (e.g., sore throat, fever) which may be a sign of neutropenia.

NOTE: As with many other drugs, certain advice to patients being treated with enalapril is warranted. This information is intended to aid in the safe and effective use of this medication. It is not a disclosure of all possible adverse or intended effects.

Drug Interactions:

Hypotension: Patients on Diuretic Therapy: Patients on diuretics and especially those in whom diuretic therapy was recently instituted may occasionally experience an excessive reduction of blood pressure after initiation of therapy with enalapril. The possibility of hypotensive effects with enalapril can be minimized by either discontinuing the diuretic or increasing the salt intake prior to initiation of treatment with enalapril. If it is necessary to continue the diuretic, provide close medical supervision after the initial dose for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and DOSAGE AND ADMINISTRATION.)

Agents Causing Renin Release: The antihypertensive effect of VASOTEC is augmented by antihypertensive agents that cause renin release (e.g., diuretics).

Other Cardiovascular Agents: VASOTEC has been used concomitantly with beta-adrenergic-blocking agents, methyldopa, nitrates, calcium-blocking agents, hydralazine, prazosin, and digoxin without evidence of clinically significant adverse interactions.

Agents Increasing Serum Potassium: VASOTEC attenuates potassium loss caused by thiazide-type diuretics. Potassium-sparing diuretics (e.g., spironolactone, triamterene, or amiloride), potassium supplements, or potassium-containing salt substitutes may lead to significant increases in serum potassium. Therefore, if concomitant use of these agents is indicated because of demonstrated hypokalemia, they should be used with caution and with frequent monitoring of serum potassium. Potassium-sparing agents should generally not be used in patients with heart failure receiving VASOTEC.

Lithium: A few cases of lithium toxicity have been reported in patients receiving concomitant VASOTEC and lithium and were reversible upon discontinuation of both drugs. Although a causal relationship has not been established, it is recommended that caution be exercised when lithium is used concomitantly with VASOTEC and serum lithium levels should be monitored frequently.

Pregnancy—Category C: There was no fetotoxicity or teratogenicity in rats treated with up to 200 mg/kg/day of enalapril (333 times the maximum human dose). Fetotoxicity, expressed as a decrease in average fetal weight, occurred in rats given 1200 mg/kg/day of enalapril but did not occur when these animals were supplemented with saline. Enalapril was not teratogenic in rabbits. However, maternal and fetal toxicity occurred in some rabbits at doses of 1 mg/kg/day or more. Saline supplementation prevented the maternal and fetal toxicity seen at doses of 3 and 10 mg/kg/day, but not at 30 mg/kg/day (50 times the maximum human dose).

Radioactivity was found to cross the placenta following administration of labeled enalapril to pregnant hamsters.

There are no adequate and well-controlled studies of enalapril in pregnant women. However, data are available that show enalapril crosses the human placenta. Because the risk of fetal toxicity with the use of ACE inhibitors has not been clearly defined, VASOTEC® (Enalapril Maleate, MSD) should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Postmarketing experience with all ACE inhibitors thus far suggests the following with regard to pregnancy outcome. Inadvertent exposure limited to the first trimester of pregnancy has not been reported to affect fetal outcome adversely. Fetal exposure during the second and third trimesters of pregnancy has been associated with fetal and neonatal morbidity and mortality.

When ACE inhibitors are used during the later stages of pregnancy, there have been reports of hypotension and decreased renal perfusion in the newborn. Oligohydramnios in the mother has also been reported, presumably representing decreased renal function in the fetus. Infants exposed *in utero* to ACE inhibitors should be closely observed for hypotension, oliguria, and hyperkalemia. If oliguria occurs, attention should be directed toward support of blood pressure and renal perfusion with the administration of fluids and pressors as appropriate. Problems associated with prematurity such as patent ductus arteriosus have occurred in association with maternal use of ACE inhibitors, but it is not clear whether they are related to ACE inhibition, maternal hypertension, or the underlying prematurity.

Nursing Mothers: Milk in lactating rats contains radioactivity following administration of ^{14}C enalapril maleate. It is not known whether this drug is secreted in human milk. Because many drugs are secreted in human milk, caution should be exercised when VASOTEC is given to a nursing mother.

Pediatric Use: Safety and effectiveness in children have not been established.

Adverse Reactions: VASOTEC has been evaluated for safety in more than 10,000 patients, including over 1000 patients treated for one year or more. VASOTEC has been found to be generally well tolerated in controlled clinical trials involving 2987 patients.

HYPERTENSION: The most frequent clinical adverse experiences in controlled trials were: headache (5.2%), dizziness (4.3%), and fatigue (3%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in controlled clinical trials were: diarrhea (1.4%), nausea (1.4%), rash (1.4%), cough (1.3%), orthostatic effects (1.2%), and asthenia (1.1%).

HEART FAILURE: The most frequent clinical adverse experiences in both controlled and uncontrolled trials were: dizziness (7.9%), hypotension (6.7%), orthostatic effects (2.2%), syncope (2.2%), cough (2.2%), chest pain (2.1%), and diarrhea (2.1%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in both controlled and uncontrolled clinical trials were: fatigue (1.8%), headache (1.8%), abdominal pain (1.6%), asthenia (1.6%), orthostatic hypotension (1.6%), vertigo (1.6%), angina pectoris (1.5%), nausea (1.3%), vomiting (1.3%), bronchitis (1.3%), dyspnea (1.3%), urinary tract infection (1.3%), rash (1.3%), and myocardial infarction (1.2%).

Other serious clinical adverse experiences occurring since the drug was marketed or adverse experiences occurring in 0.5% to 1% of patients with hypertension or heart failure in clinical trials in order of decreasing severity within each category:

Cardiovascular: Cardiac arrest; myocardial infarction or cerebrovascular accident, possibly secondary to excessive hypotension in high-risk patients (see WARNINGS, *Hypotension*); cardiac arrest; pulmonary embolism and infarction; rhythm disturbances; atrial fibrillation, palpitation.

Digestive: Ileus, pancreatitis, hepatitis or cholestatic jaundice, melena, anorexia, dyspepsia, constipation, glossitis.

Nervous/Psychiatric: Depression, confusion, ataxia, somnolence, insomnia, nervousness, paresthesia.

Urogenital: Renal failure, oliguria, renal dysfunction (see PRECAUTIONS and DOSAGE AND ADMINISTRATION).

Respiratory: Bronchospasm, rhinorrhea, asthma, upper respiratory infection.

Skin: Herpes zoster, pruritus, alopecia, flushing, photosensitivity.

Other: Vasculitis, muscle cramps, hyperhidrosis, impotence, blurred vision, taste alteration, tinnitus.

A symptom complex has been reported which may include fever, myalgia, and arthralgia; an elevated erythrocyte sedimentation rate may be present. Rash or other dermatologic manifestations may occur. These symptoms have disappeared after discontinuation of therapy.

Angioedema: Angioedema has been reported in patients receiving VASOTEC (0.2%). Angioedema associated with laryngeal edema may be fatal. If angioedema of the face, extremities, lips, tongue, glottis, and/or larynx occurs, treatment with VASOTEC should be discontinued and appropriate therapy instituted immediately. (See WARNINGS.)

Hypotension: In the hypertensive patients, hypotension occurred in 0.9% and syncope occurred in 0.5% of patients following the initial dose or during extended therapy. Hypotension or syncope was a cause for discontinuation of therapy in 0.1% of hypertensive patients. In heart failure patients, hypotension occurred in 6.7% and syncope occurred in 2.2% of patients. Hypotension or syncope was a cause for discontinuation of therapy in 1.9% of patients with heart failure. (See WARNINGS.)

Clinical Laboratory Test Findings:

Serum Electrolytes: Hyperkalemia (see PRECAUTIONS), hyponatremia.

Creatinine, Blood Urea Nitrogen: In controlled clinical trials, minor increases in blood urea nitrogen and serum creatinine, reversible upon discontinuation of therapy, were observed in about 0.2% of patients with essential hypertension treated with VASOTEC alone. Increases are more likely to occur in patients receiving concomitant diuretics or in patients with renal artery stenosis. (See PRECAUTIONS.) In patients with heart failure who were also receiving diuretics with or without digitalis, increases in blood urea nitrogen or serum creatinine, usually reversible upon discontinuation of VASOTEC and/or other concomitant diuretic therapy, were observed in about 11% of patients. Increases in blood urea nitrogen or creatinine were a cause for discontinuation in 1.2% of patients.

Hemoglobin and Hematocrit: Small decreases in hemoglobin and hematocrit (mean decreases of approximately 0.3 g % and 1.0 vol %, respectively) occur frequently in either hypertension or heart failure patients treated with VASOTEC but are rarely of clinical importance unless another cause of anemia coexists. In clinical trials, less than 0.1% of patients discontinued therapy due to anemia.

Other (Causal Relationship Unknown): In marketing experience, rare cases of neutropenia, thrombocytopenia, and bone marrow depression have been reported.

Liver Function Tests: Elevations of liver enzymes and/or serum bilirubin have occurred.

Dosage and Administration: *Hypertension:* In patients who are currently being treated with a diuretic, symptomatic hypotension occasionally may occur following the initial dose of VASOTEC. The diuretic should, if possible, be discontinued for two to three days before beginning therapy with VASOTEC to reduce the likelihood of hypotension. (See WARNINGS.) If the patient's blood pressure is not controlled with VASOTEC alone, diuretic therapy may be resumed.

If the diuretic cannot be discontinued, an initial dose of 2.5 mg should be used under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, *Drug Interactions*.)

The recommended initial dose in patients not on diuretics is 5 mg once a day. Dosage should be adjusted according to blood pressure response. The usual dosage range is 10 to 40 mg per day administered in a single dose or in two divided doses. In some patients treated once daily, the antihypertensive effect may diminish toward the end of the dosing interval. In such patients, an increase in dosage or twice-daily administration should be considered. If blood pressure is not controlled with VASOTEC alone, a diuretic may be added.

Concomitant administration of VASOTEC with potassium supplements, potassium salt substitutes, or potassium-sparing diuretics may lead to increases of serum potassium (see PRECAUTIONS).

Dosage Adjustment in Hypertensive Patients with Renal Impairment: The usual dose of enalapril is recommended for patients with a creatinine clearance >30 mL/min (serum creatinine of up to approximately 3 mg/dL). For patients with creatinine clearance ≤30 mL/min (serum creatinine ≥3 mg/dL), the first dose is 2.5 mg once daily. The dosage may be titrated upward until blood pressure is controlled or to a maximum of 40 mg daily.

Heart Failure: VASOTEC is indicated as adjunctive therapy with diuretics and digitalis. The recommended starting dose is 2.5 mg once or twice daily. After the initial dose of VASOTEC, the patient should be observed under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, *Drug Interactions*.) If possible, the dose of the diuretic should be reduced, which may diminish the likelihood of hypotension. The appearance of hypotension after the initial dose of VASOTEC does not preclude subsequent careful dose titration with the drug, following effective management of the hypotension. The usual therapeutic dosing range for the treatment of heart failure is 5 to 20 mg daily given in two divided doses. The maximum daily dose is 40 mg. Once-daily dosing has been effective in a controlled study, but nearly all patients in this study were given 40 mg, the maximum recommended daily dose, and there has been much more experience with twice-daily dosing. In addition, in a placebo-controlled study which demonstrated reduced mortality in patients with severe heart failure (NYHA Class IV), patients were treated with 2.5 to 40 mg per day of VASOTEC, almost always administered in two divided doses. (See CLINICAL PHARMACOLOGY, *Pharmacodynamics and Clinical Effects*.) Dosage may be adjusted depending upon clinical or hemodynamic response. (See WARNINGS.)

Dosage Adjustment in Heart Failure Patients with Renal Impairment or Hyponatremia: In heart failure patients with hyponatremia (serum sodium <130 mEq/L) or with serum creatinine >1.6 mg/dL, therapy should be initiated at 2.5 mg daily under close medical supervision. (See DOSAGE AND ADMINISTRATION, *Heart Failure*, WARNINGS, and PRECAUTIONS, *Drug Interactions*.) The dose may be increased to 2.5 mg b.i.d., then 5 mg b.i.d. and higher as needed, usually at intervals of four days or more, if at the time of dosage adjustment there is not excessive hypotension or significant deterioration of renal function. The maximum daily dose is 40 mg.

For more detailed information, consult your MSD Representative or see Prescribing Information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, PA 19486. J6VS18R2(817)

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CONTRAINDICATIONS: Hypersensitivity to acetaminophen or hydrocodone.
WARNINGS:
Allergic-Type Reactions: VICODIN/VICODIN ES Tablets contain sodium metabisulfite, a sulfite that may cause allergic-type reactions including anaphylactic symptoms and life-threatening or less severe asthmatic episodes in certain susceptible people.
Respiratory Depression: At high doses or in sensitive patients, hydrocodone may produce dose-related respiratory depression.
Head Injury and Increased Intracranial Pressure: The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a preexisting increase in intracranial pressure. Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries.
Acute Abdominal Conditions: The administration of narcotics may obscure the diagnosis or clinical course of patients with acute abdominal conditions.
PRECAUTIONS:
Special Risk Patients: VICODIN/VICODIN ES Tablets should be used with caution in elderly or debilitated patients and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture.
Cough Reflex: Hydrocodone suppresses the cough reflex; as with all narcotics, caution should be exercised when VICODIN/VICODIN ES Tablets are used postoperatively and in patients with pulmonary disease.
Drug Interactions: Patients receiving other narcotic analgesics, antipsychotics, antianxiety agents, or other CNS depressants (including alcohol) concomitantly with VICODIN/VICODIN ES Tablets may exhibit an additive CNS depression. The use of MAO inhibitors or tricyclic antidepressants with hydrocodone preparations may increase the effect of either the antidepressant or hydrocodone. The concurrent use of anticholinergics with hydrocodone may produce paralytic ileus.
Usage in Pregnancy:
Teratogenic Effects: Pregnancy Category C. Hydrocodone has been shown to be teratogenic in hamsters when given in doses 700 times the human dose. There are no adequate and well-controlled studies in pregnant women. VICODIN/ VICODIN ES Tablets should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.
Nonteratogenic effects: Babies born to mothers who have been taking opioids regularly prior to delivery will be physically dependent. The withdrawal signs include irritability and excessive crying, tremors, hyperactive reflexes, increased respiratory rate, increased stools, sneezing, yawning, vomiting, and fever.
Labor and Delivery: Administration of VICODIN/VICODIN ES Tablets to the mother shortly before delivery may result in some degree of respiratory depression in the newborn, especially if higher doses are used.
Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk and because of the potential for serious adverse reactions in nursing infants from VICODIN/VICODIN ES Tablets, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.
Pediatric Use: Safety and effectiveness in children have not been established.
ADVERSE REACTIONS:
The most frequently observed adverse reactions include light-headedness, dizziness, sedation, nausea and vomiting. These effects seem to be more prominent in ambulatory than in nonambulatory patients and some of these adverse reactions may be alleviated if the patient lies down. Other adverse reactions include:
Central Nervous System: Drowsiness, mental clouding, lethargy, impairment of mental and physical performance, anxiety, fear, dysphoria, psychic dependence and mood changes.
Gastrointestinal System: The antiemetic phenothiazines are useful in suppressing the nausea and vomiting which may occur (see above); however, some phenothiazine derivatives seem to be antianalgesic and to increase the amount of narcotic required to produce pain relief, while other phenothiazines reduce the amount of narcotic required to produce a given level of analgesia. Prolonged administration of VICODIN/VICODIN ES Tablets may produce constipation.
Genitourinary System: Ureteral spasm, spasm of vesical sphincters and urinary retention have been reported.
Respiratory Depression: Hydrocodone bitartrate may produce dose-related respiratory depression by acting directly on the brain stem respiratory center. Hydrocodone also affects the center that controls respiratory rhythm, and may produce irregular and periodic breathing. If significant respiratory depression occurs, it may be antagonized by the use of naloxone hydrochloride. Apply other supportive measures when indicated.
DRUG ABUSE AND DEPENDENCE:
VICODIN/VICODIN ES Tablets are subject to the Federal Controlled Substance Act (Schedule III). Psychic dependence, physical dependence, and tolerance may develop upon repeated administration of narcotics; therefore, VICODIN/ VICODIN ES Tablets should be prescribed and administered with caution.
OVERDOSAGE:
Acetaminophen Signs and Symptoms: In acute acetaminophen overdosage, dose-dependent, potentially fatal hepatic necrosis is the most serious adverse effect. Renal tubular necrosis, hypoglycemic coma, and thrombocytopenia may also occur. Early symptoms following a potentially hepatotoxic overdose may include: nausea, vomiting, diaphoresis and general malaise. Clinical and laboratory evidence of hepatic toxicity may not be apparent until 48 to 72 hours post-ingestion.
Hydrocodone Signs and Symptoms: Serious overdose with hydrocodone is characterized by respiratory depression (a decrease in respiratory rate and/or tidal volume, Cheyne-Stokes respiration, (cyanosis), extreme somnolence progressing to stupor or coma, skeletal muscle flaccidity, cold and clammy skin, and sometimes bradycardia and hypotension. In severe overdosage, apnea, circulatory collapse, cardiac arrest and death may occur.

Revised June 1989
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Nov 8. **Dermatology Update.** 3 Cat I Credits. Contact: Charlotte L. Hoag, Medical Education Coordinator, Office of Medical Education, The Mary Imogene Bassett Hospital, One Atwell Rd, Cooperstown, NY 13326-1394. Tel: (607) 547-3926.

FOREST HILLS

Nov 5. **Child Abuse and the Abuser—A Double Concern.** 3½ Cat I Credits. Academy of Medicine of Queens County. Contact: Daniel Chansky, MD, CME Committee, Academy of Medicine of Queens County, 112-25 Queens Blvd, Forest Hills, NY 11375. Tel: (718) 268-7300.

MANHATTAN

Nov 3. **Sports Medicine: Protecting the Young Athlete.** 6 Cat I Credits. *Nov 4–5.* **Teaching Humanistic Medicine: An Exploration of Goals, Techniques and Experiences.** 15 Cat I Credits. *Nov 9–11.* **Substance Abuse: The Office Management of the Chemically Dependent Patient.** 15 ½ Cat I Credits. *Nov 18–19.* **Review of GU Pathology—Workshop for Urologists.** 16 Cat I Credits. *Nov 30–Dec 1.* **Cooperative Care: The Ten-Year Success Story of an Innovative Hospital-Patient Care Delivery System.** 11 Cat I Credits. NYU Medical Center. Contact: NYU Medical Center, Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

Nov 4–5. **27th Annual Bernard H. Eliasberg Symposium. Management of the High Risk Cardiac Patient: Transition into the Next Decade.** 8 Cat I Credits. Mt Sinai Medical Center. Contact: Ms Joan D. Esbri, Dept of Anesthesiology, Box 1010, Mt Sinai Medical Center, 1 Gustave L. Levy Pl, New York, NY 10029. Tel: (212) 241-6426.

Nov 17–19. **16th Annual Current Critical Problems in Vascular Surgery.** 22 Cat I Credits. Waldorf-Astoria Hotel. Contact: Office of Continuing Medical Education, Albert Einstein College of Medicine/Montefiore Medical Center, 3301 Bainbridge Ave, Bronx, NY 10467. Tel: (212) 920-6674.

Nov 18. **New York Society for Clinical Ophthalmology Fall Meeting.** *Nov 30–Dec 2.* **Aesthetic Rhinoplasty '89—State of the Art.** Waldorf Astoria Hotel. Contact: Francine Leinhardt, Course Coordinator, Manhattan Eye, Ear & Throat Hospital, 210 E 64th St, New York, NY 10021. Tel: (212) 838-9200, ext 2776.

NEW HYDE PARK

Nov 18. **Third Annual Conference on Common Orthopaedic Problems in Children.** 8 Cat I Credits. Long Island Jewish Medical Center. Contact: Ann J. Boehme, CMP, Associate Director for Continuing Education, Schneider Children's Hospital of Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

AROUND THE NATION

CALIFORNIA

Nov 2–4. **The San Francisco Otology Update: 1989.** 22 Cat I Credits. Mark Hopkins Hotel, San Francisco. *Nov 10–12.* **35th Annual Group Therapy Symposium: Focus on Leadership.** Sir Francis Drake Hotel, San Francisco. Contact: Extended Programs in Medical Education, University of California, Room U-569, San Francisco, CA 94143. Tel: (415) 476-4151.

Nov 3–5. **Ninth Annual Interventional and Imaging Radiology Course.** 18 Cat I Credits. Hotel del Coronado, San Diego. *Nov 10–12.* **Advanced Seminars in Diagnostic Imaging.** 18 Cat I Credits. Ritz Carlton Resort Hotel, Laguna Niguel. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077-1925. Tel: (404) 641-9773.

Nov 9–11. **AIDS and Other Sexually Transmitted Diseases.** 16 Cat I Credits. Universal City. Contact: The American

(*continued on p 11A*)

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COMMENTARIES

Current challenges to the practice of medicine

In January of 1988, the Council of the Medical Society of the State of New York (MSSNY) adopted a strategic plan for the society. One of the key objectives of the plan is to have the society become a more aggressive advocate for physicians' interests which we have been attempting to pursue.

The following are some of the issues that have received a great deal of attention from the society in 1989:

- Mandatory Medicare assignment
- The state's health care crisis resulting from under-reimbursement and over-regulation
- Medical liability
- The section 405 Code regulations and their effect on patient care and graduate medical education
- Relations with specialty societies
- Infectious waste
- Quality of care
- Physician recredentialing
- Physician reimbursement
- Universal health insurance
- Long term care

The Physician Payment Review Commission (PPRC) recently reported to Congress and is recommending a national fee schedule for Medicare based on a resource based relative value scale (RBRVS) with regional differences based on overhead and medical liability premiums. This could mean increases in fees for most primary care physicians and reductions for surgeons. Reductions would average about 20% overall and would be phased in over a two-year period.

Although the commission has rejected mandatory assignment as a general rule, it does support it for the 1.5 million elderly (3–5%) living below the federal poverty line (roughly $6,000) (cash income only). Thus, there will be limits on balance billing.

The Physician Payment Review Commission is also recommending expenditure caps for part B Medicare spending somewhat similar to a formula used by the Canadian provinces. This is being opposed by the American Medical Association (AMA) as outright rationing of care for which the American people will not stand. Again, it is simply the inevitable result of policies of over-regulation and under-reimbursement.

The first signs of a hospital crisis in our state emerged in the spring of 1987 when length of stay and occupancy levels sharply rose in New York City hospitals. The current catastrophic situation results, in our judgment, from short sighted health planning policies. Locally based health planning does not exist in New York State, but rather the current health planning process is centered in, and has become dominated by, the regulatory process of the New York State Department of Health (DOH). Objectivity and independence have gone by the wayside because funding is almost exclusively provided by the DOH and emphasis has been placed on the elimination of thousands of so-called excess beds.

Unfortunately, the Health Systems Agencies (HSAs) and the health planning process itself have merely become extensions of the DOH. What has become even more offensive to physicians is bureaucratic intrusions into clinical decision making under the guise of quality control. But, we should not be too surprised. As someone once said, "Only in politics are facts negotiable."

A few statistics illustrate the severity of the crisis. It is estimated that on any given day, there are more than 4,000 elderly patients waiting for nursing home beds in the state. New York State has 43 nursing home beds per 1,000 population over the age of 65. The US average is 59 beds per 1,000 population and New York ranks 43rd out of 50 states in this regard.

Today, the average length of stay in New York City hospitals is 9.8 days, compared with a nationwide average of six days. While the average occupancy rate has dropped to 60% nationally, the state DOH data show that the average occupancy rate at New York City hospitals has climbed 10% in the past year to 95.4%. The same pattern prevails throughout the state.

There are disturbing signs all around us that the health care system in New York State is beginning to crumble. This is why the society recently wrote to Governor Cuomo recommending the creation of a blue ribbon commission comprising experts from outside New York State to look at our system. We have also asked for the dismissal of the commissioner of health.

Hospital emergency rooms are backed up with patients who are suffering the effects of substance abuse, mental illness, malnutrition, homelessness, and acquired immunodeficiency syndrome (AIDS). Meanwhile, hospital beds are filled with frail elderly patients who cannot be discharged because of insufficient nursing home beds. Faced with these demands, hospitals at the same time are encountering serious shortages of health care professionals and a lack of adequate funding to even pay for them. And the new section 405 regulations which became effective July 1, 1989, will cost hospitals an additional $300 million to implement.

Is it any wonder, then, that New York no longer seems to be the center of medical excellence, teaching, and technologic innovation? Pioneering research and new procedures have become associated with places like Salt Lake City, Utah; Louisville, Kentucky; Loma Linda, California; etc.

Moreover, the climate for the practice of medicine in New York State is becoming less attractive, especially to young physicians. Excessive regulations, coupled with a department of health that displays a continuing disdain for the profession, the AIDS crisis, and unrealistic reimbursement policies have all contributed to a deteriorating practice environment. The quantity and quality of medical school applicants have dropped significantly to the extent that it is probably the easiest time in many, many years to gain admission to most medical schools in New York. I am also told that although our residencies are filled, the quality of those entering residencies in New York is certainly not what it used to be since many of our top students are going out of state.

The news from the national level is not any better either. Since 1984, physicians' charges have been subjected to both fee freezes and limitations on the amount Medicare patients can be billed, known as the maximum allowable charge (MAAC). Therefore, physicians are prohibited by law from billing Medicare patients above the physician's individual MAAC which is generally 15–20% below what non-Medicare patients are charged.

In 1986, spending on entitlement programs, such as Medicare and Medicaid, was $466 billion—47.1% of the entire federal budget, compared with 27.6% for defense and 16.3% for non-defense discretionary spending, and only 15.1% of the $466 billion in entitlement spending is means-tested.

Entitlement programs hold the key to the federal budget deficit and the future viability of the nation's economy. We need the courage to forge a new social welfare system that transcends the special interests of the entitlements lobby.

Unless the nation addresses the restructuring of our entitlement programs, Medicare faces heavy long term financing problems which threaten to produce either untenable payroll tax burdens for workers or deep benefit reductions for retirees. Perhaps Congress, by requiring the elderly to pay for their new catastrophic benefit, is recognizing the need to reform our entitlement programs. If this nation does not move from age-based programs to need-based (perhaps merging Medicare and Medicaid into a single program) programs in the near future, we are likely to have intergenerational conflict.

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The medical malpractice mediation panel

In the early 1970s, the New York State Supreme Court in New York County organized an informal medical malpractice panel with the hope that this would help to dispose of cases before they actually reached trial. The general format consisted of an informal presentation by all sides before a panel consisting of a judge, a physician, and an attorney. After the presentation, the panel would have a private discussion and report a nonbinding recommendation to the attorneys. Some cases were ultimately settled under this format.

With the recognition of the "malpractice crisis," legislation in 1975, Judicial Law § 148-a, formalized the concept of a malpractice mediation panel to operate statewide. In 1985, Judicial Law § 148-a(1) was amended to exclude Suffolk County and the Fifth Judicial District, consisting of the counties of Herkimer, Jefferson, Lewis, Oneida, Onondaga, and Oswego.

Presumably, it was the purpose of the legislation that created the formal panel process to develop an atmosphere that would help to dispose of malpractice cases prior to trial. The leverage within the legislation to promote dispositions is the power of a panel to make a unanimous finding which is admissible as evidence during a trial. A panel can find in one of three ways: there can be a unanimous finding in favor of the physician; there can be a mixed or no finding; or there can be a unanimous finding of liability against the physician. The mixed or no finding becomes meaningless. A unanimous finding for or against the defendant becomes admissible as evidence during the trial. It is similar in effect to an expert opinion.[1] The panel's finding is not binding on the jury, but it is a piece of evidence for its consideration. Judicial Law § 148-a(8) provides that in the event that a unanimous panel finding is submitted to the jury, the attorney and/or physician member of the panel may be called as a witness at the trial by any party to testify as to the basis of the panel's decision. It is the obvious intent of the legislation to pressure the losing party at a panel into negotiating a disposition. Negotiations are part of the entire judicial system; consequently, the aim of the panel is quite consistent with the aim of the judicial process, which is to resolve disputes.

The creation of a formal mediation panel was accomplished with the support and cooperation of the Medical Society of the State of New York. This cooperation has continued, and the most critical contribution by county medical societies is the selection of physicians to serve on the panels.

In order to provide a fair panel hearing, the court notifies the medical society in the county of the need for a physician in a particular specialty. It is the responsibility of the county medical society to select and provide a physician not only with the appropriate medical credentials, but also one who will render an objective opinion. Physicians who volunteer for this very important role must appreciate that this unique position is quasi-judicial. Consequently, a physician should not volunteer for or accept the position of a panel member unless he is confident that he can render an impartial analysis of the evidence.

The material to be submitted to a panel for consideration has been standardized. The Bill of Particulars, appropriate medical and hospital records, x-rays, and written contentions of the various parties are all submitted in advance for review by the members of the panel. The contention papers are not exchanged among the parties. The procedure does not provide for a transcript because the hearings are informally conducted. What has never been clarified, however, is what rules apply in order for panel members to determine liability.

Although we know that a panel hearing is not the same as a trial, it is not clear by what standard the panel is to measure the information or evidence it receives. How is a physician panelist to make a judgment? At the end of a trial, the judge gives a "charge" to the jury which carefully describes the law that covers a malpractice case. However, it is not clear that the panelist physician is receiving the same sort of instruction.

The most rudimentary jury charge in any negligence case, not limited to medical malpractice, is that the plaintiff has the burden of proof. The quality of the plaintiff's proof must be superior to that of the defendant. In the event that the plaintiff's proof is not superior to or is equal to that of the defendant, then the defendant wins. A tie goes to the defendant. That is the law. There is no reason why the same rule should not apply at a panel. Consequently, when weighing the evidence before it, a panel member should consider whether the plaintiff has prevailed on the burden of proof.

In addition, there cannot be malpractice, even with a departure from the standard of practice, unless there is "proximate cause." That is, the departure must be a substantial contributing factor to some injury claimed by the plaintiff. I have been stunned to hear from a panel physician after a panel hearing that it was his presumption that all that was required for a finding of malpractice was that there be a finding of some error on the part of the physician. The law requires that any error, before it is actionable, must produce an injury. Consequently, if a physician orders an inappropriate medication, but the order produces no injury, then there has been no malpractice. The plaintiff has the burden of proof on both the issue of a departure from the standard of care and the issue of proximate cause. A 1978 decision held that a finding of liability by a panel must include both a departure from practice and proximate cause.[2]

A charge at the end of a trial will also instruct a jury that malpractice consists of a physician's departing from some standard of practice that was the standard at the time the treatment was rendered. Obviously, if the treatment under review was rendered in 1980, a 1987 standard of practice is not applied.

On one occasion, defending a surgeon before a panel, I was very disappointed with a unanimous finding of liability. It was our view that although something untoward had occurred, probably as the result of anesthesia, we did not believe that the surgeon was responsible. Subsequently, I asked the physician panelist for the basis of his finding. The response was, "Well, the surgeon was the captain of the ship." I then explained, too late, that there is no legal concept in New York of liability for the "captain of the ship," and that there should not have been a finding on that basis.

After another disappointing panel result, the panelist explained that the adverse finding was the result of his assumption that the poor result sustained by the patient must have been the result of malpractice. That is, the panelist started with the injury and worked backward. The law, however, requires prospective analysis starting with the information available to the defendant at the time of the treatment. The law specifically provides that the injury is not evidence of malpractice. Retrospective reasoning is prohibited at the trial; it should not be permitted at a panel.

It is not uncommon to hear a panelist casually mention after a hearing that it was suggested that he should not insist on a unanimous finding, because the plaintiff "should have his day in court." Every panelist should understand that a finding does not eliminate anyone's right to a full trial. Everyone gets his day in court no matter what is done by a mediation panel.

Normally, a copy of the Bill of Particulars is submitted to the panel. That is a document which the plaintiff is compelled by law to serve on the defendant early in the development of the litigation. Its purpose, among other things, is to give notice to the defendant of specific allegations of negligence.

Although the legal issues at a panel would normally be in the province of the judge or the attorney, the physician should review the allegations in the Bill of Particulars. One must be alert to whether the plaintiff's claims in the contention papers are consistent with the claims in the Bill of Particulars. If some new theory or claim has been raised in the contention papers, the defendant will be at a severe disadvantage. The defendant never sees the plaintiff's contention papers, therefore his contention papers must be in response to the Bill of Particulars. Nothing is more frustrating than defending a case at a panel and being confronted with a new theory of the case.

As a panelist, one must be prepared to ask why the panel would consider anything except what is claimed in the Bill of Particulars. That document is intended, by law, to delineate the issues a physician must defend at trial. Why should the physician have a greater burden at the panel?

The malpractice mediation panel is now part of the judicial process. Physicians must make a good faith effort to participate in the process. There will be occasions when the care and treatment physicians will review will be a disappointment, and one has an obligation to say that. One also has a right to know the rules before one casts any judgment. Panelists should insist on that right and insist that any discussion is in accord with the rules.

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1. *Lipsius v White*, 91 App Div 2d 271 (2nd Dept 1983).
2. *Kletnieks v Brookhaven Mem Assoc, Inc*, 68 App Div 2d 994, 406 NYS 2d 114 (2nd Dept 1978).

Mortality patterns among a Native American population in New York State

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ABSTRACT. This study investigated patterns of mortality among a Native American tribe, the Seneca Nation of Indians (SNI). The names of 962 tribal members reported to have died in New York State between 1955 and the end of 1984 were identified through a review of tribal roll books maintained by the Seneca Nation. Positive matches were obtained for 796 (83%) of these individuals using New York State mortality files for the period under investigation. Standardized Proportionate Mortality Ratios (PMR) were computed for major causes of death based on cause-specific mortality patterns in the New York State population for each sex during the same time period. Significantly elevated risks of mortality were observed for all infectious diseases, tuberculosis, diabetes mellitus, cirrhosis, and accidents. Depressed mortality ratios were noted for deaths due to all cancers combined, and for cancers of the lung, pancreas, breast, and lymphatic/hematopoietic cancers. Changes in mortality risks over time were also observed.

(NY State J Med 1989; 89:557-561)

There have been few studies concerning mortality patterns from chronic diseases among Native populations. Studies to date have focused on populations residing in the southwestern region of the United States, Alaska, and Canada.[1-4] The majority of these studies reflect changes in mortality patterns over time, with deaths due to chronic diseases outpacing deaths due to infectious diseases in recent times. The current study focuses on mortality patterns among a northeastern Native American tribe, the Seneca Nation of Indians (SNI), from 1955 through 1984.

The present study not only reports on SNI mortality patterns for an entire 30-year period, but also highlights changes in mortality over this period. Proportionate mortality studies are useful in the generation of new hypotheses and in the conduct of preliminary tests of etiology. They also are a quick and cost-effective means not only of assessing overall mortality patterns in a special population, but also in the identification of newly emerging causes of mortality with which public health professionals must be concerned.

METHODS

The SNI is one of approximately six Iroquois tribes that occupy lands in New York State. The SNI is a matrilineal society whose members primarily reside on two reservations in western New York State. The study group consisted of all members of the Seneca Nation of Indians reported to have died in New York State during the period 1955 through 1984. Names of deceased tribal members were identified through a review of annual necrology listings and annuity rolls maintained by the SNI. Through these means, names and other identifying information were obtained for 962 members reported as having died in New York State during the period under investigation. A computerized tape containing identifying information on this group was submitted to the New York State Department of Health for matching against state mortality files for the period under investigation. Positive matches were obtained for 796 (82.7%) of these individuals. Death certificate information was abstracted to obtain data on name, sex, age at death, and underlying cause of death. Cause of death was encoded according to the International Classification of Diseases.[5]

Standardized Proportionate Mortality Ratios (PMR) were calculated for the entire period under investigation (1955–1984), as well as for the three ten-year periods comprising the overall period of study (1955–1964, 1965–1974, 1975–1984). Observed number of deaths for a particular cause were divided by sex-, age-, and calendar-year-specific expected numbers of deaths.[6] Expected numbers of deaths are based on the ratio of each cause to all causes in New York State general population at the midpoint of each ten-year period. The PMR is an indirect method of age adjustment; in this study age was stratified into 20 five-year age-intervals (eg, 0–4 yr, 5–9 yr, etc) for males and females separately. In effect, this adjusts for any differences in the age and sex composition between the study group and the comparison population. While PMRs do not provide information on the total force of mortality, they are thought to provide as good approximations of cause-specific risks as those obtained from the more sensitive standardized mortality ratios.[6]

In addition to presenting cause-specific standardized PMRs among the SNI between 1955 and 1984, comparisons are made in this paper of the first and third periods to highlight major shifts in mortality over time. Ratios for the second period were mostly intermediate to observations in each of the two other periods, and by themselves do not provide significant contributions to this presentation. The statistical significance of the difference between observed and expected numbers of cases was determined with reference to a Poisson distribution. Confidence intervals

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From the Departments of Education (Dr Michalek) and Cancer Control and Epidemiology (Dr Cummings), Roswell Park Memorial Institute, Buffalo, NY, the Bureau of Cancer Epidemiology, New York State Department of Health (Dr Mahoney), Albany, NY, and the Seneca Nation Health Department (Mr Hanley and Ms Snyder), Salamanca, NY.

Address correspondence to Dr Michalek, Assistant Director of Education, Roswell Park Memorial Institute, 666 Elm St, Buffalo, NY 14263.

(95%) were constructed on the basis of the tables constructed by Bailar and Ederer[7] and Mantel.[8]

RESULTS

A total of 962 members of the SNI were reported to have died in New York State between 1955 and 1984. The cause of death was ascertained for 796 (82.7%) of these individuals. Repeated submissions to Vital Statistics utilizing relaxed matching criteria (ie, decade of death rather than year of death) failed to increase appreciably death ascertainment. Individuals for whom cause of death could not be secured may have died out of New York State. Age at death by sex was equivalent with the exception of the 20–29 year age group in which males (10.1%) exhibited a higher proportion of deaths than females (3.9%). The median age at death among SNI members during 1955–1964 was 55.9 years (mean, 51.9 yr); while for 1975–1984, the median age at death was 64.6 years (mean, 60.9 yr). Median age at death is presented since it is a better indicator of central tendency than an arithmetic mean, which is affected by extremes in the tails of the distribution. The distribution of deaths among the study population is shown in Figure 1.

Table I presents data on cause-specific PMRs by sex for the entire study period (1955–1984). Cause-specific PMRs for sexes combined reveal statistically significant elevated risks of mortality from infectious diseases, tuberculosis, diabetes mellitus, cirrhosis, and accidents. Fewer than expected deaths were observed for all cancers combined and, specifically, for cancers of the lung, pancreas, and breast, and lymphatic/hematopoietic cancers.

Male/Female Differences. Males exhibited significantly elevated risks of mortality from infectious diseases, tuberculosis, diabetes mellitus, cirrhosis, and accidents. Significantly depressed levels of mortality were observed for all neoplasms combined, lymphatic/hematopoietic cancers, and pneumonia.

Females exhibited significantly elevated risks of mortality from diabetes mellitus and accidents. Among females, deficits of mortality were observed for cancers of the breast, lymphatic/hematopoietic system, pancreas, and for all neoplasms combined.

Trends Over Time. Table II presents cause-specific PMRs by sex for the first (1955–1964) and third (1975–1984) ten-year periods of the investigation. Standardized PMRs for infectious diseases increased from 2.48 to 3.48. Most of this increase is attributable to the increase in the PMR for male deaths, which rose from 2.06 to 4.98. The risk of death due to tuberculosis increased sharply from the first to third period for both sexes; the overall PMR increased from 1.86 in the first period to 10.34 in the last period.

Trends in Cancer Mortality. PMRs for all neoplasms combined rose slightly from 0.42 to 0.60, for the first and third ten-year periods, respectively, but remained significantly below expected levels. While the female ratio remained relatively stable (0.42 and 0.47), the male ratio increased from 0.42 to 0.70. PMRs for colorectal cancer increased from 0.40 in the first period to 0.95 in the third period. This increase is largely due to the increase in the male ratio, which increased from 0.27 to 1.45. While no lung cancer deaths were observed in the first period, 17 were observed during the third period, yielding a ratio of 0.97 for males and 0.54 for females; 0.85 overall.

PMRs for cervical cancer were found to be elevated for both time periods—1.55 in the first period and 1.91 in the third. PMRs for female breast cancer remained significantly depressed for both periods, although to a lesser extent for the last period. Ratios of 0.15 for the first period and 0.37 for the third period were observed. Remaining cancer sites exhibited some shifting over time, but the numbers remain too small to draw any meaningful conclusions.

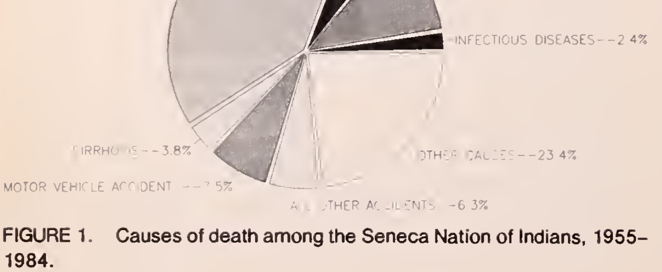

FIGURE 1. Causes of death among the Seneca Nation of Indians, 1955–1984.

Other Trends. PMRs for diabetes mellitus remained significantly elevated over time—3.19 in the first period and 3.02 in the last. However, female ratios declined from 3.46 to 2.04, while male ratios increased from 2.84 to 4.09 over time. The overall tuberculosis ratio increased from 0.68 to 1.96; females exhibited an increased ratio from 0.47 to 1.20, while the male ratio dramatically increased from 0.88 to 2.38.

The risk of death from all accidents combined remained significantly elevated for both periods, with females exhibiting a greater risk than males in the last period (1.73 vs 1.30). When the accident category was further subdivided, it was interesting to note that while the risk of death due to motor vehicle accidents decreased for males over time (1.70 vs 0.96), female risks rose from 1.20 to 1.81. Regarding accidental causes of death other than those related to motor vehicles, female ratios decreased slightly from 1.71 to 1.58, while male ratios increased from 1.56 to 1.93 between the first and last period.

DISCUSSION

In this study, several significant changes in mortality patterns over time were observed among the SNI. A significant change was observed in age at death between 1955 and 1984 (t-test; $p < 0.05$). In the first period under observation, the median age at death was found to be 59.9 years, while for the final period it increased to 64.6 years. By comparison, the median age at death for the United States population in 1959–1961 was 74.3 years and 77.6 years for 1979–1981.[9] While median age at death in this study population increased over time, it still remained substantially lower than that of the general population.

Standardized Mortality Ratios (SMR) from the New Mexico Vital Statistics Report[2] for the period 1979–1982 demonstrated that Native Americans experienced excess mortality from all causes of death combined (SMR=1.27), all accidents (SMR=3.9), diabetes mellitus (SMR=2.27), influenza and pneumonia (SMR=2.0), and cirrhosis of the liver (SMR=2.5). The Standardized Mortality Ratio represents the ratio of the number of deaths observed among a particular group followed over time to the number of deaths that would have been expected if the group had the same rate of death as the comparison population experienced over that period of time. A deficit of deaths due to malignant neoplasms (SMR=0.58) was also observed. These findings are consistent with those observed in this study.

Cregan and Fraumeni[10] also reported lower overall cancer mortality rates among Native Americans relative to rates in both black and white populations. Increased mortality was observed for cancer of the gallbladder (SMR=2.96). Similar results were reported by Gallagher and Elwood[11] in a study of cancer mortality among Indians in the Canadian province of British Columbia. This population exhibited lower mortality for all cancers combined, and for cancers of the stomach, colon, lung, and prostate. Excess mortality was observed among females for gallbladder and cervical malignancies. Young and Frank[12] reported depressed cancer mortality among the Cree and Ojibwa for males but not for females. Both sexes exhibited increased mortality due to kidney cancer, and a deficit of deaths due to lung cancer. Females alone exhib-

TABLE I. Cause-Specific Proportionate Mortality Ratios* by Sex, Seneca Nation of Indians, 1955–1984

Cause/Sex (ICD-9)		Observed No. of Deaths	Proportionate Mortality Ratio	(95%) Confidence Interval
Infectious diseases (0010–1399)	Male	12	2.82	1.53–4.91
	Female	7	2.05	0.82–4.23
	Combined	19	2.48	1.49–3.87
Tuberculosis (0100–0189)	Male	5	3.20	1.03–7.48
	Female	2	2.20	0.27–7.90
	Combined	7	2.83	1.14–5.84
All malignant neoplasms (1400–2089)	Male	57	0.64	0.49–0.82
	Female	36	0.44	0.30–0.62
	Combined	93	0.54	0.44–0.67
Buccal (1400–1499)	Male	2	0.76	0.09–2.73
	Female	0	0.00	0.00–2.78
	Combined	2	0.54	0.07–1.94
Stomach (1510–1519)	Male	1	0.23	0.01–1.26
	Female	2	0.69	0.08–2.50
	Combined	3	0.41	0.09–1.20
Colorectal cancer (1530–1549)	Male	13	1.08	0.57–1.85
	Female	5	0.43	0.14–1.00
	Combined	18	0.76	0.44–1.20
Liver (1550–1559)	Male	1	0.46	0.01–2.53
	Female	2	0.97	0.12–3.50
	Combined	3	0.71	0.14–2.06
Gallbladder (1560–1569)	Male	0	0.00	0.00–2.56
	Female	1	0.59	0.11–23.32
	Combined	1	0.35	0.06–13.83
Pancreas (1570–1579)	Male	2	0.45	0.05–1.61
	Female	0	0.00	0.00–0.81
	Combined	2	0.25	0.03–0.88
Lung (1620–1629)	Male	16	0.63	0.36–1.02
	Female	5	0.62	0.20–1.44
	Combined	21	0.63	0.39–0.96
Breast (1740–1749)	Female	4	0.21	0.06–0.53
Cervix (1800–1809)	Female	5	1.33	0.43–3.09
Prostate (1850–1859)	Male	6	1.02	0.37–2.21
Kidney (1890–1899)	Male	1	0.48	0.01–2.66
	Female	1	0.79	0.02–4.40
	Combined	2	0.60	0.07–2.15
Lymphatic/hematopoietic (2000–2089)	Male	4	0.35	0.09–0.89
	Female	4	0.49	0.13–1.25
	Combined	8	0.41	0.18–0.80
Diabetes mellitus (2500–2509)	Male	21	3.37	2.08–5.15
	Female	21	3.00	1.85–4.58
	Combined	42	3.17	2.26–4.70
Cerebrovascular (4300–4389)	Male	26	0.95	0.62–1.39
	Female	25	0.81	0.52–1.19
	Combined	51	0.88	0.65–1.15
Major cardiovascular (3900–4599)	Male	177	0.87	0.75–1.00
	Female	139	0.99	0.83–1.16
	Combined	316	0.92	0.82–1.03
Hypertension with heart disease (4010–4059)	Male	7	1.39	0.56–2.86
	Female	8	1.34	0.58–2.64
	Combined	15	1.36	0.76–2.24
Cirrhosis (5710–5719)	Male	21	2.03	1.25–3.11
	Female	9	1.41	0.65–2.67
	Combined	30	1.80	1.21–2.56
All accidents (8000–9499)	Male	77	1.41	1.11–1.75
	Female	33	1.89	1.29–2.67
	Combined	110	1.52	1.25–1.84
Motor vehicle accidents (8100–8259)	Male	44	1.31	0.95–1.76
	Female	16	1.64	1.00–2.66
	Combined	60	1.39	1.01–1.78
Other accidents (8000–8079, 8260–9499)	Male	33	1.56	1.07–2.18
	Female	17	2.20	1.28–3.52
	Combined	50	1.73	1.28–2.28

* Indirect age-adjusted, five-year age intervals; rates based on New York State residents, excluding New York City, 1955–1984.

ited increased mortality due to gallbladder cancer.

We observed that the SNI experienced similarly lowered mortality due to malignancies. Overall, the proportion of SNI female deaths from cancer was less than one half that expected compared to New York State residents. The proportion of SNI male deaths due to malignant neoplasms was only 64% of expected. The SNI exhibited a deficit of deaths due to cancers of the lung, pancreas, breast, and lymphatic/hematopoietic cancers. While no lung cancers were observed in the first period, 17 lung cancer deaths (PMR=0.85) were observed in the last period. The deficit of lung cancers among Native populations has been attributed to the low prevalence of smoking among these groups.[12,13] The dramatic increase in lung cancer deaths is likely reflective of later adoption of cigarette smoking and possibly a change in mortality from other competing causes of death. While other studies of Native populations[13–15] demonstrated heightened mortality due to cancers of the gallbladder, we failed to replicate these findings. In the entire 30-year period, only one death from gallbladder cancer was observed. This may be due to genetic and/or behavioral differences between the SNI and other Native populations.

SNI females were observed to have a slight excess of deaths due to cancer of the cervix and a deficit of deaths due to breast cancer. Similar excesses in cervical cancer mortality have been reported among other American[10] and Canadian[11] Native populations. It has been postulat-

TABLE II. Proportionate Mortality Ratios for Selected Causes of Death by Sex and Time Period

Cause/Sex (ICD-9)		1955–1964			1975–1984		
		Observed No. of Deaths	Proportionate Mortality Ratio	95% Confidence Interval	Observed No. of Deaths	Proportionate Mortality Ratio	95% Confidence Interval
Infectious diseases (0010–1399)	Male	4	2.06	0.80–7.55	7	4.98	2.40–12.4
	Female	5	2.95	1.15–9.12	2	1.68	0.47–13.9
	Combined	9	2.48	1.30–5.40	9	3.47	1.83–7.6
Tuberculosis (0100–0189)	Male	2	2.15	0.59–17.78	2	10.09	2.79–83.30
	Female	1	1.47	0.26–58.06	1	10.89	1.96–430.38
	Combined	3	1.86	0.63–9.03	3	10.34	3.54–50.17
All malignant neoplasms (1400–2089)	Male	10	0.42	0.22–0.87	31	0.70	0.49–0.95
	Female	11	0.42	0.23–0.83	17	0.47	0.29–0.80
	Combined	21	0.42	0.27–0.87	48	0.60	0.45–0.81
Lung cancer (1620–1629)	Male	0	0.00	0.00–0.58	14	0.97	0.58–1.78
	Female	0	0.00	0.00–3.33	3	0.54	0.18–2.61
	Combined	0	0.00	0.00–0.49	17	0.85	0.53–1.47
Diabetes mellitus (2500–2509)	Male	5	2.84	1.21–8.78	11	1.09	2.28–8.17
	Female	8	3.46	1.76–8.03	6	2.04	0.94–5.57
	Combined	13	3.19	1.87–6.00	17	3.02	1.89–5.19
Major cardiovascular diseases (3900–4599)	Male	62	1.04	0.80–1.36	70	0.80	0.63–1.03
	Female	36	0.95	0.68–1.37	67	1.08	0.85–1.39
	Combined	98	1.01	0.82–1.23	137	0.92	0.78–1.10
Cirrhosis (5710–5719)	Male	2	0.88	0.24–7.25	11	2.38	1.33–4.76
	Female	1	0.47	0.08–18.40	3	1.20	0.41–5.79
	Combined	3	0.68	0.23–3.28	14	1.96	1.17–3.59
All accidents (8000–9499)	Male	32	1.64	1.16–2.42	29	1.30	0.90–1.94
	Female	10	1.46	0.80–3.04	12	1.73	1.00–3.35
	Combined	42	1.59	1.18–2.22	41	1.40	1.03–1.96

ed that this may be due to an underutilization of cervical cytology screening programs. Lowered risk of breast cancer among Native populations has been thought to be partially attributable to an earlier age at first birth.[3] Recent data indicate that Indian mothers are twice as likely as white mothers to give birth before age 20.[16]

Accidents accounted for approximately 14% of all SNI deaths over time, yielding a PMR 50% higher than the reference population. Mao et al[3] reported an SMR of 2.9 for accidental causes of death in Canadian Indians. An SMR of 3.9 was noted for accidental deaths among Native Americans in New Mexico.[2] It should be pointed out that the SNI reside primarily on reservations located in rural areas. Reasons for such excesses may be similar to those advanced for excesses in accident-related mortality observed among residents of rural areas across the United States.[18] These include poorly paved roads, higher speed limits, use of utility vehicles, and underutilization of seat belts.

SNI members also experienced a greatly increased risk of mortality from diabetes mellitus. A PMR of 3.17 was observed for both sexes combined (males=3.37 and females=3.00). While the proportionate excess was relatively stable over time, there was variation by sex. Male risks increased from 2.84 to 4.09 over time, while female risks decreased from 3.46 to 2.04. Increased risk of mortality due to diabetes has also been observed in other Native populations.[2,3] A health survey of SNI members in 1975 indicated that the prevalence of diabetes was 26%.[17] While diabetes is a controllable disease, it is obvious that there is still a great need to screen Native populations for diabetes as well as to ensure compliance with medical recommendations in order better to control the untoward health sequelae of this disease.

Elevated risks of mortality from infectious diseases were observed for the entire study period (PMR=2.48), increasing from 2.48 to 3.47 over time. While ratios for males and females were similar in the first period, ratios for males doubled by the last period to 4.98 and the female ratio decreased to 1.68. Higher risks of death due to infectious diseases have also been observed in other Native populations.[2,3] Of particular interest are mortality ratios observed for tuberculosis. Overall ratios increased from 1.86 to 10.34 over time. Three deaths were observed in the most recent period, while only 0.29 were expected. This finding is in stark contrast to reported observations of declining mortality rates from tuberculosis.[19] Mortality rates from tuberculosis among nonwhites have been declining along with those for whites, but still exhibit a twofold increased risk.[20]

CONCLUSIONS

This study presents results of a standardized proportionate mortality analysis of the Seneca Nation of Indians. While PMRs may not be as sensitive in determining the total force of mortality as standardized mortality ratios resultant from cohort studies, they are useful in estimating cause-specific mortality and highlighting differential patterns over time. The PMR methodology was employed in this study based on the data available for study and an interest in identifying changes in SNI mortality patterns over time relative to the experience of the general New York State population.

It is obvious from the data presented that more battles need to be waged successfully to combat avoidable, premature mortality in this Native American population. Based on this study, health care resources should continue to be directed toward programs aimed at reducing death and disability resulting from accidents, diabetes, tuberculosis, cirrhosis, and infectious diseases. Where possible, primary preventive efforts should be established and educational campaigns mounted to develop appropriate health behaviors among tribal members. Additional research needs to be undertaken to investigate accessibility, adequacy, and fuller utilization of health services provided to Native populations. The mere offering of certain health services is inadequate and mechanisms need to be established to ensure utilization of services and compliance with medical recommendations resultant from such offerings.

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The awarding of honors to medical clerks

Results of a national survey

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ABSTRACT. We conducted a national survey of medical schools to determine the frequency of the awarding of honors to third-year clinical clerks and fourth-year subinterns in internal medicine. Of 126 schools surveyed, 86.5% responded. Of those responding, 66% award honors to their junior clerks and 47% award honors to their fourth-year students rotating on the internal medicine service. Consistent criteria do not exist among programs and even within some programs for such awards. The number of students receiving the awards or the percentage of the class that is felt to qualify for honors is also highly variable.

The attainment of honors is an important criterion for residency selection as well as a positive predictor of future performance of candidates. The writing of the dean's letter, residency selection, and the date of the National Residency Matching Program results announcement are now occurring later in the academic year than previously, thus making the results of the fourth-year medicine rotations available to residency selection committees by the time choices have to be made. We conclude, therefore, that it seems useful for all medical schools to award honors not only to third-year clerks but also to senior students.

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Identification and recognition of medical student merit during clinical rotations by the awarding of honors is not universal among medical schools. In the process of deciding whether to recommend instituting the awarding of honors to fourth-year students in internal medicine, we reviewed the placement of an honors designation and the frequency with which medical schools use such a grade. The process of resident selection and the factors considered in such selection have been the subjects of many publications over the last several years.[1–8] Parameters of performance in medical schools as compared with performance during residency have also been studied.[9–11] In one study,[4] the receipt of honors by a clinical clerk was highly regarded in the evaluation of the clerk as a candidate for a residency position. Indeed, program directors considered a grade of honors during the clerkship of far greater importance than honors awarded during the preclinical years.

METHODS

At a meeting of the Executive Committee of the Department of Medicine of the Health Sciences Center, State University of New York at Stony Brook, the question was raised whether the department should begin to award honors to its fourth-year students, as it already did to third-year clinical clerks who were felt to deserve such an award. In response to this question, a committee was appointed to investigate the current practices of awarding honors in the clinical years in medical schools throughout the country. We conducted a national survey concerning the awarding of honors in both the third and fourth years and surveyed the literature to judge the importance of this practice.

A letter was sent to the Chairman of the Department of Medicine of all 126 United States medical schools, inquiring whether or not their departmental policies provided for the awarding of honors to third-year clinical clerks and/or to fourth-year subinterns. If the answer was yes, chairmen were asked whether or not there was a quota and whether or not they used written criteria in their evaluation of students for the awarding of honors. Chairmen were also asked to provide a copy of the written criteria, if available. A follow-up letter was sent to those chairmen who did not respond to the original request for information. No data were collected concerning clinical clerks or subinterns in any discipline other than internal medicine.

RESULTS

Honors for Clinical Clerks. One hundred and nine responses were received. Seventy-two of the responding schools award honors to third-year clinical clerks and 37 do not (Table I). Of the 72 schools that award honors, at least 19 also give grades of high pass, near honors, or just short of honors. The percent of clinical clerks awarded honors varies from "very few" to 25%. No school said that it had a fixed quota, but most schools reported that only 5% to 15% of their clinical clerks were given an honors grade. In some schools an

TABLE I. The Awarding of Honors to Third-year Medical Clinical Clerks and Fourth-year Medical Subinterns in 126 United States Medical Schools

	Number of Medical Schools	
	Clinical Clerks	Subinterns
Honors awarded	72 (66%)	51 (47%)
No honors awarded	37 (34%)	44 (53%)
Subtotal	109	95
No response	17	31
Total	126	126

From the Department of Medicine, Queens Hospital Center Affiliation of the Long Island Jewish Medical Center, Jamaica, NY (Dr Rosner), the Department of Medicine, Monmouth Medical Center, Long Branch, NJ (Dr Shagan), the Department of Medicine, Long Island Jewish Medical Center, New Hyde Park, NY (Dr Wallace), the Health Sciences Center, State University of New York at Stony Brook, (Drs Rosner & Wallace), and the Hahnemann University School of Medicine, Philadelphia, PA (Dr Shopin).

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A or A− grade or a numerical grade ranging from 83 to 96 automatically qualifies the clinical clerk for honors. In other schools, the top 5% to 20% of the class is considered for honors. Although most schools annually give honors to approximately the same percentage of their clinical clerks, one school reported that in some years no honors are awarded, whereas in other years relatively large numbers of honor grades are given.

Thirty-two schools submitted copies of their clerkship evaluation forms, and many more sent written descriptions of their grading systems. Very few schools, however, have detailed written criteria for the awarding of honors, a fact readily acknowledged in the letters by the departmental chairmen or their designees responding to our request for such criteria. Some schools make a general statement such as "the honors grade is to distinguish students who excel in meeting the course objectives," or "honors are awarded to the exceptional student, performing far superior to the expected level," or "a select few can be given a grade of honors if their performance is substantially better than their peers and they exceed the objectives of the clerkship," or "honors grade is given to a student whose overall performance has been sufficiently strong to merit special recognition."

Some schools have statements a little more specific than the aforementioned, such as "honors grade is reserved for the small group of students (about 10%) who excel in almost every phase of the clerkship and are clearly of outstanding ability. Such students should be considered candidates for AOA and for graduate training in highly competitive university programs." Alternatively, "honors grade is reserved for that small percentage of students (about 10%) who are clearly superior in every aspect of their performance during a clerkship, subinternship, or elective. These students possess not only unusual scholarship and industry, but will also have strong qualities of leadership and excellence in interpersonal relationships." Or, "honors are reserved for outstanding students who are exceptional in all aspects: knowledge, clinical skills, performance and judgment; and personal and humanistic qualities." Another approach states the following: "one of the best students in the group; has achieved predominantly excellent ratings in knowledge, skills (history and physical exam), analytic and/or planning ability and at least above average in presentations and participation. There are no problems with honesty, conscientiousness, reliability or patient relationship."

Some schools attempt to quantify the criteria for the awarding of honors as follows: "91.5% final grade plus a minimum grade of B on the written exam plus a minimum evaluation of very good on the oral exams"; or "above average to superior ratings in all clinical categories, a mean average of at least 80% on objective tests, and successful passing the oral exam and patient history and physical examination"; or "clinical grade of honors and a board score which is at or greater than the 85th percentile"; or "outstanding ward performance, outstanding interpersonal skills/professional attitude, outstanding written records, and a National Board minimum score of 100 points above the mean for our students taking the examination"; or "clinical grade of honors plus very good to outstanding written examination score"; or "more than half honor grades and more than half of the descriptors must be in the excellent column"; or "honors grade in *each* clinical rotation *and* written examination score of at least median for the group or honors in one rotation *and* high satisfactory in the other *with* an examination score in the top 25 percent of the group."

Nearly all schools use evaluation forms, some of which are very simple and provide for one-page "check-offs" and/or narrative, whereas others are quite elaborate and include detailed assessments of the students' intellectual capacity and ability to synthesize, fund of general medical knowledge, growth in fund of knowledge of discipline, performance level (with attention to consistency and performance at the level expected), quality of histories and physicals, quality of presentations, judgment, professional habits and attitudes (including honesty, industry, initiative, responsibility, handling of criticism), and relationships with patients and their families, peers, and superiors.

Most clinical clerkships also require oral, practical, and written examinations, and many schools use the National Board of Medical Examiners' test as an additional criterion in evaluating the assignment of honors. In addition to awarding honors for outstanding clinical clerks, a handful of schools give a best student award for the top student of the medical clerkship who has demonstrated leadership qualities, outstanding scholastic performance, and significant research abilities and accomplishments.

Departments of medicine at two schools have a special Honors in Medicine program by invitation only for all students receiving an A or A− in the third-year medicine clerkship. At one school, students who choose to participate in the program work closely with a faculty preceptor on a project of their choice. Students who complete their projects and submit written reports, which are then reviewed by the Honors Committee, are recognized at the time of graduation.

One school formerly awarded honors to clinical clerks but no longer does so, and three schools that formerly did not grant honors to clinical clerks now do so. At least seven schools assign honors to third-year clinical clerks but not to fourth-year subinterns, and at least three schools award honors to subinterns but not to clinical clerks. Several schools give combined third- and fourth-year honors.

Honors for Subinterns. Only eight schools have written criteria whereby honors are awarded to fourth-year subinterns. Three schools have a policy statement such as an honors grade is awarded "in recognition of a clearly superior mastery by the student of the body of knowledge and skills covered in a given course or clerkship," or to the "exceptional student," or for "performance truly exceptional; outstanding in all or nearly all respects." Two schools attempt to quantify the assigning of honors to students with an "overall average of 4.5 or better [on a scale of 1 to 5]" or "average score greater than 4.0 and the majority of overall evaluations are honors with at least one attending honors evaluation and test score greater than 500." One school states that "honors grade is recommended for the student who demonstrates outstanding knowledge and clinical judgment, technical skills, professional initiative as well as professional maturity as student/physician. In some cases students with outstanding qualities in some of the above criteria and commendable performance in others are also recommended for Honor grades." Another school asserts that for honors grade "the student demonstrated a good data base and had the ability to integrate it in the management of patients; interacted well with patients, medical staff, students, and nursing staff; showed good technical skills; assumed full responsibility for patient management and was able to function independently. Overall, the student functioned at intern level."

Finally, one school's policy is that an honors grade "is reserved for those who are clearly superior in every aspect of their performance during a subinternship, or elective. It should be given to subinterns who perform as well as a good intern with whom they are paired. The student is efficient with a typical case load (four patients) and can handle a seriously ill patient with an intern's level of skill. Fund of knowledge should be good in all areas and outstanding when pertaining to specific problems in their patients. They should show outstanding initiative and skill in identifying the important questions/issues of care in their own patients and, within the limits of their training, be guiding care rather than simply following directions. The Honors student accepts responsibility for the education of others on the ward team and is consistently motivated by high standards of scholarship and patient care."

With rare exceptions, honors are awarded to subinterns without a written examination and are based solely on subjective evaluations by house staff and attending physicians.

The selection of students who will receive honors both for third-year clinical clerkships and/or for fourth-year subinternships is usually made by an evaluation committee or the departmental executive committee following the completion of the clerkships and subinternships.

Reasons for Not Awarding Honors. Among the 36 schools that stated they do not award honors, all but seven did not offer a reason. One departmental chairman responded, "we don't have honors for anything and I doubt that we ever will." Another chairman said that "I can speak for all medical students, not only those in Internal Medicine, since we have a school policy. That policy is that we do not provide grades or honors to our students at any level. I must admit that the question arises every several years about whether or not this policy should be changed. Nonetheless, it currently stands as noted." Another respondent asserted that their university "does not award honors in the clinical years. This is a policy of the institution and, therefore, there can be no written criteria for such an evaluation." Yet another chairman said "we do not at this time award honors to third-year clerks and fourth-year subinterns. I think, however, that it is an excellent idea."

Another response was the following: "Some departments, ie, Neurology, Pediatrics, give awards to one or two students in the Junior and Senior classes. The faculty of the Department of Medicine has discussed this policy on numerous occasions and decided that we would not give such awards. We usually have five, ten or fifteen percent of any given class who do an outstanding job. We have felt that it was perhaps inappropriate to select one or two for an award. Therefore, we have not engaged in this activity."

Yet another school replied that: "We do not give honor ratings to either our third or fourth year medical students. The third-year medical students receive a letter grade (A, B, C, etc.). The fourth-year students receive either a pass or fail rating. We formerly awarded honor ratings to the fourth-year clerks, however, it was felt by the general faculty, not the faculty of the Department of Medicine, that this was abused and the procedure was discontinued. I favor the availability of an honor's rating to recognize an excellent performance by the student."

Finally, one departmental chairman wrote: "The Department of Medicine at our University does not award Honors to third year clinical clerks or subinterns in recognition of excellence. Indeed, I am opposed to this practice, since it potentially causes extra competitiveness between fellow students and does not recognize the excellent contributions of medical students who are not recipients of awards."

Among the 36 schools that do not award honors to clinical clerks or subinterns, at least seven schools give a special award "for excellence in internal medicine" to "the best overall clinical clerk," or "the most outstanding student on the clerkship," or "the student with the highest grade as a third-year clinical clerk," or "the student who has most distinguished himself or herself in internal medicine." Some schools give letters of commendation in addition to or instead of awarding honors. The practice of not awarding honors was not less common in newly established medical schools compared to the very prestigious schools, which might consider all their students to be excellent.

DISCUSSION

Of 126 programs surveyed, 86.5% responded. Of those responding, 66% award honors to their junior clerks, and 47% award honors to their fourth-year students rotating on the internal medicine service. Consistent criteria do not exist among programs and even within some programs for such awards. The number of students receiving the awards or the percentage of the class that is felt to qualify for honors is also highly variable.

Yet several authors have noted that the attainment of honors is an important criterion for being selected by a residency selection committee and also important for predicting the performance of the resident during his or her first year. Young et al[1] stated that the use of the "attainment of an honors grade in clinical medicine seems appropriate as a selection criterion since the clerkship defines clinical as well as cognitive skills." They also assert that "performance in clinical medicine is one of the few variables that has been shown to correlate with judgments of clinical performance during the residency." Wagoner et al[3] also indicate that class rank and honors grades are important. In another study, honors in the clinical clerkship ranked fifth in importance among parameters used by program directors to evaluate candidates for residency.[4] Hughes, Golmon, and Patterson[6] indicate that their faculty felt that the student's grade on the first part of the National Board of Medical Examiners examination, the third-year medicine clerkship grade, and academic honors were, in that order, the best predictors of the faculty's rating of students, and that the best predictors of National Residency Matching Program Rank were these same test scores and academic honors along with the third-year medicine clerkship grade. Weiss et al[12] note that "the present and overwhelmingly prevalent system categorizing clerkship performance as honors-pass-fail is capable of predicting performance during the PGY-1 residency." Keck et al[10] point out that the fourth-year internal medicine clerkship instructor's evaluation of students was significantly predictive of subsequent internship performance. Consistency of performance in medical school by the student was felt by Gardner[11] to be important in predicting the performance of that student in his first year of surgical internship.

Since so many program directors and residency selection committees feel that the achievement of an honors grade is an important parameter in their selection of candidates for residency as well as a positive predictor of the future performance of candidates, it seems useful for all medical schools to award honors in the third-year clerkship. It is helpful to the student in achieving his or her optimum placement and to the residency program director or selection committee in making their choices.

Honors at the fourth-year level are considered somewhat less important. Part of the reason seems to be that the results of the fourth-year medicine rotations have not been generally available to the selection committees by the time choices had to be made. Since the writing of the dean's letter, residency selection, and the date of the National Residency Matching Programs' results are now occurring later in the academic year than previously, the awarding of honors in the fourth-year medicine rotation may assume increasing importance.

We therefore conclude that it is reasonable, whatever the grading preference of the school, to have parameters for the awarding of honors at both the third- and fourth-year levels. These criteria should, however, be consistent within the medical school for all departments. The criteria and parameters should clearly reflect the overall performance of the student during his or her clerkship—academically, in patient care performance, and in the student's attitudes and functioning with patients, colleagues and other professionals with whom he or she works. If awarded in this manner, a grade of honors, especially if achieved in both the third and the fourth years of clinical medicine training, can accurately predict the eventual functioning of the student during the first year of postgraduate training, whatever the specialty of that training.

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. . . Of great interest to researchers are genetically linked conditions common to Indians. Gallbladder disease occurs more than twice as often among Indians in the Southwest as among non-Indian populations. Gallbladder disease among these Indians is associated with a low average age for cholecystectomies, a high incidence of common duct stones, and a high rate of cancer, chiefly gastric and biliary. Hepatitis is 26 times more common among the Navajos, even with those who do not have alcohol available, than among the general population. Diabetes is reported commonly in many tribes, including the Seneca. Certain blood groups are characteristic nationally; type O is predominant (83 percent in Navajos), and almost 100 percent are Rh positive. Hemoglobinopathies are absent, but methemoglobinemia is quite common. Newborn infants tend to show jaundice.

There is no good evidence to show that, aside from diabetes, any of these conditions are common among the Seneca, who have been studied by Doeblin and his co-workers. In their genetic studies of Seneca Indians, they have reported on haptoglobins, transferrins, G-6-PD deficiency hemoglobinopathy, color blindness, blood groups, diabetes, and hyperglycemia. Doeblin et al. also reviewed different morphologic traits, such as clinodactyly, mid-digital hair, ear-lobe attachment, hyperextensibility of the thumb, and dermatoglyphics.

Otitis media is a very common disorder among the Seneca. The possibility of a genetic variation which makes otitis more common in Indians has not been ruled out. But most authors associate the high incidence of this infection with nutritional and other undefined causes.

. . . There has been a tremendous improvement in Indian health nationally since the Indian Health Service took over responsibility for Indian health in 1955. Average life expectancy for Indians has increased from 60 to 65 years, compared with average life expectancy for whites, which has increased from 68 to 71 years (still leaving a marked discrepancy). Deaths have decreased from 50 to 90 percent for such disorders as influenza, pneumonia, certain diseases of early infancy, tuberculosis, enteritis, and other diarrheal diseases. The infant death rate has decreased from 62.5 to 18.7 deaths per 1,000 live births, a 70 percent decrease. Maternal mortality rates decreased by 80 percent.[16] There is still a lower incidence of heart disease, cerebrovascular accidents, and malignant neoplasm among the Indians than among the rest of the population.[1] However, there has been no reduction in the number one cause of death: accidents. Also, the incidence of cirrhosis of the liver, diabetes, and suicide is increasing. . . .

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Lyme disease: A review of its clinical features

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Lyme disease (LD) is a complex, multisystemic infectious disease caused by the spirochete *Borrelia burgdorferi*.[1] The disease affects people of all ages and both sexes and has become a major tickborne illness in the United States. Since its recognition in southern Connecticut in 1975, it has been spreading throughout the United States, and in certain regions, including Westchester County and Long Island, it has assumed the pattern of an emerging epidemic.[2,3] The true incidence is unknown due to underreporting. Cases have been described in Europe, Russia, Canada, China, Japan, and Australia.[4,5]

This paper reviews the history, epidemiology, clinical manifestations, laboratory testing, and treatment of LD. In addition, recent advances in the understanding of disease pathophysiology are presented.

HISTORY

Lyme disease was first described in 1975 in children and adults who lived in the Lyme, Connecticut, area, many of whom were initially diagnosed as having juvenile rheumatoid arthritis. This unusual clustering of the disease was brought to the attention of the rheumatology department at Yale University School of Medicine. Subsequent investigations by Dr Allen Steere and his colleagues resulted in the description of "Lyme" arthritis.[6] In the first report, Steere described 51 cases—39 children and 12 adults—who had acute episodes of an intermittent mono- or oligoarthritis. The onset of the arthritis was often preceded by an expanding red rash, later recognized as erythema chronicum migrans (ECM), which sometimes occurred after an arthropod bite. This clinical picture and the occurrence of the rash and the arthritis predominately in the summer and fall suggested an arthropod-borne illness.

The ECM rash, pathognomonic for LD, had been described in Europe in 1909 by a Swedish physician, Afzelius,[7] who observed the rash in one of his patients bitten by a tick from the *Ixodes* family. In 1922, Garin and Bujadoux[8] described neurologic sequela following an ECM rash. Subsequently, numerous cases of ECM were reported in Europe and were noted to be associated with the bite of a sheep tick, *Ixodes ricinus*.[9] In 1941, Bannwarth[10] described an individual with an ECM rash at the site of a tick bite, in whom a radiculopathy, paralysis of one arm, and meningitis subsequently developed. The clinical picture of chronic lymphocytic meningitis with neuropathy and radiculopathy became known as Bannwarth's syndrome.[11] Swedish physicians Lennhoff and Hellerstrom[12] postulated a spirochetal etiology for ECM and meningitis. This led to the common practice of treating ECM empirically with penicillin. The first case of ECM in the United States was reported by Scrimenti[13] in 1970 from Wisconsin. After the description of LD and the recognition that ECM was part of the clinical picture, it was suspected that ticks were important in the transmission of the illness. The tick *Ixodes dammini* was subsequently identified as the vector for LD transmission in the Northeast.[14] The exact etiology of LD was not known until spirochetes were cultured from the ticks by Willy Burgdorfer[15] in 1982 and later from the skin, blood, and spinal fluid of infected patients.[16,17] In 1984, the newly discovered organism was named *Borrelia burgdorferi* after its discoverer. This then allowed the development of diagnostic tests for LD and the recognition of an expanded clinical spectrum of the disease.[18]

EPIDEMIOLOGY

Lyme disease occurs predominately in three areas of the United States: the Northeast, with a clustering of cases in southern Connecticut and Rhode Island, Massachusetts, New York (Westchester County and Long Island), and the states along the Atlantic coast; the Midwest, mainly Wisconsin and Minnesota; and the West, mainly Washington, Oregon, and northern California.[19] These areas correspond to the distribution of the *Ixodes* ticks. The tick vectors vary from one region to another,[20] eg, *Ixodes dammini* in the East and Midwest and *Ixodes pacificus* in the West. Other tick vectors include *Ixodes scapularis* in the Southwest and *Ixodes ricinus* in Europe. *Dermacentor variabilis* (American dog tick) is not involved in LD transmission among humans.

Entomologic surveys in Connecticut have also demonstrated *B burgdorferi* in the gut tissues of deer flies, horse flies, and mosquitos, although any role in the transmission of LD by these vectors is unproven. The rapid spread inland may be caused by migrating birds.

Ixodes dammini, or deer tick, the vector for LD in the Northeast, is an eight-legged, wingless, parasitic arachnid. It has different hosts in its adult, nymphal, and larval stages.[21] Adult ticks are abundant in fall and spring. They prefer to feed on larger animals such as the white-tailed deer. Man is only an incidental host. Nymphal ticks, which are numerous in spring and summer, parasitize small and medium-sized mammals such as the white-footed mouse (*Peromyscus leucopus*), squirrels, and occa-

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sionally man. The nymphal ticks are no larger than pencil tips. Larvae, abundant in the late summer and fall, also commonly feed on the white-footed mouse. Animals bitten by an infected tick become a reservoir of *B burgdorferi* and are responsible for sustaining the spirochete in the environment. The immature nymphal ticks are highly aggressive, and it is this form that is most responsible for transmission of LD to man. The variable prevalence rates of LD from one geographic area to another are explained in part by differences in the percentage of infected *Ixodes dammini* ticks in the different areas.[21] The LD season extends from early spring to late fall. The peak incidence in the Northeast is in the summer, which correlates with the nymphal stage of the tick life cycle. The explosion in the deer population in rural and suburban areas appears to be directly related to the increase in *Ixodes dammini* and focal epidemics of LD.

MICROBIOLOGY

B burgdorferi is a small, unicellular microorganism with flagellae.[16] The spirochete carries various plasmids (self-replicating cytoplasmic DNA fragments) and grows in modified Kelly's medium.[16,22] Antigenic variation in the spirochete may occur with multiple subpassages in culture.[23] This might account for some of the differences observed between different laboratories in testing for anti-Borrelial antibodies to diagnose LD.

B burgdorferi is very difficult to culture from human tissues and fluids. Although the reason for this is not known, some researchers postulate that this may be due to a scarcity of organisms and a predominately intracellular location, but this theory lacks definitive proof. Visualization of the spirochete in tissue sections is also difficult and is not useful for routine diagnosis. The microorganism stains with Dieterle silver or can be detected by the fluorescent antibody technique.[24,25]

CLINICAL FEATURES

Early Disease. Soon after a tick bite, there may be a local reaction caused by "mechanical irritation," which can last for 2–3 days. This is unrelated to borrelial infection. However, with infection, a flu-like illness may occur within days to weeks. This may be associated with chills and fever, as well as malaise, fatigue, backache, headache, and lethargy. The early manifestations and the frequency of their occurrence are described in Table I.[26]

Erythema chronicum migrans, the hallmark of LD, usually begins 3–30 days after tick exposure. Benach and Coleman[27] in a study of LD in New York State found that only a minority of patients with ECM recalled a tick bite. ECM occurs in 60–75% of all patients with LD. It often occurs in the thigh, groin, or axilla. It begins as a red macule or papule and expands in a circular fashion to achieve varying diameters, occasionally very large (60 cm). The lesions tend to have bright red borders with central clearing and induration. Vesiculation or necrosis are rare. In almost half the patients, smaller, multiple annular skin lesions develop soon after the appearance of the initial ECM rash.[26,28] ECM usually fades without scarring within 3–4 weeks, although it may last for some months. Histologic evaluation reveals a lymphoplasmacytic infiltrate in both the superficial and deep dermis around dermal blood vessels and appendages.[29] Spirochetes can sometimes be demonstrated in skin biopsies.[30]

TABLE I. Early Features of Lyme Disease*

Symptoms/Signs	Frequency (approx %)
Constitutional symptoms (malaise, fever, fatigue)	80
Headache	65
Neck stiffness	50
Arthralgia	50
Myalgia	45
Lymphadenopathy	40
Backache	25
Sore throat	15
Malar rash	13
Conjunctivitis	10

* The frequency of these features was based on a population of patients who had ECM (adapted from Steere et al[26]).

Additional dermatologic manifestations include malar rash (13%), diffuse erythema, urticaria, and rarely, acute septal panniculitis (tender, nodular skin lesions).[31] Other early clinical findings are shown in Table I.

The intensity of early LD can be mild, with ECM and minor constitutional features, or very severe, with multiple skin lesions, high fever, and marked fatigue, arthralgia, and headache. Symptoms often fluctuate in intensity over time. The differential diagnosis at this time includes viral infections, infectious mononucleosis, secondary syphilis, systemic lupus erythematosus, and other connective tissue diseases.

Late Disease. Several weeks to months after initial exposure and usually many weeks after ECM, untreated patients may develop symptoms and signs of more serious organ involvement. The major problems at this stage may be neurologic, cardiac, or arthritic.

Neurologic. Approximately 10–15% of patients develop frank neurologic abnormalities, as shown in Table II.[32] Cranial neuropathy, especially Bell's palsy, is a frequent neurologic feature. This may be bilateral, and LD is the most common cause of bilateral Bell's palsy in a Lyme disease-endemic area.[33] Even without frank meningitis or encephalitis, patients often experience moderate to severe headache associated with sleep disturbance and difficulty in concentration. Bannwarth's meningoradiculopathy may be partial or full blown, but is seen more frequently in Europe than in North America. Approximately 60% of patients will have multiple neurologic features simultaneously. The neurologic symptoms are often migratory, and typically persist for months.[32] The cerebrospinal fluid

TABLE II. Neurologic Features of Lyme Disease*

Feature	Frequency (%)
Meningitis	100
Encephalitis	29
Cranial neuritis (Bell's palsy) (bilateral in ⅓)	50
Peripheral neuritis	29

* Thirty-eight patients, all with meningitis (adapted from Pachner and Steere[32]).

(CSF) findings are generally consistent with an aseptic meningitis (lymphocytic pleocytosis, with normal to elevated protein, normal glucose, and no bacteria). Both an increased IgG/albumin ratio and oligoclonal bands are found in some cases.[34] Antibodies to *B burgdorferi* may be detected in the serum or CSF in LD with neurological involvement.[35] The neurologic symptoms gradually remit, although the encephalitic features and motor radiculopathies may persist for many months.

Cardiac. In approximately 8% of patients, cardiac involvement develops, often within a few weeks to months of disease onset.[36] It is generally brief and mild, consisting of varying degrees of atrioventricular block, including complete heart block. There may be no symptoms with milder involvement, but a temporary pacemaker may be required with more severe degrees of block. The block is usually above the bundle of His.[37] Some patients have clinical and investigative evidence of diffuse myopericarditis.[36] The symptoms in these patients may persist longer than the few weeks usually seen with milder involvement, and a fatal case of cardiac involvement in LD has been described.[38] In that case, a few spirochetes were seen in the myocardium. The differential diagnosis of cardiac involvement in LD includes rheumatic fever, viral myocarditis, and connective tissue diseases.

Arthritic. While arthralgias are common during the early stage of LD, frank arthritis usually does not begin until after a few months of infection and may be delayed for some years. Approximately 60% of untreated patients will develop arthritis within two years.[39] The arthritis is often abrupt in onset and usually affects only one or a few joints (mono- or oligoarticular), primarily the knees (Table III). The arthritis is characterized by remissions and relapses, each cycle lasting from days to months. Low-grade fever and fatigue may accompany episodes of synovitis. Even if untreated, the arthritis may eventually remit spontaneously. Chronic persistent synovitis may develop in 10% of patients with untreated Lyme arthritis, and this can result in erosive damage.[40] These patients may not respond well to antibiotic therapy.

The synovial fluid is inflammatory, with the white blood cell count usually in the thousands and with a predominance of polymorphonuclear leukocytes. On pathologic examination, Lyme synovitis features an intense lymphoplasmacytic infiltrate similar to rheumatoid arthritis.[25] Scarce spirochetes or spirochetal antigens have been demonstrated in a perivascular distribution in the synovium using Dieterle silver stain or monoclonal antibodies.[24,25] *B burgdorferi* has never been cultured from synovial tissue and only once from synovial fluid.[41] However, patients with Lyme arthritis usually have antibodies to *B burgdorferi* in both blood and synovial fluid.[39] The differential diagnosis includes other causes of acute and chronic monoarthritis or oligoarthritis in children and adults, such as juvenile rheumatoid arthritis, septic arthritis, crystal-induced arthritis (gout, pseudogout) and Reiter's syndrome.

TABLE III. Lyme Arthritis*

Joint Involved	Frequency (%)
Knee	96
Shoulder	50
Ankle	43
Elbow	39
Temporomandibular	39
Wrist	32
Hip	29
Small joints	14

* Based on 28 patients with frank Lyme arthritis (adapted from Steere et al[39]).

Other Features. Panophthalmitis, ischemic optic atrophy, and interstitial keratitis have been rarely reported.[42] In addition, myositis, hepatitis, and adult respiratory distress syndrome have been described.[43-45] Chronic central nervous system infection with *B burgdorferi* has been incriminated as a cause of some cases of multiple sclerosis, amyotrophic lateral sclerosis, and Alzheimer's disease.[46,47] However, there have been no studies to confirm this, and we have been unable to demonstrate evidence of borrelial infection in a large number of patients with Alzheimer's disease.[48]

Babesiosis, a tickborne disease resulting in hemolytic anemia, has been described in a patient with coincident LD.[38] Two chronic cutaneous features have been described extensively in Europe.[49] Acrodermatitis chronica atrophicans usually appears as a violaceous plaque or nodule on the dorsum of hands or feet. The lesions develop, on average, three years after the tick bite or ECM. It will resolve with appropriate antibiotic therapy. Lymphocytoma cutis, which resembles a cutaneous lymphoma, is generally seen on the earlobe.

Congenital Infections. Maternal infection with *B burgdorferi* during early pregnancy may result in adverse fetal outcome.[50,51] However, preliminary results of a controlled study did not show an increased prevalence of congenital malformations among infants with detectable anti-borrelial antibodies in cord blood.[52]

PATHOGENESIS

The widespread tissue distribution of *B burgdorferi* suggests that the spirochete spreads hematogenously and persists in affected tissues. During the first few weeks of illness, immunity appears to be suppressed with a poor response of mononuclear cells to both *B burgdorferi* antigens and to mitogens.[53] Also, during this time, there may be little or no detectable serum antibody response, although serum IgM levels may be elevated.[54] After the first several weeks of infection, mononuclear cells begin to respond to Lyme spirochetal antigens, and this is soon followed by a detectable serum antibody to *B burgdorferi*.[55] Initial antibody response is mainly IgM and is restricted in its specificity, but with continuous infection, IgG antibodies directed against a large number of borrelial antigens appear.[56] Both cellular and humoral antigen-specific responses are concentrated at the sites of inflammation. That is, borrelial reactive lymphocytes and anti-borrelial antibodies have been found selectively concentrated in the cerebrospinal fluid of patients with neuroborreliosis and in the synovial fluid of patients with Lyme arthritis.[53,57]

In addition to these indicators of a local immune response to the spirochete, patients with active disease have in vivo and in vitro evidence of B-cell hyperactivity including the presence in serum of high IgM levels, cryoglobulins, and immune complexes.[58,59] This suggests that factors in addition to direct spirochetal invasion, such as immune mechanisms, may be important in disease pathogenesis. This may be especially true of the arthritis in which, as previously mentioned, the organism has not been cultured from inflamed synovial tissue. In support of this hypothesis, the immune response gene DR4 occurs with increased frequency in patients with chronic Lyme arthritis.[60]

A rat model of LD has recently been described, and this might lead to further insights into the pathogenesis of the organ involvement in this illness.[61]

DIAGNOSTIC LABORATORY TESTING

In an endemic area, LD is easily diagnosed on clinical grounds if the patient has ECM. Patients who lack a history of tick bite and ECM present more diagnostic difficulty. The Centers for Disease Control[62] has developed criteria for the diagnosis of LD that are different in endemic and nonendemic areas (Table IV). However, these criteria were devised prior to the development of newer diagnostic tests and their sensitivity, specificity, and predictive value have not been thoroughly tested. Furthermore, the distinction between an endemic and nonendemic area may be a problem and is constantly changing. For instance, LD is highly endemic in north-central but not southern Westchester County.[63]

This situation and the difficulty in demonstrating the organism in tissues and culturing it from fluids have resulted in dependence on serologic testing to aid in the diagnosis of LD. Nonspecific laboratory abnormalities that correlate with active disease include a high sedimentation rate and high serum IgM levels. The most specific test in common use is the measurement of the antibody response to *B burgdorferi* in serum. This can be performed by a number of techniques including immunofluorescent antibody assay (IFA), enzyme immunoassay (ELISA), and Western blotting.[64-66] The ELISA technique is currently accepted as the preferable way to test for LD routinely, although the Western blot technique may be more sensitive and specific. However, all these tests suffer from a lack of standardization of reagents and quality control on a national level because LD testing is in its infancy.

Since measurement of an antibody response is an indirect way to test for the presence of active infection, there are major limitations to the use of present testing for LD. These limitations fall into four categories: 1) the timing of the antibody response; 2) the effect of treatment on the antibody response; 3) false positive and false negative results; 4) the diagnosis of active versus past infection.

Timing of Antibody Response. It generally takes seven to ten days from the time of initial infection for serum antibodies to *B burgdorferi* to develop, and by two to three weeks in untreated patients, antibody can reliably be detected. Thus, patients with early symptoms, including flu-like illness, arthralgias, and even ECM, may test negative.[55] Furthermore, the earliest response is usually IgM which is the most difficult to measure, although newly developed tests may prove helpful.[67] In a patient with systemic symptoms suggestive of early LD who tests negative, it is advisable to perform repeat testing some weeks later before negating the diagnosis.

TABLE IV. Diagnostic Criteria for Lyme Disease, Centers for Disease Control, USPHS

Endemic area
  Erythema chronicum migrans (ECM)
  Antibody titre ≥1:256 and involvement of ≥1 organ system*
Non-endemic area
  ECM with antibody titre ≥1:256
  ECM with involvement of ≥2 organ systems*
  Antibody titre ≥1:256 and involvement of ≥1 organ system

* Either musculoskeletal, neurologic, or cardiac.

Effect of Treatment on Antibody Response. Patients who are treated early with antibiotic therapy may never develop a detectable antibody response.[68] This may be analogous to syphilis, in which early antimicrobial therapy can abort development of a measurable humoral response. However, some of the treated LD patients have continued chronic symptoms including severe chronic fatigue, numbness of the extremities, memory loss, and joint pain.[68] That these patients had early Lyme disease is not in doubt, since they had ECM and many had demonstrable in vitro lymphocyte response to *B burgdorferi*. What is less clear is whether ongoing symptoms of this nature are truly related to ongoing borrelial infection in the absence of an antibody response. This will not be resolved until methods are available to detect reliably the presence of active *B burgdorferi* infection. In contrast to this situation, untreated patients who go on to develop more serious organ involvement (cardiac, neurologic, arthritic) uniformly test positive for antibody and often in very high titers.[35,39]

False Positive/False Negative Results. False positive results have been described in patients with treponemal diseases and other borrelial infections, eg, syphilis and relapsing fever.[69] The ELISA test for LD is usually negative in patients with viral infections, infectious mononucleosis, and connective tissue diseases.[64,65] A problem encountered in areas highly endemic for LD is the existence of asymptomatic seroconverters which may be as high as 6–8% of the population at risk.[70] There have been no studies to determine whether the nature of the antibody response in these patients differs in quality or quantity from those antibody-positive patients who had clinically apparent LD. The frequency of false negative results is unclear, since there exists no reliable test for ongoing infection, as previously discussed.

Diagnosis of Active versus Past Infection. In patients who have had LD with late symptoms and a positive IgG antibody response, the antibodies may be detectable in high, although gradually reducing, titers for many years (personal observation). It is uncertain whether or not high antibody levels protect against reinfection. In patients who have had symptoms suggestive of reinfection or relapse there have been no studies of the nature of any changes in antibody response (ie, titer, IgM:IgG ratio, repertoire of antigenic reactivity). Therefore, the diagnosis of reinfection in these patients must of necessity be based almost exclusively on clinical grounds.

A strong, specific cellular immune response to *B burgdorferi* occurs early in LD and persists for years.[71] This cellular immunity may precede the development of a measurable antibody response. Therefore, a lymphocyte stimulation test might be useful to document past exposure to *B burgdorferi* and might be helpful in the diagnosis of LD in difficult cases. At present, this test is performed at a limited number of institutions and is not available for routine clinical use.

Under active investigation are methods to detect *B burgdorferi* antigens, using monoclonal antibodies, in the urine and other fluids of patients with active LD. Potentially the most sensitive test might be the detection of borrelial-specific DNA or RNA in the white blood cells of patients with LD using the techniques of molecular biology to develop the appropriate probes.

TREATMENT

The optimal treatment of LD at any stage is not uniformly agreed on because of a lack of controlled studies. On the other hand, knowledge of the in vitro antibiotic susceptibility of the organism has provided guidelines to therapy that have proven adequate in most instances.[72] Also, because of the slow replication of the spirochete, high and sustained antibiotic levels are required in blood and tissues reliably to eradicate the infection.[73] Our treatment recommendations are based on an analytical review of the literature and our personal experience.[74]

Early Disease. Appropriate early treatment of LD is essential to prevent late complications. Adults, including nonpregnant women, and children more than eight years old should be treated with oral tetracycline, 250 mg four times a day, or doxycycline, 100 mg twice a day, for two to three weeks. The duration of treatment is empirical, although one study showed no difference in outcome when patients were randomized to a 10- or 20-day course of tetracycline.[75] Treatment failures with tetracycline, however, have been reported, and we prefer to use doxycycline for three weeks.[76]

For children less than eight years old, penicillin V, 250 to 500 mg four times a day or 50 mg/kg/day in four doses, or amoxicillin, 250 mg three times a day or 20 mg/kg/day in three doses, each given for two to three weeks, is effective.[74] For adults or children allergic to tetracyclines or penicillins, erythromycin can be used in a dosage of 250 mg four times a day or 30 mg/kg/day, but this regimen appears to be less effective. Because of this, some have advocated desensitization to penicillin or the use of vancomycin, but studies with these regimens have not been reported to date.

For pregnant women, especially in the first trimester, oral penicillin or amoxicillin as above may be used, but because of concern about transplacental transmission of the infection, many would treat with intravenous penicillin or ceftriaxone as described below.

Late Disease. Patients with mild symptoms (ie, Bell's palsy, early mild arthritis) may be treated with doxycycline in the same dosage as for early disease, but this should probably be given for a longer time (four weeks). This course can be repeated if there is an incomplete response. For patients with more severe neurologic, cardiac, or arthritic involvement, intravenous penicillin G, 20 million units daily for 14 days, may be effective.[77] However, because of occasional treatment failures with this regimen, many are using ceftriaxone, 2 g intravenously daily for 14 days, as first line therapy for serious late LD.[78] This antibiotic effectively penetrates synovial tissue and the central nervous system. Amoxicillin, 500 mg, with probenecid, 500 mg, both given orally three times a day, has been used on occasion to treat patients with late LD. It is not advisable to use this in patients with central nervous system involvement, because probenecid theoretically interferes with the accumulation of penicillins in brain tissue.[79]

Intra-articular steroid injections are, in general, contraindicated, since they may diminish the effectiveness of antibiotics.[77] Arthroscopic synovectomy may sometimes be useful for recalcitrant Lyme synovitis.

Approximately 10% of patients started on antibiotics may experience an apparent worsening of symptoms during the first 24 to 72 hours of treatment.[75] This is similar to the Jarisch-Herxheimer reaction seen in the treatment of syphilis. This is a transient reaction and can be treated with salicylates or acetaminophen.

REDUCING THE INCIDENCE OF LYME DISEASE

Because Lyme disease is a tickborne illness, elimination of the tick or its reservoirs should be the best approach to prevention or eradication of the disease. Efforts to eliminate *Ixodes* ticks through aerial spraying of pesticide or using arcaricide (Permanon)-impregnated cotton batting known as "Damminix" have achieved only small success. Large-scale reduction in the deer population is impractical. Therefore, major preventive measures have yet to be devised. For individuals in endemic areas, wearing protective clothing and the use of tick-repellent sprays, such as N,N-diethyl-meta-toluamide (DEET), may offer some protection. At present, the most important strategy in prevention is public and professional education about the areas that are endemic for LD and the early clinical features of the disease, so that treatment can be given early in its course. A recent study does not support the use of prophylactic antibiotics after a tick bite even in an endemic area.[80] Newer diagnostic tests for active *B burgdorferi* infection, early or late, will lead to more rational therapy. New treatment regimens are currently being developed and tested.

In summary, LD is a complex, multisystemic illness caused by the tickborne spirochete, *Borrelia burgdorferi*. The diagnosis of LD should be entertained in any patient with a history of ECM rash and/or symptoms suggestive of LD, especially in endemic areas. Serologic assays for antibodies to *B burgdorferi*, although neither totally sensitive nor specific, are valuable for screening for past exposure to the organism. Early treatment with recommended antibiotic regimens is essential to prevent late sequelae.

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The confidential New York State Department of Health Universal New York Health Care (UNY*Care) proposal

Medical Society of the State of New York staff observations

DONALD F. FOY, MARIO V. MENGHINI

While initial review of the confidential Universal New York Health Care (UNY*Care) draft proposal of the New York State Department of Health (DOH) (which came into the possession of MSSNY through reliable sources) identifies health insurance elements and concepts meriting some consideration, it also reveals aspects of the program that conjure the ominous specter of the current state-controlled Medical Assistance Program (Medicaid), with its obvious ramifications. Aside from the Department of Health's seemingly gratuitous references in the proposal to New York State as "a leader in health policy," the "evident achievements" of its current system, its "commitment to the poor" (who, in our opinion, do not presently have reasonable access to quality health care), and its assertion that the state's Medicaid program of the mid-1970s was "the most generous Medicaid program in the nation" (benefits-wise, possibly; payment-wise, certainly not, with New York State ranking at almost the very bottom in the nation in terms of physician payments), some elements of the proposal are interestingly reminiscent of past and present health insurance initiatives. This would include the Massachusetts Universal Health Insurance Program which the DOH is evidently emulating and upon which it hopes to "improve."

THE UNY*CARE PROPOSAL

It is interesting to note the report's early assertion that while a completely tax-based system might be the most effective and equitable approach to achieving UNY*Care goals, such an approach is not "*initially*" feasible, thus leaving one to ponder possible designs of the state for future program funding, ie, a greater tax burden for the public at large and an insurance program that is public in nature, similar to the Medicaid and Medicare programs. However, at this juncture, the report espouses "strengthening" the system of private employer-based insurance, expansion of public programs (eg, Medicaid and Medicare), and restructuring of "certain" health programs to include such coverage as long term care. Furthermore, the proposal indicates that this "universal" approach is intended to replace the state's "current fragmented payment system" in three phases, with all residents eventually being issued a UNY*Care enrollment card, ostensibly including those residents participating in such health insurance programs as Blue Cross/Blue Shield, commercial health insurance plans, Medicaid, Medicare, workers' compensation, no-fault, etc, whose coverages would apparently be enhanced by a state-required "standard" or "minimum" level of benefits package yet to be finalized.

The first phase of the program would begin approximately 18 months following the enactment of pertinent legislation and would address coverage for the more needy population of New York State, comprising 4.5 million people (25% of the state's total population). Phase two would occur approximately two to three years later and would encompass enrollment of the remainder of the state's population (identified through tax and Social Security rolls). The third and final phase would include the provision of long term care. The proposal indicates that UNY*Care will lay the groundwork for achieving an ultimate goal of "*a single, standardized set of prices based on a common methodology that applies to all payors and for all New York State residents.*"

UNY*Care would not only set uniform rates, but would also be the central payor through a limited number of health insurers contracted by the state as fiscal intermediaries on a regional basis to perform claims processing functions.

The proposal asserts that the primary "virtue" of the UNY*Care system is the treatment of all patients in a similar manner, as well as the development of a predictable budget permitting elected officials and program administrators to plan for needed system growth, including increases in reimbursement, premium rates, and new state revenues. Consequently the new system would be in the complete control of the state—presumably the DOH—regarding benefit structure, payment levels, claims processing operations, medical policy, financing requirements, and all other predictable embellishments including quality and utilization review activities. This would presumably include audit, sanctioning, and other oversight processes anticipated to be objectionable to the medical community in view of the state's historic performance.

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More specifically, if the current Medicaid system and the activities of the state Department of Health are any criteria, the anticipated bureaucratic morass, the unilateral policy-making and reimbursement rate-setting processes, the administrative burdens, and the constant regulatory interference in the practice of medicine should be matters of grave concern to the state's provider community.

SIMILARITIES TO MEDICREDIT

Setting aside the generally ominous prospect of a state controlled UNY*Care system, the current proposal's requirement for employer involvement in the provision of health insurance coverage does have conceptual similarities in some respects to past and recently suggested AMA funding proposals for insuring employed and nonemployed persons, including the AMA's previously introduced (1975) Medicredit Bill on the federal level. For example, while the UNY*Care proposal calls for a minimum standard package of health care benefits to all employees and their dependents, with subsidies to those employers not able to offer such coverage, the Medicredit proposal advocated a comprehensive benefits package predicated on employer/employee contributions to health insurance coverage, with employers experiencing substantial payroll cost under the Medicredit proposal similarly entitled to federal cash assistance or a credit against income tax. Theoretically, UNY*Care's "minimum" benefits package approach could parallel Medicredit's comprehensive benefits recommendation in scope since it advocates a broad Medicare-type coverage with the addition of preventive care services, custodial care services (through suggested inclusion under Medicare), and possibly other services not normally covered by health insurers under basic programs.

SPECIFIC PROVISIONS OF UNY*CARE

The following are some provisions of UNY*Care that appear laudable on the surface, at least as stated objectives:

- expansion of public programs to cover all children from 0 to age 17, cover all persons under 100% of poverty, subsidize persons between 100% and 200% of poverty;
- a basic benefits package "likely" paralleling the Medicare program which would eliminate current health insurance coverage gaps, thus meeting the needs of the general population;
- primary care coverage encompassing prevention and maintenance health care services;
- coverage of the $540 Medicare Part A deductible for beneficiaries below 200% of poverty;
- the planned provision of long term care coverage;
- Medicaid physician reimbursements raised "to parity with other payors" (NOTE: This is tempered by the fact that UNY*Care will establish the uniform payment rates for the "other payors");
- a UNY*Care stop/loss subsidy provision which essentially limits employers' health care costs to basic annual amounts of approximately $25,000 each for acute and major medical care. (NOTE: Services covered beyond the stop/loss provision will be financed by assessing employers a surcharge on the premiums they pay for the first $25,000 of acute care and the first $25,000 of major medical expenses.)
- a uniform "one-payor" reimbursement system for all medical services (while this element of the plan appears to be impressive, factors relevant to its implementation may become controversial, eg, the extent to which UNY*Care health insurance payments may be negotiated in good faith with the state; the expectation of increased costs to health insurers, the business sector, and employees in order to adhere to state minimum benefits requirements; the possible adverse impact of the revamped system on employment within the health insurance industry, etc).

FLAWS AND DEFICIENCIES OF UNY*CARE

Despite the lofty espousals of UNY*Care and its few basic conceptual similarities to the AMA proposals cited, the UNY*Care approach as identified in the January 1989 confidential draft appears to be a program of socialized medicine flawed with planning deficiencies, prohibitive costs, nebulously defined provisions, and questionable bureaucratic intentions, primarily as follows:

- It is extremely disturbing (but not surprising) that no input has been sought to date from organized medicine by the Department of Health in the development of the UNY*Care program, despite the fact that it has apparently been in the planning and formative stages for quite some time; in fact no official notification to the physician community was made concerning this proposal, while the DOH announced the program earlier this year through the media and to consumer advocacy groups, eg, the New York State Health Care Campaign consisting of more than 70 consumer-oriented groups. (Instead, in a seemingly measured manner perceived to infer physician philosophical concurrence with state DOH views, the draft proposal quotes an August 1988 report of the New York Academy of Medicine addressing "the central failures of our present system" in covering the uninsured, thus implying a further justification for establishment of a state universal health insurance system.)
- While UNY*Care will develop a statewide schedule of payments for medical services, enroll providers, and "negotiate reimbursement," to what extent and with whom this negotiation process will be conducted—hopefully in good faith—is not actually specified; furthermore, with current state budget constraints projected to endure for the next three years, ie, $500 million in 1990; $600 million a year in 1991 and 1992—anticipated employer financial contributions notwithstanding—the viability of the UNY*Care proposal is highly questionable.
- In conjunction with the foregoing, the proposal does not identify whether the development of relative values and applicable conversion factors—as well as the possible eventual inclusion of capitation payment and managed care ("gatekeeper") methodolo-

gies under the system—will have the benefit of physician input despite a "universal" program intended eventually to encompass the resident community of New York State and impact on all providers of health care.

- UNY*Care will specify the basic benefits package, with no indication in the proposal as to whether the physician community or the health insurance industry, which provide and cover the services and are in sound positions to offer viable suggestions concerning adequate medical coverage, will have meaningful input.
- UNY*Care will employ *mandatory assignment* (for a basic set of services), a concept vehemently opposed by the Medical Society of the State of New York (MSSNY) and organized medicine throughout the country.
- The state will be the sole "actor," playing a "larger role" in controlling health care delivery in New York State—a role in which the medical community believes the state has not demonstrated proficiency, especially in providing adequate access to health care for the needy as exemplified by its costly but deficient Medical Assistance Program.
- The state will develop a budget for UNY*Care and determine revenue needs, as well as manage program funding and expenditures; again, a role for which, in our opinion, it does not have a proven track record, as evidenced by a Medicaid program in disarray and devoid of cost-effective health care delivery.

SOURCES OF OPPOSITION

While the New York State Department of Health's UNY*Care proposal appears enormously ambitious in its design to cover essentially the entire state population in three phases, it also appears to be presumptuous and controversial to the extent that it may face serious opposition from the following sources:

- *New York's health insurance industry* may oppose state interference through insurer-required "standard" or "minimum" benefits coverage resulting in additional costly services to their programs and increased expenditures—particularly coverage for preventive care services currently excluded from most policies, as well as any ancillary care services often covered under optionally purchased riders (usually on a group coverage basis) because of the anticipated high utilization of such services; furthermore, the draft proposal does not clearly identify the role of the health insurance industry in the UNY*Care process, including any input relative to the setting of premium incomes sufficient to compensate for the enhanced benefit structures (other than the indication that UNY*Care policies may be purchased from "any State-approved" insurance company); or clearly identify whether health insurers will have appropriate input relative to fiscal intermediary claim review processes impacting on eventual insurer payouts;
- *organized medicine* for the valid concerns previously identified;
- *the hospital industry*, which is as sensitive as the physician community to state-controlled health care policy, and should be sensitive to proposed UNY*Care cost-control measures affecting the current hospital reimbursement methodology (hospital payments would be based upon a single all-payor DRG-based rate with some variations by region, hospital type, etc, as compared to the current DRG/case-based, prospectively negotiated arrangement);
- *the business community* which will likely be required to assume an increased economic burden to finance a "minimum" benefits program essentially comparable to the broad scope of the Medicare program, recast to include even more costly benefits, and with tax penalties imposed on those employers that do not offer UNY*Care coverage to their employees;
- *employees* whose premium cost-sharing may be increased as the result of a state "minimum" benefits restructuring requirement, or personnel faced with displacement due to the revamping of the system (eg, with regional intermediaries managing claims processing operations, employees currently handling such claims functions for health insurers could be adversely impacted);
- *the US Department of Health and Human Services* which may resist state overtures relative to (a) costly expansion of Medicare coverage; (b) state-established payment levels for Medicare Part B services.

Other factions may join in resisting this proposal as the UNY*Care movement unfolds. On the other hand, we should be cognizant of the possibility of some shortsighted UNY*Care support from segments of the foregoing sources:

- health insurers that may anticipate (a) reduced administrative expenses resulting from diminished marketing and enrollment functions, and (b) reduced labor costs resulting from state delegation of claims processing operations to fiscal intermediaries;
- employers who may perceive a "minimum" benefits package featuring uniform payments as a positive element of the proposal since health insurance selectivity and administration would be simplified;
- small business entities who may welcome state subsidy of UNY*Care coverage for their employees;
- certain segments of the physician and hospital communities who may perceive UNY*Care payments as palatable—especially with regard to proposed enhancement of Medicaid payments "to parity with other payors"—despite state establishment of the uniform payment rates for the "other payors." (NOTE: UNY*Care plans to utilize quality control surveys to *prevent* physicians from enjoying the improved Medicaid fee schedule without expanding access.)

CONCLUSIONS

Despite the ambitious pronouncements and embellishments contained in the UNY*Care proposal, one could preliminarily assess the DOH initiative at this point as an expansion of the Medicaid concept, ie, *a socialized medicine program designed to eventually encompass the entire New York State resident community within a relatively brief period of time.*

Once again, the New York State Department of Health is demonstrating its lack of recognition of the medical community while it has apparently been long in the process of a health care delivery program design that is projected to have enormous and lasting impact on the state's patient and physician communities if it is implemented—a plan ostensibly designed by the DOH to essentially become "the only health insurance system in town," with the department ultimately attaining greater control of the state provider community in the process.

The January 1989 UNY*Care draft proposal (still maintained by the DOH as confidential in nature) appears to be a cautiously and amorphously worded document that could conjure many different interpretations. This may have been deliberate on the part of the DOH so that the actual intent of implementing a socialized medical program for the entire state would not be clearly evident if details of UNY*Care became prematurely known; thus forestalling an early groundswell of opposition from the obvious sources until the plan is completed and ready for public and legislative consideration.

If, in fact, this is the state "game plan," it may be realistically envisioned that the DOH primarily plans to sell its UNY*Care concept directly to the public before possibly expending token efforts to obtain any meaningful input from those providers most affected by the proposal, ie, physicians, hospitals, and health insurers. For this reason, UNY*Care may very well encounter significant opposition to its implementation from these provider sources who can only be extremely wary of an imminent DOH proposal maintained in such a confidential manner. We therefore strongly suggest that the expertise of organized medicine and other providers of health care is essential to the formulation of an initiative of this magnitude—much in the same manner that the MSSNY has been invited to participate actively in the significant deliberations on access to care of the State Council on Health Care Financing (chaired by Assemblyman Richard N. Gottfried) concerning planned provision of health care coverage for the uninsured.

In the meantime, despite MSSNY desire for further details of the program, the information contained in the confidential draft should be sufficient to arouse deep concerns among all providers of health care in this state. Furthermore, in view of the manner in which this proposal has been withheld from scrutiny by organized medicine, the physician community of New York State must stand ready to mobilize and respond in appropriate, professional, and timely fashion when the final form of the UNY*Care proposal is formally unfolded.

UNY*Care—a nice, catchy title. But the Department of Health proposal by this, or any other, name still signifies *state-controlled medicine*—thus compounding the current highly regulated health care system in New York State, with implications for the rest of the country.

A 91-year-old daughter's memoir of her physician father, Dr Vacil D. Bozovsky (1865–1933)

LOUISE BOZOVSKY DAGGETT

Few immigrants come to our shores from Bulgaria. My father, Dr Vacil D. Bozovsky, did, with the firm intention of permanently returning to his beloved land, where he would spend his days as doctor-missionary to the needy of his country. He remained, though, in Chautauqua County, New York, and never ceased to be homesick. "Homesickness is one disease that cannot be cured," he would say.

He was born in Shiroka-Luka, Bulgaria, in 1865. He was awarded, as a teenager of age 14, a special medal by the Bulgarian government for guarding its frontier against the Turks, who were forever trying to invade for territory gains. My father's brother, Anton, was a general in the Bulgarian army. After the death of King Alexander, Queen Marie invited my uncle Anton and his wife Evonka to reside with her in the palace in Sofia. They accepted. Photographs show a gorgeous structure, cold and uncomfortable to live in, I'd surmise, where they spent their final years. Queen Marie planned to visit the United States in 1913 in order to seek funds for her country. She was to stop at our home and my mother worried about what to feed her. Since my father had no funds to give her he was relieved when the trip was cancelled.

My father, newly arrived from Bulgaria, penniless, and knowing no English, took a year at the Fredonia Normal School in Chautauqua County to perfect his use of our language. At the year's end, he was asked to join the faculty there as an English teacher, but refused. In 1894, fortified with his MD degree from the University of Michigan, he settled in Ohio. He later briefly joined Dr Park Lewis of Buffalo as a partner. But eventually, to please my mother, he settled in Dunkirk, Chautauqua County, where he practiced surgery.

My father's aim and purpose in life was never personal wealth. What he earned by lecturing, mostly throughout Ohio, was turned over to the Bulgarian government for charitable uses. His sole, all encompassing aim was the betterment of life for all humanity.

In 1905 he acquired the friendship of the then US Health Commissioner, Dr Harvey Wiley. Dr Wiley succeeded in obtaining two sessions with the relevant congressional committees for my father, with the idea of putting through a reform to which he was deeply dedicated. This resulted in the Pure Drugs Act. Three years later, through the efforts of President Theodore Roosevelt, this became the Pure Food and Drugs Act.

In Dunkirk, my father organized the city's first Board of Trade. He helped start the recreational Young Men's Association, or YMA. The word "Christian" was omitted so as to bar no race or creed from membership. All of this highly demanding, energy consuming activity went right along with an enormous medical-surgical practice—in Dunkirk, Fredonia, Westfield, Silver Creek, and even

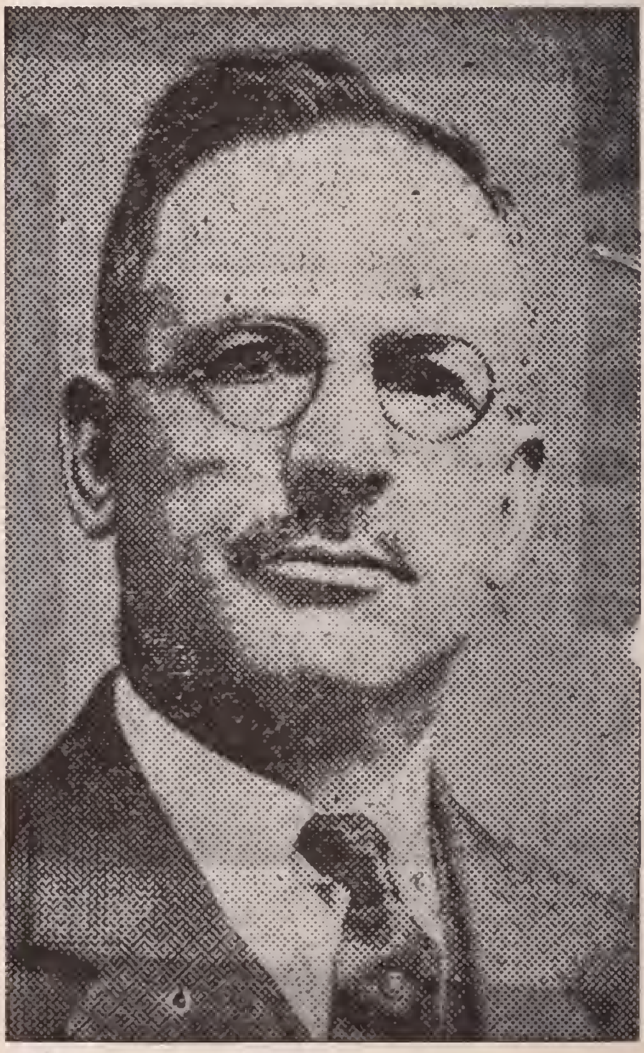

FIGURE 1. Dr Vacil Demeter Bozovsky.

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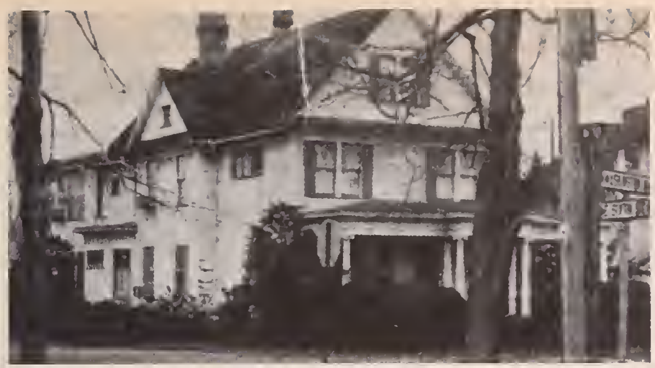

FIGURE 2. Home of Dr Bozovsky at 539 Washington Ave, Dunkirk, New York, 1911–1933.

Jamestown. He regularly gave health education talks to the high school students. I remember feeling embarrassed when I attended these talks as a student.

Two Fredonians, having been out at the Mayo Clinic in Rochester, Minnesota, came back home all agog with what Dr Will Mayo had suggested to them. Said Dr Mayo, "Why do you come here—when you have Dr Bozovsky right there in Dunkirk?" (The Mayos had been classmates of my father at college; hence their appreciation of him.)

Here is an illustration of the hardships some doctors took in stride in the early part of this century:

It is winter, sub-zero weather, in the middle of the night. To Van Buren, ten miles out from Dunkirk on Lake Erie, my father is called and, of course, responds to deliver a baby. Mac, our driver, has taken Dad out in the open sleigh, with Tom and Dan, two splendid horses. Finally, with the delivery successfully accomplished, the long, tedious ride back home is begun. It is 2 AM. Van Buren has high cliffs at Lake Erie's edge, with abrupt drops down to narrow, stony beaches, 18-foot drops, perhaps. Suddenly, as the sleigh is being yanked and roughly hauled through the deep snow, the horses come to a dead stop, and refuse to budge. Every means is used to persuade these fine, strong animals to keep on pulling. No use. So Mac and my father spend the rest of the pitch-black hours, huddled frozen in the sleigh, awaiting blessed daylight. It arrives and reveals that they are parked at the very last edge of a steep cliff. One step by Tom and Dan would have hurtled them all to their destruction! How did those horses realize that in the total blackness of the night? (He who "notices even a sparrow's fall"?)

Here is an example of the work some doctors did besides seeing patients. Sometime about 1920, a Mrs Newton of Fredonia, NY, willed money to Chautauqua County for the establishment of a tuberculosis sanitarium within its confines. At once a board of directors was organized to carry out the project as she had stipulated. The president was Dr Bozovsky, the vice-president was Dr Charles C. Goodell of Jamestown, and Judge John Lambert was a member.

All plans completed, and the site chosen, Dr Bozovsky set about devoting his time and energies to overseeing every detail of the construction of the hospital. For ten years he made the daily run to Lily Dale. He would settle for no less than Newton Memorial Hospital's being built and run on the highest possible level of efficiency. He took pains to find out who was one of the foremost authorities in the United States on tuberculosis. Then he started the challenging job of persuading Dr Walter Rathbun to come out to the country from Yale to take over as superintendent. As the hospital was completed and ready for occupancy, Dr Rathbun finally gave in, I think rather against his will. He was its very successful superintendent for many years. It was indeed a moment of triumph for my father.

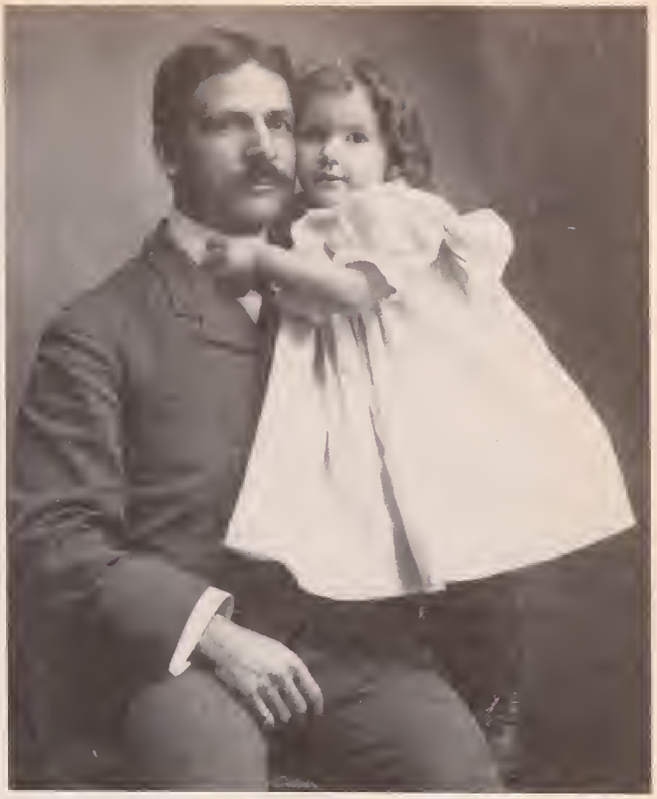

FIGURE 3. Dr Bozovsky in 1899 at the age of 34 years with his daughter Louise at the age of 2 years.

Dr Eugene Jewett, the famous orthopedic surgeon, who was a quasi-cousin of ours, told me that my father was the first physician to use animal skins for grafting. His patient was a woman who had suffered third degree burns over 80% of her body. It took him all summer, but he cured her. He first used guinea pigs; then when the hospital could procure no more of those, he had them get rabbits. The Brooks Hospital in Dunkirk had wide lawns. The patient sat out there all summer, waiting for the next skin graft. In a small city like Dunkirk, everyone knew who she was, what had happened to her, why she was there. She was a charity patient.

My dad had many special achievements in the line of his profession. His was a fascinating, crowded existence. It came to an end in June of 1933, Dr Bozovsky being then 68 years of age. Upon my remark to him earlier that age 68 is young, "Look what I have lived," he said.

After my father's death, Miss Catherine Jones, superintendent of the Dunkirk Hospital, called on us girls (my mother had died some years before) to tell us that never, in all the long time my father had operated, had he operated on a patient who did not have what he had diagnosed beforehand. This was a most unusual record for a surgeon to have in those days.

Sarcoidosis of the thyroid clinically mimicking malignancy

IRENE A. WEISS, MD; ANJALI LIMAYE, MD; VICTOR TCHERTKOFF, MD; JACOB L. BRENER, MD

Sarcoidosis is a multisystemic disease with varied manifestations, but thyroid involvement is unusual. Previously reported cases of sarcoidosis of the thyroid included patients who had either long-standing sarcoidosis or other evidence of the disease at presentation.[1,2] We report a case of an enlarging thyroid mass clinically simulating a malignancy and presenting as the sole initial manifestation of sarcoidosis.

CASE REPORT

A 62-year-old woman was referred in October 1984 for an enlarging neck mass of four months' duration, associated with dysphagia but without weight loss. In February 1983, she had been seen by the Endocrinology Service because of mild dysphagia. The physical examination at that time revealed diffuse, firm, nontender, irregular thyromegaly with predominance of the left lobe. She was euthyroid with a thyroxine (T4) of 8.9 mg/dL (normal, 5.5–11.5 mg/dL); T3 resin uptake (T3RU) of 35.5% (normal, 35–45%); and thyroid stimulating hormone (TSH) of 15.5 mIU/mL (normal, less than 7 mIU/mL). A repeat TSH level was 6.1 mIU/mL. Thyroid antibodies were not detected. The thyroid was asymmetric on 131-iodine scan. An esophagogram was normal.

The history was remarkable for Type 2 diabetes mellitus, ideopathic hypertrophic subaortic stenosis, positive purified protein derivative test in July 1984 (without clinically evident tuberculosis), and partial thyroidectomy in the remote past. No information regarding this surgery could be obtained.

Physical examination revealed diffuse thyromegaly with ill-defined borders. Within the right lobe was a tender 3 × 4-cm mass fixed to the surrounding tissues and skin. The overlying skin was erythematous, with inflammation of the previous thyroidectomy scar. There was no cervical lymphadenopathy. The liver was palpable 5 cm below the right costal margin and was smooth and nontender. The remainder of the physical examination was unremarkable. A complete blood cell count with differential, erythrocyte sedimentation rate, and routine blood chemistries were normal except for a minimally elevated alkaline phosphatase of 126 U/L (normal, up to 115 U/L), and a random blood sugar of 194 mg/dL. The T4 was 8.9 mcg/dL; T3RU, 36.9%; and TSH, 12.9 mIU/mL. The thyroid scan was similar to that of February 1983, except that a cold mass was outlined over the right lobe. The chest film was normal; however, a chest film obtained 18 months earlier showed diffuse interstitial markings.

A computed tomographic (CT) scan with contrast of the neck and chest revealed bilateral thyromegaly with scattered well-defined nodules (Fig 1). An ill-defined soft tissue mass was noted in the anterior neck with thickening of the overlying skin. Several mediastinal lymph nodes were enlarged. Ultrasound of the liver showed mildly increased echogenicity.

Due to a strong clinical suspicion of thyroid cancer, the patient was subjected to thyroidectomy. At surgery, the sternothyroid and sternohyoid muscles were adherent to the thyroid, scarred, and thickened; the thyroid was multinodular with the predominant nodule in the right lobe. There was no cervical lymphadenopathy. At pathologic examination, the thyroid was soft, reddish-brown, multinodular, weighed 30 g, and measured 2 × 3.5 × 3.5 cm. The cut surface showed multiple nodules ranging from 2 mm to 8 mm in diameter.

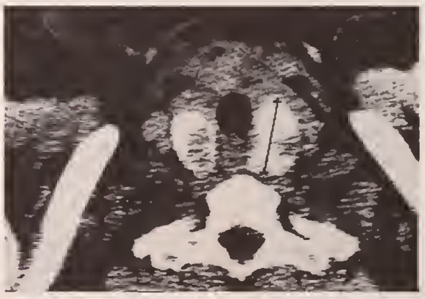

FIGURE 1. Contrast enhanced computed tomographic scan of the neck showing bilateral thyromegaly with scattered nodules, an ill-defined anterior neck soft tissue mass, and thickening of the overlying skin.

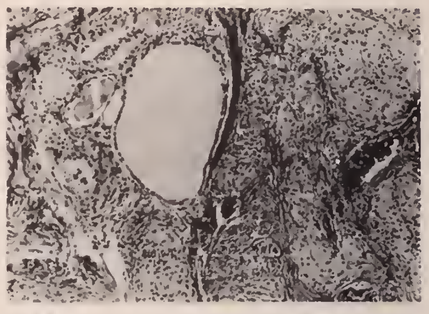

FIGURE 2. Noncaseating, cohesive epithelioid granulomata entrapping normal thyroid follicles (hematoxylin-eosin stain; original magnification × 40).

Microscopic examination revealed areas of confluent noncaseating granulomata entrapping and replacing thyroid follicles, extending into surrounding strap muscles and adipose tissue, and composed of epithelioid cells, Langhans' cells, foreign body giant cells, a few lymphocytes, and plasma cells (Figs 2, 3). A few giant cells contained refractile, needle-shaped foreign materials. Special stains and cultures were negative for mycobacterial and fungal organisms. The

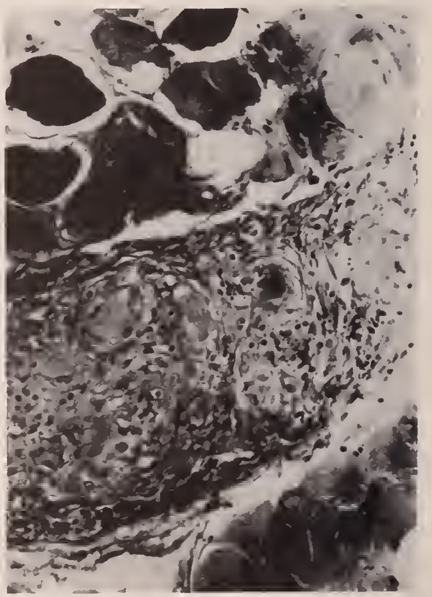

FIGURE 3. Granulomata infiltrating skeletal muscle (hematoxylin-eosin stain; original magnification × 100).

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remainder of the thyroid showed follicles of varying sizes lined by low cuboidal to columnar cells and containing varied amounts of colloid.

After surgery, the angiotensin converting enzyme level was 19.9 U/mL (normal, 11–29 U/mL). Pulmonary function tests (spirometry and lung volumes) and slit lamp ophthalmologic examination were normal. Two weeks after surgery, several erythematous macular lesions developed in both legs. Biopsy of one of these lesions revealed confluent, noncaseating epithelioid granulomata similar to those seen in the thyroid (Fig 4). Special stains and cultures for fungi and acid-fast bacilli were negative.

Three months later, similar skin lesions developed on the patient's arms at sites of insulin injections. When she changed the injection sites to her thighs, new lesions did not appear. She was last seen in June 1985 when she had an increased number of upper extremity skin lesions unrelated to insulin injections. She moved to Puerto Rico and was lost to follow-up.

DISCUSSION

The patient described here had an enlarging thyroid mass as the initial manifestation of sarcoidosis. Thyroid involvement in systemic sarcoidosis is rare. It was first described by Spencer and Warren,[3] who reported the autopsy findings of a patient with extensive sarcoidosis and microscopic thyroid involvement. Two large autopsy series of patients with sarcoidosis were reviewed by Winnacker et al.[4] In these series the frequency of thyroid involvement with sarcoidosis was 4.2% and 4.6%. Mayock et al[5] found clinical evidence of thyroid disorders in two of 145 patients with sarcoidosis. In both these patients, "thyroiditis" developed during the active phase of sarcoidosis. There was no histologic proof of sarcoid involvement of the thyroid in these cases. Hugues et al[6] reported 50 patients with intrathoracic sarcoidosis and found that 18 of them had thyroid disorders that were predominantly diffuse or nodular goiters. All patients were euthyroid except for one patient with Graves' disease. Of the four patients subjected to thyroidectomy, three showed microscopic infiltration of the thyroid by sarcoidosis. A recent report[7] described a patient who presented with a thyroid nodule that was found to be secondary to sarcoidosis. In that case the chest film was consistent with pulmonary sarcoidosis with bilateral hilar lymphadenopathy and pulmonary opacities.

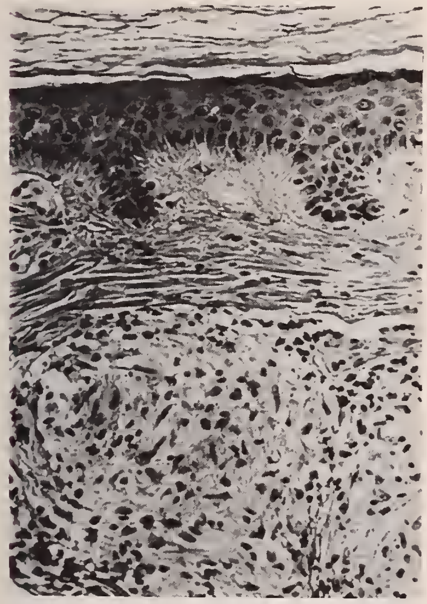
FIGURE 4. Skin with intradermal sarcoidal granulomata (hematoxylin-eosin stain; original magnification × 100).

Hyperthyroidism may be associated with sarcoidosis.[8-11] Taillandier et al[9] summarized the findings of 15 hyperthyroid patients with sarcoidosis who were subjected to thyroidectomy. In nine of these cases microscopy revealed intrathyroidal granulomata. Whether the granulomata trigger an immune response causing the hyperthyroidism or represent a coincidental finding is not known. Alternatively, patients with sarcoidosis may have histologic evidence of Hashimoto's thyroiditis. Karlish and MacGregor[11] reported five patients with sarcoidosis who had clinical evidence of Hashimoto's thyroiditis. In the three who underwent thyroid biopsy, the histology was consistent with Hashimoto's thyroiditis without mention of intrathyroidal granulomata. In a recent report,[12] a patient with sarcoidosis developed a goiter that was clinically suspicious for malignancy. On biopsy, findings were consistent with sarcoidosis of the thyroid coexisting with Hashimoto's thyroiditis. We know of only one reported case[13] in which the patient presented with mild hypothyroidism and a thyroid mass which, on biopsy, proved to be due to sarcoidosis. Therefore, hypothyroidism due to sarcoidosis is probably rare. The patient described here had mild hypothyroidism which may have been due to previous surgery or sarcoidal infiltration or both.

The lesions of sarcoidosis may be found in scars and vaccination sites.[14] This patient had biopsy-proven sarcoidal involvement of skin and, subsequently, clinically similar lesions developed at sites of insulin injections. We hypothesize that disease manifestations developed in the area of her previous thyroidectomy scar and that the granulomatous process subsequently involved the thyroid gland. Lofgren et al[15] reported 11 patients who had undergone surgery, six of them for thyroidectomy. When these patients later developed pulmonary sarcoidosis, the surgical scars became inflamed. Biopsies of scars of these patients revealed foreign body granulomata with a "marked sarcoid tissue reaction." Unfortunately, we did not obtain a biopsy of this patient's previous thyroidectomy scar.

There is a recent report[16] of sarcoidosis initially presenting as a breast mass in two patients. In both cases the patient had previously undergone biopsy of the breast in which the mass subsequently developed. In these cases and in the one presented here, the previous surgery may have helped trigger the sarcoid tissue reaction.

Tuberculosis is a rare cause of thyroid nodules.[17] In this patient, the granulomata were noncaseating. Stains for acid-fast bacilli were negative, as were cultures for *Mycobacterium* tuberculosis. A positive PPD, such as this patient manifested, may be seen in patients with sarcoidosis. In a study of 350 patients with sarcoidosis,[18] 14% had a positive PPD when they were initially seen. Therefore, tuberculous thyroiditis is extremely unlikely in this patient despite the positive PPD.

In summary, we describe a patient with a history of a partial thyroidectomy who subsequently presented with an enlarging thyroid mass which was found to be due to sarcoidosis. This case is reported because of the unusual presentation of sarcoidosis as a thyroid mass simulating malignancy.

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Pulsed Doppler-ultrasonic diagnosis of carotid body tumor

DENNIS P. TIHANSKY, MD, PHD; PAUL S. PORTER, RDMS

Carotid body tumors are uncommon and may simulate thrombosed aneurysms, enlarged lymph nodes, or cervical cysts on physical examination. In the past, radiographic alternatives for their detection included angiography, computerized axial tomography (CT), and radionuclide scintiscanning. We describe a noninvasive method utilizing ultrasonography and Doppler spectral analysis of these hypervascular masses. We believe this is the first published report on the multiplicity of spectral waveforms within such a tumor.

The study was performed with a 7.5-MHz dynamic Acuson transducer with simultaneous Doppler spectral analysis. The carotid arteries were imaged transversely and longitudinally along their lengths, while duplex spectrograms were recorded within these arteries and throughout the neck mass. The Doppler probe angulation was also varied obliquely, in order to enhance sampled waveforms.

CASE REPORT

A 50-year-old woman presented with a two-year history of an asymptomatic, slowly enlarging 6.0 × 3.0-cm mass in the left anterior cervical region below the angle of the mandible. The family history was unremarkable. However, her original medical examination revealed a bruit over this area, so that a carotid or jugular venous aneurysm was initially suspected.

Based on this early discovery, a computed tomographic (CT) scan was ordered with and without intravenous contrast injection. The latter images were taken during drip infusion after bolus injection, and showed a homogeneous, relatively nonenhancing lesion above the carotid bifurcation with possible involvement of the internal carotid artery. Consequently, a partially thrombosed aneurysm of the internal carotid artery was considered more likely than a venous abnormality or a tumor. Follow-up examination without surgical intervention was then elected.

During the patient's latest follow-up examination, a duplex scan was ordered to ascertain any hemodynamic compromise in the carotid arteries. Real-time transverse scans (Fig 1) showed the mass splaying both internal and external carotids, with greater narrowing of the former vessel. However, no significant flow disturbance or perturbation was observed. The mass was hypoechoic but not completely echo-free. There were numerous tiny irregular anechoic spaces scattered throughout the mass, thought to represent small vascular conduits or spaces because of their Doppler shift signals.

Longitudinal scanning delineated a patent internal carotid arterial lumen near its origin, but more distally the artery could not be separated from adjacent soft tissue. While this distal finding could be a partially thrombosed vessel, technical difficulties of scanning in the upper neck precluded an accurate diagnosis.

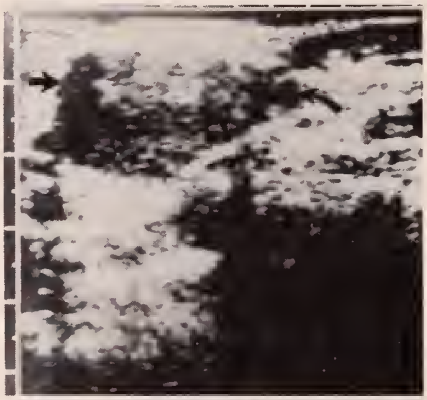
FIGURE 1. Hypoechoic mass abutting internal (straight arrow) and external (curved arrow) carotid arteries.

On the other hand, pulsed Doppler scans were more definitive and revealed important tissue characteristics. Spectral analysis identified three distinct waveforms, implying that the mass was vascular rather than a clot. The probe gate was moved randomly throughout the mass, internally and along its borders and throughout the tiny anechoic or vascular spaces.

The most frequent waveform (Fig 2) was an "arteriole spectrum," in which the systolic component had a sharp peak and fast decrescendo without flow perturbation. The dicrotic notch in these small vessels was similar to that in the carotid arteries. Despite gain adjustments, these waveforms were weak or low-powered, most likely due to the small number of erythrocytes flowing through these channels.

A more dampened flow pattern (Fig 3) was also observed. The frequency of high amplitude components correlated with the respiratory cycle. Hence, these waveforms were labelled as a "venule spectrum," since they depicted venous flow.

The third waveform (Fig 4) had a mixed pattern of both arteriolar and venule components. Its frequency followed the cardiac cycle, but systolic flow was quite dampened.

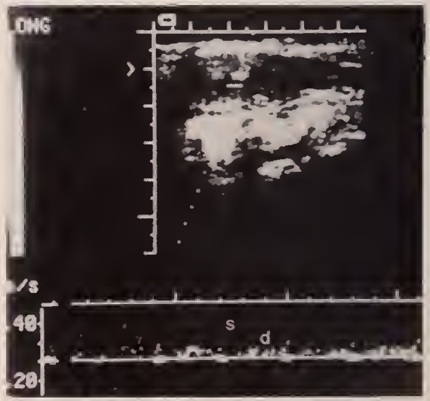

FIGURE 2. Arteriole spectrum with weak systolic (s) and diastolic (d) components.

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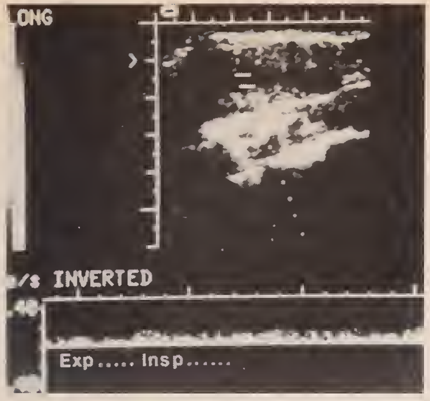

FIGURE 3. Venule spectrum with velocity increase from expiration (exp) to inspiration (insp).

Moreover, the spectrum was dense with slight pre-systolic backflow (eg, negative velocity component), implying some perturbation and increased erythrocyte pooling.

From these Doppler curves, a pre-surgical diagnosis of carotid body tumor rather than thrombosis was reported. Sonographic evidence of such a hypervascular mass above the carotid bifurcation is virtually diagnostic of this tumor.[1] As expected, this diagnosis was confirmed at surgery. At follow-up six months after surgery, the patient remained asymptomatic without tumor recurrence.

Discussion

The carotid body tumor, also known as a chemodectoma or nonchromaffin paraganglioma, is relatively rare and arises from chemoreceptive tissue at the carotid bifurcation. It grows cephalad between the external and internal carotid arteries, and later may occasionally encircle these branches or the carotid bulb. The tumor is typically slow growing and asymptomatic, although it sometimes causes dysphagia, hoarseness, or cough due to pressure on the hypoglossal or vagus nerves or the sympathetic chain. Bruits are sometimes heard over the mass, but rarely does it interfere with intracerebral blood flow.[2]

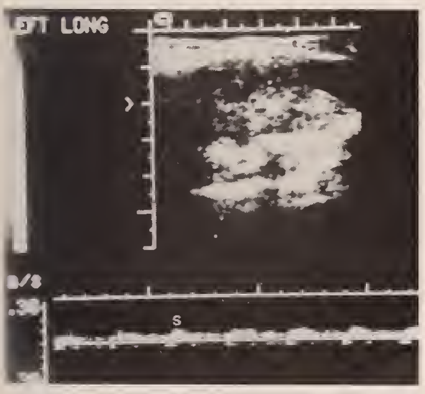

FIGURE 4. Intermediate spectrum in vacuoles or cystic spaces with low velocity during systole (s).

On histologic evaluation, carotid body tumors are highly vascular with clusters of epithelioid or supporting cells between blood vessels. Although catecholamines, including epinephrine, norepinephrine, and serotonin, are found in these cells, reports of functioning tumors inducing hypertension or tachycardia are unusual.[3] Tissue alterations such as necrosis, hemorrhage, or small vessel thrombosis are also uncommon.

The vast majority (95%) of these chemodectomas are benign, but can locally invade or recur after surgery. Among the malignant variety, metastases usually occur to adjacent neck structures such as cervical lymph nodes or the thyroid gland, although distant organs can be affected via hematogenous spread.[1] Bilateral tumors have a high heredofamilial incidence of 26%, but otherwise are observed in only 3% of cases.[4]

Prior to therapy, the hypervascularity of the tumor and its abutment of carotid arteries make open neck biopsy too risky. In addition, percutaneous aspiration biopsy is seldom helpful,[5] thus making radiologic evaluation essential for the diagnosis.

The treatment of choice is surgical excision, since the tumor, even if dormant for years, may grow insidiously and eventually cause pain or nerve palsies. Most carotid body tumors are easily treated by extirpation via subadventitial dissection, but advanced growth may require reconstruction of the carotid artery with the risk of hemiparesis.[6] In a few cases, surgery is contraindicated, wherein radiotherapy may be useful to prevent intracranial spread or to treat residual tumor, even though chemodectomas are generally radioresistant. Given any therapeutic approach, however, lifetime follow-up is recommended because of the possibility of malignancy, multicentricity, or delayed tumor recurrence, especially among patients with a positive family history.[7]

Among radiologic procedures, angiography remains the most definitive technique, short of biopsy, for the diagnosis and delineation of carotid body tumors.[2] The angiographic pattern is relatively consistent, with the vascular tumor usually fed by branches of the external carotid artery, although in some cases additional feeders may originate from the internal carotid artery or even the vertebral artery.[8] Circulation times reveal a rapid and short arterial phase (lasting under 1.5 sec), immediately followed by a more uniform tissue accumulation or parenchymatous phase (1.5–6 sec duration). Early venous (or arteriovenous) shunting is observed in almost half of these tumors,[8] while prominent tissue opacification in small "vascular channels" occurs after contrast enhancement.[9]

Ancillary techniques to angiography include CT scans, which can determine tumor extent and metastases.[9] However, because contrast enhancement is so rapid within this tumor, the scans must be performed immediately after bolus injection. The lack of rapid imaging may account for negative results in our earlier CT examination. Another technique involves technetium-99m radionuclide scintigraphy,[10] which can be effective as a screening technique to limit more invasive angiographic procedures to abnormal cases.

Only in recent years has ultrasound been used for the evaluation of cervical chemodectomas.[2] Several reports on gray-scale imaging demonstrated its utility in detecting the hypervascularity of such tumors.[11,12] In a few cases, Doppler analysis revealed single waveforms within each tumor, ranging from a prevalently venous flow to a combined arteriovenous signal with a high-grade diastolic component.[13,14] Until now, no study has demonstrated a variety of waveforms within any paraganglioma. Other neck masses, including thyroglossal cysts, cystic hygromas, hemangiomas, and some metastatic nodes, have been diagnosed by real-time sonography.[15,16] However, Doppler scanning has been limited to the analysis of high frequency signals in some thyroid carcinomas or overactive goiters.[17]

In summary, duplex ultrasonography is useful in the identification of tiny vascular flow patterns, and can also be used as a reliable screening test and diagnostic modality for carotid body tumors. Compared to CT, it is less costly and lacks radiation. It also avoids the pitfall of false-negative results from CT scans obtained after contrast infusion. In contrast to nuclear scans, sonography yields better resolution of both intra- and extra-tumoral vascularity. It can be performed prior to, or as an adjunct to, angiography, in order to screen patients who do not require surgery and thus avoid significant radiation and risks from angiography.

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Late onset post-pneumonectomy empyema

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In a patient with a history of lung cancer, the symptoms of fatigue, malaise, and weight loss are highly suggestive of recurrence. When the symptoms include an elevated alkaline phosphatase level, the diagnosis of recurrent tumor seems certain. However, a similar clinical presentation can occur in patients who develop late onset post-pneumonectomy empyema.

Empyema in the post-pneumonectomy space occurring months or years after pneumonectomy may be difficult to diagnose. We have recently seen a patient in whom empyema occurred four years after pneumonectomy. The symptoms of weight loss and abnormal laboratory tests strongly suggested recurrent malignancy. An early consideration of late onset post-pneumonectomy empyema in the patient would have saved the patient much anxiety and an extensive diagnostic work-up. We are presenting this case in order to alert clinicians to this entity.

CASE REPORT

A 60-year-old man was admitted to the Buffalo Veterans Administration Medical Center with complaints of easy fatigability, night sweats, and a 7-kg weight loss (10% of patient's body weight) over a six-month period prior to admission. Four days prior to admission he experienced the onset of diffuse pain in the right lateral aspect of his chest on coughing. One day prior to admission he noticed a swelling on the right lateral aspect of his chest.

Five years prior to this admission, the patient was found to have moderately well-differentiated squamous cell carcinoma of the right lung. The work-up at that time demonstrated a T3NOMO lesion with the tumor less than 1 cm distal to the carina. He received five months of chemotherapy followed by mediastinal radiation. Repeat bronchoscopy showed significant regression of the previously noted mass. After a negative mediastinoscopy, the patient underwent a right pneumonectomy. No tumor was identified in the resected specimen. A postoperative chest film revealed complete opacification of the right hemithorax with some shift of the mediastinum to the right.

Nine months prior to the present hospital admission the patient had a bilateral inguinal herniorrhaphy at another hospital without any complications. One month later he noted general weakness, fatigue, anorexia, and night sweats. Four months prior to the current admission, he was admitted to the Buffalo Veterans Administration Medical Center due to continuation of these symptoms, and underwent an extensive evaluation. Physical examination was remarkable only for thoracotomy scar and findings consistent with a pneumonectomy on the right side. He was afebrile throughout his hospital course. He had a mild normochromic normocytic anemia with an elevated platelet count in the range of 500,000/mm^3. His alkaline phosphatase level was 450 U/L (normal range, 30–115 U/L), but the remainder of the serum chemistries were normal. A bone scan was normal. The rest of the work-up included ear, nose, and throat evaluation, serum protein electrophoresis, liver spleen scan, thyroid and adrenal function tests, serum acid phosphatase, and computed tomography of the chest, abdomen, and head. All of these studies were negative. The intermediate strength purified protein derivative was positive. Repeated sputum samples were, however, negative for acid-fast bacillus staining and culture. The patient was discharged from the hospital without a diagnosis, only to be readmitted this time with new onset of swelling on the right side of the chest wall.

Physical examination during the current admission revealed a soft, 10 × 12-cm fluctuant mass along the right lateral chest wall below the previous thoracotomy scar. The patient was afebrile. There was no lymphadenopathy, spinal deformity, or tenderness. There was dullness and absent air entry on the right side of the chest, which was unchanged since the previous admission. The remainder of the examination was normal.

The patient's admission blood count showed a white blood cell count of 14,800/mm^3 with a moderate left shift; hemoglobin, 11.5 g/dL; hematocrit, 35.4%. The chest film showed complete opacification of the right side of the chest and was unchanged from the previous examination. The serum alkaline phosphatase level was 540 U/L. The gamma glutamyl transferase was 363 U/L (normal range, 8–43 U/L). A needle aspirate of the fluctuant thoracic mass revealed greenish yellow pus which grew microaerophilic streptococci on anaerobic cultures. The cultures for acid-fast bacilli on the aspirate specimen were negative. The patient underwent a rib resection and open drainage of his empyema. After surgery, the alkaline phosphatase level declined on serial determinations and returned to normal, and the patient returned to his original weight. On subsequent followup of three years' duration, there has been no evidence of recurrent tumors.

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DISCUSSION

Post-pneumonectomy empyema usually occurs within three months of the pneumonectomy. An empyema that first produces symptoms or signs three months after resection in a patient with a benign postoperative course is unusual but has been reported. The longest reported interval between pneumonectomy and empyema in the literature has been 33 years.[1] In most cases, the diagnosis was established either because empyema necessitatis developed or a bronchopleural or esophagopleural fistula developed revealing a new or altered air fluid level on chest radiograph.[2] Kutty et al[3] reported a case in which a Gallium 67 scan was positive in a patient who had empyema seven years after pneumonectomy and suggested that such a scan might be a valuable diagnostic tool in a patient in whom there is a suspicion of empyema.

There have been many questions about the fate of the pleural space following pneumonectomy. Suarez et al[4] studied autopsy material in 37 patients who had undergone pneumonectomy during their lifetime and found that in 27 of these patients there was a fluid-filled space regardless of the time interval between the pneumonectomy and the autopsy. The amount and character of the fluid in such cases was not related to the interval between the surgery and death. It has been suggested that this fluid may become the nidus for hematogenously seeded bacteria. Holden and Woller[5] described a patient who had a bacteroides empyema of the post-pneumonectomy space which occurred three weeks after a perforated appendix and bacteroides peritonitis, presumably due to hematogenous spread, as there was no evidence of a subphrenic abscess on clinical or postmortem examination. It is possible that the microaerophilic streptococci in the pleural space in our patient might have been seeded from the blood stream either during the barium enema performed prior to his herniorrhaphy or at the time of intubation for anesthesia at the time of the herniorrhaphy. However, definitive proof for these possibilities is lacking.

The microorganisms responsible for late onset post-pneumonectomy empyema are varied and include both aerobic and anaerobic organisms. It is postulated that in addition to hematogenous seeding, the organisms can also be implanted at the time of original surgery and can smoulder in the bronchial stump or a chest wall suture until empyema takes place. For instance, *Aspergillus* empyema resulting from the nidus of exposed silk bronchial suture material has been reported.[6]

The most interesting laboratory finding in this patient, and the one that led to the suspicion of recurrent malignancy, was the elevated alkaline phosphatase level. The serum enzyme increased with the development of empyema and declined after drainage. The simultaneous elevation of gamma glutamyl transferase in this patient led us to believe that the elevated alkaline phosphatase activity was probably of hepatic origin and related to his chest infection. Fang et al[7] have described similar occurrences of marked elevation in serum alkaline phosphatase activity with minimal or no elevation in serum bilirubin due to intrahepatic cholestasis as a result of systemic infection.

It should be emphasized that a strong index of suspicion is needed to diagnose late onset post-pneumonectomy empyema, and that whenever the possibility is entertained, diagnostic needle aspiration should be carried out.

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Scrotal ischemia after intravenous vasopressin therapy for hemorrhagic esophageal varices

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Hemorrhagic esophageal varices are associated with a high mortality. The control of hemorrhage may be achieved by vasopressin infusion, and a potent vasoconstrictor can reduce splenic blood flow and portal venous pressure.[1] However, several undesirable complications, in addition to cardiac damage, have been reported during such infusions.[2–5] We report two rare cases of scrotal ischemia following intravenous vasopressin infusion.

CASE REPORT

Case 1. A 74-year-old man was admitted with hematemesis. He had a history of biopsy-proven cirrhosis with many episodes of ascites and hepatic encephalopathy in the preceding five years. On admission, he was jaundiced and somnolent. Spider angiomata, ascites, pitting edema, and asterixis were present; the testes and scrotum were not unusual. Upper gastrointestinal endoscopy disclosed four esophageal varices with cherry-red spots indicating recent hemorrhage. Treatment included blood transfusion and intravenous vasopressin infusion at a rate of 0.66 U/min for the first hour and 0.4 U/min thereafter. Bleeding ceased 24 hours later, and the medication was then discontinued. Bleeding recurred 12 hours later,

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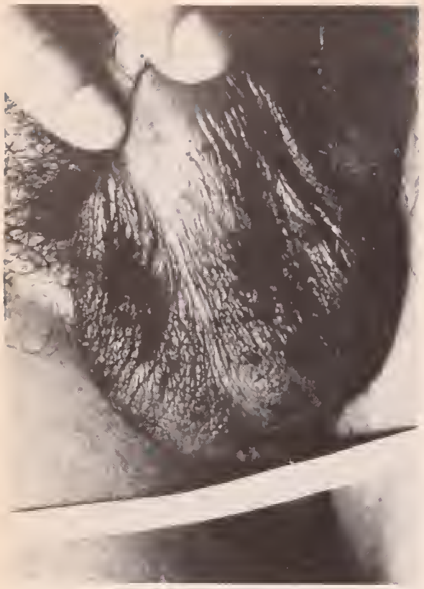

FIGURE 1. The scrotum of the patient in case 2 showing several patches of ischemic discoloration; photograph was taken ten hours after the occurrence of ischemia.

and the patient received a continuous infusion again with doses ranging between 0.2 and 0.4 U/min. On the fourth day, he became comatose, though vital signs were stable. The bleeding continued at this time, and the entire scrotum was noted to be black and purple in color. Because the significance of this discoloration was not recognized, the infusion was not discontinued. On the sixth day, multiple erosions developed in this ischemic scrotum. A comatose state persisted despite vigorous management of hepatic encephalopathy. The patient died on the eighth day.

Case 2. A 63-year-old man was admitted with hematemesis. One month prior to the admission, he was found to have cirrhosis and hepatoma. On admission, he was alert with positive findings including icteric sclera, anemic conjunctiva, hepatomegaly, and ascites. The scrotum and its contents were not remarkable. Upper gastrointestinal endoscopy disclosed four esophageal varices and oozing blood. Hemorrhage did not stop in spite of the use of a Sengstaken-Blakemore tube for 24 hours. Vasopressin was then infused intravenously at a rate 0.6 U/min. Six hours later, the patient complained of severe abdominal pain, particularly in the umbilical area, with radiation to the scrotum. On physical examination, there was local tenderness of the abdomen without other significant signs. The dose was tapered to 0.3 U/min, and sharp scrotal pain persisted despite subsidence of the abdominal pain. Twenty hours after the infusion began, several patches of black-purple mottling of the skin of the scrotum developed. Their sizes varied from 0.5 cm to 2 cm (Fig 1). Vasopressin was discontinued immediately. Bleeding did not return; there was no further painful sensation, ischemia, or erosion of the scrotum. All discoloration disappeared one week later without any specific treatment.

DISCUSSION

Although the ultimate improvement in mortality for lethal hemorrhagic esophageal varices by vasopressin infusion is controversial, its initial efficacy for the cessation of hemorrhage can reach 50%.[1] Moreover, intra-arterial therapy has not been proven superior to the intravenous route.[6] Therefore, in order to stop hemorrhage, intravenous vasopressin infusion is commonly prescribed. As a vasoconstrictor, vasopressin's cardiac side effects are well known and occur frequently.[1] They were absent in these two cases. Several other unusual complications such as nipple necrosis,[2] tongue necrosis,[3] cutaneous necrosis,[4,7] ischemic colitis,[8] and even lethal portal vein and superior mesenteric vein thrombosis[5] have been reported. These conditions have developed with doses ranging from 0.2 U/min to 1.0 U/min, through either intravenous or intra-arterial routes. The elapsed time of noticed complications ranges between one hour and five days following the start of infusion.

Scrotal ischemia or necrosis associated with vasopressin infusion is very rare. We failed to recognize this significant complication in case 1. By reviewing the first published report of vasopressin-induced scrotal damage, however, we were alerted to this possibility.[9] Half a year later, the patient in case 2 developed the same side effect, but recovered quickly after prompt discontinuation of vasopressin therapy.

This kind of complication has not been reported in any language to our knowledge in the past 20 years, except for the two cases in the report by Gogel et al.[9] Does continuous infusion of vasopressin for the treatment of hemorrhagic esophageal varices really cause scrotal ischemia? A comparison of the four cases of vasopressin-induced scrotal ischemia, including two cases of Gogel et al[9] and two of ours, revealed that the ischemic lesions developed in patients without any underlying vascular factors such as diabetes or shock that would increase the patients' vulnerability to ischemia. None of the patients had any history of vascular occlusive disease. Despite the differences in the regimen, dosage, and duration of the infusion of vasopressin in these cases, all the lesions were characterized by black and purple mottling or discoloration of the entire skin of the scrotum, with presence of erosions or ulcerations in the more severe cases. Edema of the scrotum, although reported in case 1 of the report by Gogel et al,[9] seemed not an absolute sign of ischemic changes of the scrotum, since the other three cases were free of edema.

The above comparison indirectly suggests the possibility that the scrotal ischemia is due to the infusion of vasopressin. It is our opinion that this side effect may not be rare, since many victims of hemorrhagic esophageal varices experience severe illness and are kept covered with blankets. If any cutaneous or scrotal ischemia related to the vasopressin infusion develops, it would easily be missed, particularly in non-reporting and/or unconscious subjects. However, sharp pain over the scrotum may be an early warning sign prior to the occurrence of ischemia, as was manifested by the patient in case 2 in this report. If this sharp pain occurs, the prompt discontinuation of infused vasopressin may be considered.

Why vasopressin has selective ischemic effects on organs of some individuals is unknown. However, we believe that the ischemic effect may not be related to the dosage of infused vasopressin, because many victims of hemorrhagic esophageal varices never develop either scrotal or cutaneous damage even with high dosages.[1] In view of the possibility of these undesirable complications, any patients with hemorrhagic esophageal varices receiving vasopressin infusion should be monitored for vital signs, undergo electrocardiographic scanning, and be inspected over the entire body for cutaneous complications.

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LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Confidential Tay-Sachs carrier screening

TO THE EDITOR: Shapiro and Shapiro[1] point out that Tay-Sachs carrier detection testing prior to pregnancy is more desirable than intra-pregnancy testing "because it is technically easier, less expensive, and associated with less anxiety and less urgency. However, more than 80% of individuals referred for testing were pregnant women and/or their partners."

A much better time to screen for the Tay-Sachs carrier state is prior to marriage. Yet, how does one eliminate the psychic burden on young men and women discovered to be heterozygotes?[2] What are the psychological problems created by the information that one is a carrier for a fatal genetic disease? Should a known carrier refuse to marry a mate who has not been tested? Should two carriers break up an engagement or a marriage if they learn they are both carriers as a result of a screening program? Should a young person inquire about the Tay-Sachs status of a member of the opposite sex prior to meeting that individual on a social level? When should a person who knows he or she is a carrier tell this fact to an intended spouse? Should one sacrifice primary prevention of Tay-Sachs disease by mate selection to avoid psychosocial consequences? Is this method of disease prevention an attractive aspect of genetic screening for recessives?

One must remember that 29 of 30 Jewish people tested for the carrier state are found to be free of the Tay-Sachs gene.[3] It is certainly desirable for these 29 of each 30 tested to have peace of mind. Is the anxiety of the 30th person on learning that he or she is a carrier sufficiently great to warrant not testing at all? Obviously not! However, one cannot minimize the possible psychosocial trauma to such an individual.[4,5]

The social stigma of being a carrier of the Tay-Sachs gene is not fully appreciated. Misinformed and/or uninformed people may look at carriers in the same manner as patients with epilepsy and leprosy were looked at half a century ago, ie, as individuals afflicted with a "taboo" disease, to be shunned and ostracized from normal social contact. Discrimination against carriers of Tay-Sachs disease may also occur in a variety of areas, if the experience of sickle cell screening is repeated. Individuals found to have sickle cell trait were dismissed from their jobs.[6] Several life insurance companies charged higher premiums for individuals with sickle cell trait or refused to insure them at all.[7] Several airlines rejected flight attendants from employment in that capacity if they had sickle cell trait. The United States Air Force, until recently, did not train black recruits with sickle cell trait to become pilots.[8] Job seeking was thus made more difficult for carriers of this hemoglobinopathy. Is this fate also to be suffered by people who, on screening, are found to be carriers of the Tay-Sachs gene? Total confidentiality in screening might avoid such problems and should be an essential part of all such programs.

Such total confidentiality and anonymity is provided by the Dor Yeshorim Tay-Sachs Screening Program. The cornerstone of the program is to prevent the birth of homozygous Tay-Sachs babies by testing boys and girls of marriageable age in total anonymity, enabling all those tested to be guaranteed that marriage with any prospective mate who was also tested would produce offspring free of this dreaded disease.

To be tested, one calls the Dor Yeshorim office for an appointment for a blood test at one of a number of cooperating laboratories. No names are asked, nor recorded during the entire process. The clients are given a card with a number which is the only form of identification used. No test results are divulged at this time.

When a marriage match is suggested, the clients or their parents ask for the test identification number of the other party just as naturally as they would ask for details about education, income, etc. They then call a special telephone number mentioning only the identification numbers of the prospective mates. If neither one, or if only one, is a carrier of the Tay-Sachs gene, the caller is told that there is no reason why the proposed match should not go forward. If, however, both prospective mates are diagnosed as carriers, the caller is advised that the match is not appropriate and that another match should be sought to avoid tragedy.

The Dor Yeshorim program has tested many thousands of Jewish men and women since its inception in 1984 and has prevented several dozen marriages with potentially catastrophic outcomes. The program is provided as a public service and should be made widely known not only to obstetrician/gynecologists but to all physicians in New York State and across the country. For further information, write or call Dor Yeshorim, 160 Wilson St, 1-B, Brooklyn, NY 11211; hotline: (718) 384-6060.

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Kashin-Beck disease

TO THE EDITOR: I read with interest the June 1989 issue of the *Journal*, especially the articles on tickborne infections and that on Kashin-Beck disease.[1-3] The latter disease is reported in the article to be an osteoarticular disorder that incapacitates some two million people in a mainly rural, mountainous, forested geographical area of northern China including Manchuria, adjacent Siberia, and northern Korea. The etiology of this disease, according to the article, is an "enigma" and is thought to be of environmental origin. Various possible causes mentioned in the article included impure water, poorly ventilated housing, lack of sunlight, deficiency states, and moldy grain (mycotoxins).

Having read this article on Kashin-Beck disease in juxtaposition to those concerning Lyme disease, it seems quite reasonable to me that perhaps Kashin-Beck disease is also a spirochetal-caused illness similar to Lyme disease. The endemic geographical area would almost certainly harbor ticks. Arthritis is a late effect of Lyme disease and, thus, could also conceivably be a late effect of a prior or continuing infection in Kashin-Beck disease. This seems to be a plausible hypothesis which could be tested by those in the geographical area involved.

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TO THE EDITOR: I read with great interest the excellent review article on the history of Kaschin-Beck disease by Sokoloff[1] in the June 1989 issue of the *Journal*. It was the Chinese who explored the causal relation between selenium deficiency and the pathogenesis of Keshan disease and Kaschin-Beck disease.[2] Since Dr Sokoloff is a pathologist himself, I thought he might like to add another reference on the pathologic study of metacarpo-interphalangeal joints in Kaschin-Beck disease, the report by Chu and Tsui,[3] to his otherwise very extensive list of references.

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In reply. I thank Drs Sobin and Cheng for their letters.

There is no clinical or anatomical evidence for an inflammatory process in Kashin-Beck disease. This is in contrast to Lyme disease, in which chronic synovitis has been clearly documented.[1]

The paper by Chu and Tsui has been cited by me in another review that deals more directly with the pathology of Kashin-Beck disease.[2]

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Medical malpractice

TO THE EDITOR: The Medical Society of the State of New York (MSSNY) has been a cohesive, stabilizing force for the physicians of New York State. The Medical Society through its membership has enhanced quality care for patients and improved medical education. It is an honor and a privilege to belong to it.

To continue its preeminence in the field of socioeconomic medicine and in the political arena into the next generation, the MSSNY must reexamine and reevaluate the following critical problems.

The medical malpractice crisis that began in 1974 has not improved but has escalated. From the unavailability of malpractice insurance in the early 1970s to the tort reforms of the 1980s, the number of medical liability cases has increased, as has the cost of malpractice insurance for physicians.

Attempts to reduce medical malpractice through tort reforms, arbitration, no fault insurance, contracts, and mediation have not succeeded. The no fault insurance system has not worked in either New Zealand or Sweden.[1] Malpractice premiums keep going up every year. In 1988, the rate increase in premiums was 5%; however, there may be yearly surcharges of 8% beginning in 1989, to be followed by 25% surcharges in 1991 if deemed necessary by the Superintendent of Insurance.

The malpractice system today works for the benefit of the plaintiffs' lawyers and defense lawyers. (Only 27% of every dollar goes to the plaintiff.[2])

Some critics have said that improving the skills, improving the quality of care, more consultations, more supervision by professional review organizations, better communication between physicians and patients, and even working in health maintenance organizations[3] will dramatically decrease malpractice claims. However, none of these techniques have diminished the number of malpractice claims, and premiums keep escalating.

In 1985 the Urban Institute sponsored a symposium on the malpractice crisis. Yet no clear-cut answers were offered to ameliorate the crisis for physicians. Does the present malpractice system serve its goal of injury prevention, compensation, or retribution? In my view it only serves the financial interests of lawyers. There needs to be a change in our thinking. The negligence system needs to be replaced with another system. Or is this malpractice crisis an insoluble problem?

Since the malpractice crisis is a national problem, perhaps it should be resolved at the federal level, rather than by individual states. Perhaps the malpractice premiums should be broad-based, with the federal and state government, the health care industry, the pharmaceutical industry, and physicians proportionally funding the premiums. This would be equitable to all groups.

The New York Medical Political Action Committee (NYMPAC) needs to be strengthened as a political and lobbying organization. For the millions of dollars that NYMPAC has spent, it has not achieved its goals with state legislators or with the governor. Yet, the chiropractors have spent far fewer dollars, and every year, through lobbying efforts in the state legislature, have had at least one bill passed favorable to them.

When I was secretary-treasurer of the Empire Medical Political Action Committee (EMPAC) some years back, I urged and recommended that the money spent in political lobbying could be well spent by encouraging politically active physicians in various parts of the state to run for office. The expenditure of these political monies should be strongly encouraged. Successful candidates would have a needed voice in the state legislature, especially with regard to the malpractice and health laws.

The bulk of membership of the MSSNY comes from fee for service practitioners. There is room for all types of practice in the state but MSSNY should actively encourage private practice and support it.

MSSNY has been in the forefront of discussions with third party payers concerning medical problems associated with the Omnibus Budget Reconciliation Act of 1986 (PL 99-509). MSSNY and the county medical societies are available to help physicians with Medicare problems. However, many physicians are not aware of these services. MSSNY and the county medical societies should periodically advise physicians of these available services, especially with respect to PL 99-509, which establishes limits on charges to Medicare beneficiaries by nonparticipating physicians. Under Section 933-2(c) (PL 99-509), nonparticipating physicians who do not accept assignment must refund to beneficiaries any amount collected for services which are determined medically unnecessary by third party payers. Physicians are confused, intimidated, and overwhelmed by the intricacies of Public Law 99-509.

Physicians need help. They need the help of the MSSNY to advise them and possibly represent them with their problems in dealing with third party payers. Physicians would then feel that their yearly dues have been well spent, and that the medical society has an interest in their medical practices and their self-preservation.

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MALPRACTICE SUITS

Three members of the Association have had the misfortune in being attacked by the blackmailing litigant, which in two of the cases immediately followed attempts to compel payment of bills.

It is with great interest that we are able to note that in every case the doctor has had the courage of his conviction and will compel these libelers to pay in the end. In one case the attorney for the plaintiff was called upon to pay $20 costs of a motion and he has not made a move since.

The counsel for the Association has been somewhat hampered by members coming after having other counsel in the case. There can be but one "attorney of record" and he is apt to demur to Mr. Lewis coming into the case after the case is well started, and our counsel cannot take part unless asked to do so. It is far better if the member go direct to the president of the Association and thereby save himself the expense of an attorney.

It will take a little time for the lay public to learn that members of the New York State Association are poor subjects to attempt to frighten, as their legal defense is furnished gratis, but as soon as they learn it through the publicity given to a suit or two, the blackmailing variety of malpractice suits will cease. This class constitutes 97 percent of all of such suits brought.

It seems almost incredible that after all has been said and done, many of the members of the Association are not aware of this medical defense. If it were thoroughly known the membership would double within the year.

EDITORIAL
(*NY State J Med* 1903; 3:78)

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Alcohol abuse: Opportunities for prevention

Alcohol abuse prevention efforts are often categorized as neo-prohibitionist and thus perceived to reflect a futile moralistic attempt to regulate personal human behavior. However, prevention has been shown to be effective. The increase in the legal purchase age, for example, from 18 to 19 and subsequently to 21, has resulted in a reported 50% decrease in alcohol purchases by 16–20-year-olds, as well as a 33% reduction in drinking to intoxication and a 21% decrease in self-reported driving after drinking in that age group. Groups such as Mothers Against Drunk Driving (MADD), Students Against Driving Drunk (SADD) and Remove Intoxicated Drivers (RID) have focused attention on the consequences of alcohol abuse on the highways and have been effective in raising awareness of control strategies that exist within the criminal justice system.

The New York State Department of Health, in conjunction with the State Division of Alcoholism and Alcohol Abuse, is planning a strategy to intensify prevention efforts throughout the state. Plans to develop cooperative initiatives with professional organizations and local health units are part of the overall approach. One element of the plan calls for the department to more fully take advantage of interactions with clients it currently serves to assess their alcohol abuse awareness and behavior and to provide personal information or referral as appropriate. The following are potential complements to this approach which may be undertaken by health care providers in private, institutional or public settings.

Opportunities within the Patient Management Setting*

Routine Screening—Patient Interview

a. Ask questions about the patient's drinking practices, ie, average number of drinks per day; frequency of "binge" drinking. Avoid nondescriptive characterization of drinking behavior such as "social drinking."

b. Ask questions about behavioral and social consequences of drinking (which are often the precursors of clinical evidence). For example, ask if the patient has lost any work or school time as a result of alcohol; or has had accidents or injuries following alcohol consumption; or has experienced problems with friends or family that are related to alcohol use.

c. Include the four *CAGE* questions. Have you ever:

1. Thought about Cutting down on your drinking?
2. Felt Annoyed when others criticize your drinking?
3. Felt Guilty about drinking?
4. Used alcohol as an Eye opener?

A positive response to two or more of these questions is suggestive of alcohol problems and merits further discussion with the patient.

d. Record drinking history and consequence information in the medical record to serve as a baseline to evaluate future consumption and behavior.

Intervention Strategies

a. Provide basic information about the drug alcohol and its ability to impair judgment and motor skills, its association with adverse behavioral and social consequences and its relationship to injuries and chronic disease. Cite relevant statistical information.

b. Use a number of drinks/body weight chart to depict the typical dosage necessary for impairment and intoxication.

c. Use medical authority in discussing the health implications of alcohol abuse. Pregnant women and women planning pregnancy should be advised to avoid alcohol. Those whose consumption behavior is indicative of dependence should be advised that their condition represents an illness deserving of appropriate professional attention.

d. Provide referral to community resources for alcoholism and alcohol abuse prevention and treatment. Make the patient, and his/her family as appropriate, aware of the types of available resources. Whenever possible, do not just give an agency name and phone number. Have your office set up a specific appointment time.

e. Consult colleagues, local hospitals, alcohol treatment specialists or the State Division of Alcoholism and Alcohol Abuse about the types and locations of services available in your area.

f. Follow-up on referrals both with the treatment provider and with the patient to monitor status. Provide reinforcement as necessary.

This three-part series on alcohol abuse (the first and second parts to the series appeared in the March and April issues of *Epidemiology Notes*) has been adapted from a Health Department report, *Alcohol and Public Health: Implications for New York State.* To receive a copy of the full report, contact the New York State Department of Health, Division of Epidemiology, Corning Tower, Room 503, Empire State Plaza, Albany, NY 12237, (518) 474-1055. Comments and suggestions on alcohol abuse prevention are especially welcome.

Other background resources include:

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*Many of these recommendations are excerpted or adapted from the federal Office of Substance Abuse Prevention publication, *The Busy Physician's Five-Minute Guide to the Management of Alcohol Problems.*

1) Moore MH, Gerstein DR, eds.: *Alcohol and Public Policy: Beyond the Shadow of Prohibition.* Washington, DC, National Academy Press, 1981.

2) Olson S and Gerstein DR: *Alcohol in America, Taking Action to Prevent Abuse.* Washington, DC, National Academy Press, 1985.

3) Public Health Service: *Alcohol and Health. Sixth Special Report to the United States Congress,* DHHS Publication No. (ADM) 87-1519, Washington, DC, Government Printing Office, 1987.

4) *Prevention File, Community Responses to Alcohol Problems,* University of California, San Diego, Extension Program on Alcohol Issues (quarterly publication).

AIDS in New York State: An update

AIDS has exacted a heavy toll of illness, suffering and death in New York State. Through December 1988, more than 20,000 residents have been stricken with AIDS and over 11,000 have died prematurely as a result. In New York City, AIDS is now the leading cause of death for males ages 30–59 and for females ages 1–9 and 30–39.

New York State continues to have the highest number of reported cases, comprising almost 25% of the United States total. Compared with national statistics, New York State has a higher proportion of cases among intravenous drug users (IVDUs), minorities, women and children. The annual incidence of AIDS in the state has risen from less than 500 in 1982 to nearly 7,000 new cases in 1988.

Geographic Distribution

Figure 1 shows the cumulative incidence of AIDS by county of residence. Of all AIDS cases reported in the state (excluding cases diagnosed among state prison inmates), 85% have been residents of New York City and 11% residents of other parts of the state. Within New York City, the cumulative incidence rates per 100,000 range from 578.1 in Manhattan to 78.8 on Staten Island. The overall case rate for New York City is 241.4 per 100,000. In contrast, the rest of the state experienced a cumulative incidence rate of 20.5, with a range of 0 to 51.0 per 100,000 among counties. AIDS cases outside New York City have clustered in the downstate area and in major metropolitan areas.

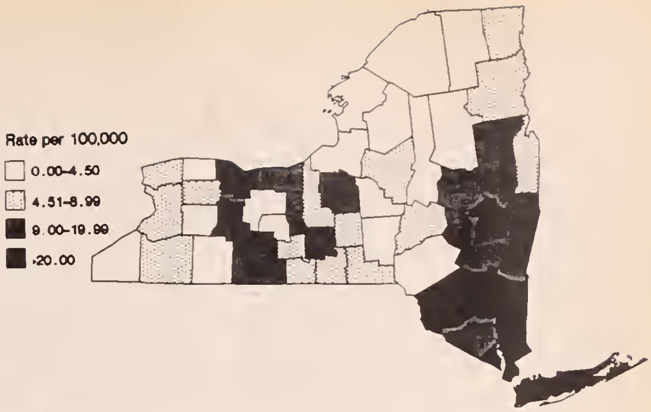

FIGURE 1. Cumulative incidence of AIDS by county of residence at diagnosis, New York State, December 31, 1988 (excludes cases among New York State prison inmates). Rates are based on 1985 NYS Department of Commerce Official Population Projections.

Risk Factors

The majority of AIDS cases have occurred among homosexual/bisexual males (9,666)(Table I), however, the percent of total cases among this population has declined from 60% prior to 1986 to 47.2%. A slowing of the rate of increase of new cases has been observed among homosexual/bisexual males which may be associated with the adoption of risk reduction practices within this group. White males accounted for 63% of the homosexual/bisexual cases, with 19% among blacks and 16% among Hispanic men. The highest percentage of cases has occurred in men 30–50 years old who live in Manhattan.

AIDS among IVDUs in the state has consistently represented a higher proportion of cases than the national average. Thirty-six percent of the total cases were IVDUs compared to less than 20% nationally. Surveillance data indicate a dramatic acceleration in the number of AIDS cases among IVDUs. In 1988, the number of AIDS cases diagnosed among IVDUs (1,928) exceeded the number of reported homosexual/bisexual cases (1,670) for the first time. The emergence of intravenous drug use as the predominant risk factor has major implications for the course of the epidemic and for potential spread to heterosexual partners and offspring of drug users.

Although the annual number of cases attributed to heterosexual contact has increased steadily, the number of cases that fall into this category is less than 5% of the total AIDS cases. A total of 623 heterosexually transmitted cases were reported through December 1988, 596 (95%) of whom were female sex partners of people at risk for AIDS. The most common risk group of the sexual partners was intravenous drug use accounting for 500 cases (84%); bisexual males accounted for 55 cases (9%). There were 380 individuals with AIDS for whom no risk category has been established other than that they were born in countries where heterosexual HIV transmission is believed to be common.

The percentage of cases attributed to transmission by blood transfusions or the use of blood products has remained low. A total of 218 cases (1.1%) have been associated with blood transfusion, and 52 cases (0.3%) with the use of anti-hemophilia factor concentrates and other blood products.

Age

The diagnosis of AIDS has occurred primarily in young and middle-aged adults (Figure 2). Approximately 48% of all AIDS cases have occurred among people ages 30–39, 22% among ages 40–49 and 18% among ages 20–29. Over 77% of new cases diagnosed in 1988 were in those ages 25–44. Since AIDS typically strikes in the prime of life, it also affects women primarily during their reproductive years; 83% of women with AIDS have been between the ages of 20 and 45.

Race/Ethnicity

Within New York State, blacks and Hispanics have borne a disproportionate share of the AIDS burden. Blacks represented 33% (6,776 cases) of all AIDS cases through December 1988, even though only 13% of the total population is black. Approximately 26% (5,312 cases) were identified as Hispanic, while only 10% of the population is Hispanic. Whites comprise 40% (8,178 cases) of

TABLE I. AIDS Cases by Race/Ethnicity and Risk Group, New York State, December 31, 1988

	White		Black		Hispanic		Other		Missing/ Unknown		Total	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Homosexual/bisexual males	6,097	63.1	1,864	19.3	1,571	16.3	86	0.9	48	0.5	9,666	100.0
IV drug users	1,223	16.5	3,328	45.0	2,803	38.0	10	0.1	30	0.4	7,394	100.0
Homosexual/bisexual IVDU males	331	35.7	330	35.6	260	28.0	3	0.3	3	0.3	927	100.0
Hemophiliac	41	78.8	5	9.6	6	11.5	—	—	—	—	52	100.0
Heterosexual contacts	114	18.3	250	40.1	254	40.8	3	0.5	2	0.3	623	100.0
Foreign born-NIR*	—	—	379	99.7	0	—	1	0.3	—	—	380	100.0
Transfusion	138	63.3	51	23.4	26	12.0	3	1.4	—	—	218	100.0
Undetermined	191	24.1	332	41.8	251	31.6	11	1.4	9	1.1	794	100.0
Pediatric	43	10.2	237	56.2	141	33.4	0	0.2	1	0.2	422	100.0
Total	8,178	39.9	6,776	33.1	5,312	25.9	117	0.6	93	0.5	20,476	100.0

* Individuals born in countries where the major mode of transmission is believed to be through heterosexual contact and for whom there is no identified risk otherwise.

the AIDS cases and 75% of the state population. In 1988, for the first time during the AIDS epidemic, the number of new cases diagnosed among blacks exceeded new cases among whites.

As AIDS has increasingly affected IVDUs as well as their heterosexual contacts and offspring, there has been a corresponding increase in the number and proportion of black and Hispanic cases. Blacks and Hispanics comprise 59% of all AIDS cases attributed to IV drug use. More than 80% of all females with AIDS are black or Hispanic, as are 90% of all pediatric AIDS cases.

Pediatric Cases

A total of 422 pediatric AIDS cases (<13 years) have been reported through December 1988 (Table II); 375 (89%) were infected perinatally. Only 18 (4%) were infected through contaminated blood products. Among all children with AIDS, 378 (89%) had onset of illness by age four. Male and female infants are affected nearly equally. However, there is a wide discrepancy by race. Only 43 (10%) of pediatric cases have occurred among whites. Blacks account for 237 (56%) of the pediatric cases and Hispanics account for 141 (33%) of the cases. This racial pattern reflects the increasing number of AIDS cases occurring among black and Hispanic women, especially IVDUs.

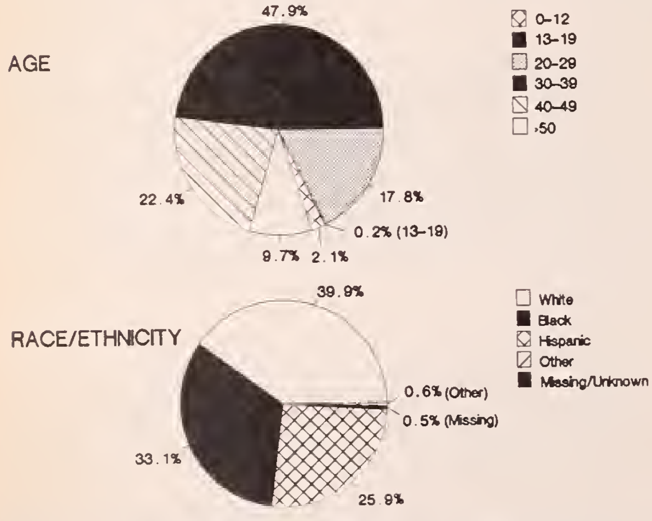

FIGURE 2. AIDS cases by age and race, New York State, December 31, 1988.

State Prison Populations

More than 40,000 individuals are housed in the New York State prison system; blacks, Hispanics, residents of New York City and those with a history of drug use are overrepresented. Through December 1988, 838 inmates have been diagnosed with AIDS; 791 (95%) of these cases have drug use identified as the risk factor. More than 400 state inmates have died from AIDS which now accounts for over 70% of inmate deaths.

Of all prisoners diagnosed with AIDS, 396 (47%) are Hispanic, 318 (38%) black and 116 (14%) white. Ninety-six percent of all inmates diagnosed with AIDS are male. The annual incidence of AIDS among prisoners has reflected the increasing incidence among IVDUs in the general population. The number of cases has increased each year since 1981 with an incidence greater than 400 per 100,000 inmates per year over the last three years.

Associated Diagnoses

Pneumocystis carinii pneumonia (PCP) is the most

TABLE II. Pediatric Cases by Race/Ethnicity, Sex, and Risk Factor, New York State, December 31, 1988

	Number	Percent
Race/Ethnicity		
Black	237	56.2
Hispanic	141	33.4
White	43	10.2
Unknown	1	0.2
Total	422	100.0
Sex		
Male	216	48.8
Female	206	51.2
Total	422	100.0
Risk Factor		
Hemophilia/Coagulation disorder	3	0.7
Maternal transmission	375	88.8
Transfusions	15	3.6
Undetermined/other	29	6.9
Total	422	100.0

common HIV-related diagnosis, affecting more than 62% of all patients. Kaposi's sarcoma (KS), a rare form of cancer, has been observed in 26.6% of homosexual/bisexual males with AIDS. However, in IVDUs, KS was found in only 2.75% of these cases. The prevalence of KS has declined in homosexual/bisexual cases from 50% in 1982 to less than 15% of those diagnosed in 1987 and 1988. Opportunistic infections that attack IVDU AIDS patients about twice as frequently as homosexual/bisexual cases include Candidiasis esophageal and cryptococcosis. Tuberculosis and syphilis (including congenital syphilis) have increased significantly in New York City during the last few years. Some evidence suggests that HIV infection can activate a pre-existing latent tuberculosis infection. The increase in syphilis has not been directly linked with HIV infection, but evidence suggests that individuals with sexually transmitted diseases are more susceptible to infection with HIV through sexual contact.

This article was adapted from the second edition of *AIDS in New York State*. This report summarizes AIDS surveillance and HIV seroprevalence data, projects AIDS trends through 1994 and estimates present and future health care resource requirements and costs. Limited copies of the report are available from New York State Department of Health Public Affairs Group, Corning Tower, Room 1455, Empire State Plaza, Albany, NY 12237.

FROM THE LIBRARY

PHYSIOLOGIC AND THERAPEUTIC ACTIONS OF ALCOHOL

When the painstaking researches of modern scientific work bring out new facts on old subjects it is worth our while to reconsider our ideas and, if necessary, to rearrange them. . . . The use of alcohol in medicine is so widespread that the subject will be of interest to all, and an accurate idea of the physiologic effects will be of use to us in considering how and when to use it in our daily work. I think that most of us were taught that alcohol in moderate doses was a cardiac and arterial stimulant, increasing the force and frequency of the heart's action, and simultaneously by dilating the capillaries increased the flow of blood through them; that it was a cerebral stimulant; a stomachic; by many considered a tonic; that it was a food, supplying energy though not stored up in the body; that it was a retarder of tissue changes, restricting waste and checking the secretion of nitrogen. There have recently appeared some new experiments and critical reviews of the whole subject which I think must force us to change some of these views. . . .

. . . It is in the infectious diseases that many of our best clinicians hold that alcohol is a benefit and cannot be replaced by any other substitute. Osler expresses it in saying: "We are still without the agent which can counteract the gradual influence of poisons which develop in the course of acute febrile diseases, such as typhoid and pneumonia, * * * the chief effect of which is exercised on the circulation, increasing the rapidity of the pulse and inducing a progressive heart failure. To meet this indication the general experience of physicians still points to alcohol as the most trustworthy remedy. Although some hold that alcohol in this condition is not indicated, I believe that it is, in many instances, the only remedy capable of tiding the patient over the most dangerous period."

. . . My personal belief is that many patients suffering from infectious diseases do not need alcohol, and are better without it; certain patients need it under certain conditions, and are benefited by it. It is also my belief, founded on clinical experience, that because a patient has been accustomed to alcohol is not a sufficient reason for its administration in infectious diseases. Many alcoholics suffering from pneumonia will do better and recover quicker if deprived of alcohol than if given it.

When all is said concerning the value under certain circumstances of alcohol as a food, its use as such certainly belongs to its therapeutic place in medicine and not to its use in every-day life. Atwater correctly says that its place as a nutrient is where the user is unable, because of either debility or disease, to otherwise obtain fitting and sufficient nourishment from ordinary food materials.

Clinical experience is the final proof of the value of alcohol in disease, and though this experience has caused a lessening in its employment, compared with former times, and scientific research has proved its field of usefulness to be more restricted than was formerly accredited to it, alcohol remains a useful and beneficial drug. Like all our best remedies, it is a poison, and in its therapeutic use this fact should never be forgotten. . . .

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(NY STATE J MED 1903; 3:436–443)

PEDIATRIC HEAD INJURIES: A HANDBOOK

By A. Loren Amacher, MD. 129 pp, illustrated. St Louis, Mo, Warren H. Green, Inc, 1988, $24.50 (softcover)

This book's intended audience is the allied health professional. The author, A. Loren Amacher, reviews his 15-year experience as a pediatric neurosurgeon. The first half of the monograph reviews the salient points of the neurosciences (anatomy, physiology, and pathophysiology), while the other half deals with their clinical aspects, including prevention and rehabilitation. Although the handbook makes many good points, it is too brief for consideration by neurologists and neurosurgeons.

The author emphasizes several points important to the care and treatment of the head injured child. A head injury that produces unconsciousness must not be considered trivial. In severe head injuries, young children, in contrast to adults, often can recover function. The brain must have a steady flow of glucose and oxygen. Recent memory, and therefore learning, is frequently affected in even trivial concussions. Superficial events like scalp lacerations and linear skull fractures often dissipate the traumatic force, and in doing so, can decrease brain injury.

In his handbook, Amacher discusses the treatment of multiple trauma and its importance to head injured patients. Intracranial pressure monitoring and its treatment with hyperventilation, hyperosmolar agents, and various drugs are discussed. The author also dispels many misconceptions regarding head injuries, including the fact that steroids have not been proven to be beneficial in the treatment of cerebral edema, post-traumatic seizures frequently are not an indicator of future epilepsy, and most linear skull fractures are of little or no consequence.

There are several controversial issues about which the author tends to be opinionated. For example, he is against the use of prophylactic antibiotics in patients with basal skull fractures and cerebrospinal fluid leaks. He strongly favors the use of the Glasgow Coma Scale, which excludes many important parameters such as pupillary responses, breathing patterns, and oculomotor and brainstem reflexes. Later in the chapter, however, even the author talks about the importance of a skilled observer in picking up minor changes in alertness, breathing patterns and pupillary size as the first sign of an expanding intracranial hemorrhage. The Glasgow Coma Scale is of value in monitoring comatose patients, but not to the exclusion of the other parameters which have stood the test of time.

Amacher has successfully expressed the most important aspects of pediatric head injuries. He discusses several neurologic residuals, including behavioral and learning disabilities, and the importance of prevention and rehabilitation centers for head injured patients. He covers the gamut of pediatric head injuries in a clear and concise manner. The handbook can be read and understood in a matter of a few hours and would benefit most allied health professionals as well as emergency room physicians in the care and treatment of pediatric head injuries.

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TRAUMA

Edited by Kenneth L. Mattox, MD, Ernest Eugene Moore, MD, and David V. Feliciano, MD. 960 pp, illustrated. East Norwalk, Conn, Appleton & Lange, 1988. $125.00 (clothcover)

This is a multi-authored volume providing not only the latest innovations in the care of the injured patient, but a scholarly review of the developmental history of current surgical practice. The editors wisely begin with an overview of the current magnitude of the trauma problem, an authoritative history of trauma care, and recent developments in prehospital management and trauma center design. Especially noteworthy is the chapter on the medicolegal aspects of trauma care, stressing the responsibilities of all members of the treatment team in maintaining an evidentiary trail.

Separate chapters stress trauma scoring and the importance of registries, the general assessment of the trauma patient, and the steps in managing the patient during the resuscitative and operative phase. The specific chapters on system and organ injuries are complete and current up to the beginning of the current year, and each is accompanied by an extensive bibliography worthy of a collective review. In the last part of this enormous compendium, 11 chapters treat special problems in trauma ranging from pediatric trauma, through military considerations about injured patients, to trauma in the geriatric age group. Finally, complications in trauma care are discussed in an authoritative series of monographs.

This is a superb volume. Each of the authors is recognized both nationally and internationally as an expert in his respective field. The currency of the information contained in this volume, as well as the exhaustive references, make this the premier choice of textbooks in this newly recognized field of surgical practice.

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CURRENT TRENDS IN UROLOGY

Volume 4. Edited by Martin I. Resnick, MD. 190 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1988. $48.50 (hardcover)

This book is authored by 17 widely recognized authorities in the field of urology and represents an important contribution to the literature. The authors represent ten institutions which are currently among the leading urological centers in the United States. The book contains ten chapters, all dedicated to treatment of urological diseases. Five chapters deal exclusively with the treatment of urological malignancies, including radical retropubic prostatectomy, systemic and local treatment for genitourinary malignancies, radiation for prostatic cancer, and recent advances in the treatment of testicular cancer. The remaining five chapters cover new concepts in treating impotence, the use of the laser, urological management of spinal cord injury patients, female incontinence, and bladder substitution.

Despite a great number of contributing authors the book is evenly written, which gives great credit to the editor. Most of the chapters not only review current knowledge in the field, but also eloquently elaborate controversial points, especially those chapters dedicated to neoplasms. This book is not only required reading for urologists but informative for general practitioners, medical oncologists, gynecologists, radiologists, radiotherapists, and others interested in new advances in urologic medicine. All chapters are concisely written. It is hoped that Dr Resnick will continue with his outstanding contributions to the urological literature.

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THE ESSENTIAL BOOK OF TRADITIONAL CHINESE MEDICINE. VOLUME I: THEORY

By Liu Yanchi. 305 pp, illustrated. New York, Columbia University Press, 1988. $40.00 (hardcover)

THE ESSENTIAL BOOK OF TRADITIONAL CHINESE MEDICINE. VOLUME II: CLINICAL PRACTICE

By Liu Yanchi. 479 pp, illustrated. New York, Columbia University Press, 1988. $80.00 (hardcover)

Volume I. There are several reasons to recommend this volume for beginners in traditional Chinese medicine. Many English books on this subject are specialized texts on acupuncture, which can cause some readers to equate the two. The present volume is a comprehensive introduction, covering all aspects of traditional Chinese medicine. Compared to other books on traditional Chinese medicine, this volume is easily readable and understandable, reflecting a successful collaboration between the author and his English translators.

There have been a number of recurrent problems with previous books on this subject. Numerous books in English introduce readers to concepts and theories as well as to treatment modalities (mostly only on acupuncture), without making an attempt to link the treatment modalities with basic concepts and theories. Many books omit diagnostic approaches altogether, even though diagnosis is the cornerstone linking the basic concepts and theories to treatment approaches. A few books have attempted to link the fundamental theories with practice, without logical explanations.

This volume is devoted to the theory of traditional Chinese medicine, which is often difficult for Western beginners to comprehend. The author attacks this issue by writing in a lecture-note style, whereby beginners are guided in a step-by-step fashion in grasping the meaning of fundamental theories. Each subject is described in a succinct and deductive manner. This book also details methods of examination and the differentiation of symptom complexes. In short, the author, Dr Liu Yanchi, associate professor at the Beijing college of traditional Chinese medicine, has done a superior job in explaining the fundamentals and their logical extensions into the formulation of diagnostic approaches and management decisions.

The sponsorship of this book by the Chinese Ministry of Public Health does not automatically grant it authoritative status in the field. However, it does mean that the content of this book meets the official standards and requirements of the training curriculum in traditional Chinese medicine in China. This book can be viewed as the mini-curriculum in traditional Chinese medicine, and will be helpful in preparing those who are interested in pursuing a career in it.

Since the introduction of Western medicine in China, there has been a call for integrating Western with traditional Chinese medicine. The latter views the patient as a whole; it adopts a holistic approach with particular attention to demographic, social, emotional, environmental, occupational, and situational characteristics to assess the patient's health status in order to map management plans in restoring or improving it. Epidemiological and observational approaches are employed for differentiation of symptom complexes. These theoretical approaches are not much different from their Western counterparts.

The author recognizes the difference in explaining the interrelationships of internal organs (ie, the Zangs and Fus) among physicians who are trained in Western medicine, and those who are trained in traditional medicine. He describes the Chinese version of five elements in a highly professional fashion. The five elements are wood (liver and gall bladder), fire (heart and small intestine), earth (spleen and stomach), metal (lungs and large intestine), and water (kidneys and urinary bladder).

Future scientific research is needed to uncover new knowledge derived from the theory of traditional Chinese medicine, and to facilitate its possible integration with Western medical sciences.

Although many physicians trained in Western medicine may not appreciate the physiological basis of the channel system, they usually do recognize the acupuncture points in relieving pain. Several studies are currently underway to identify the mechanism of action of these acupuncture points. Future studies may be able to identify those effective points in which physiologic explanations are available, and a reconciliation of the theories of traditional Chinese medicine and Western medicine may be possible.

Volume II. This volume is devoted to the clinical practice of traditional Chinese medicine. The scope as defined by the author includes common ailments frequently encountered in a community setting. The diagnosis of these is based on the theory of traditional Chinese medicine which is described in volume I. As illustrated by case histories, there exists an astonishingly high probability of establishing the diagnosis of these common medical problems by employing the theory of traditional Chinese medicine, and by methods used in modern Western medicine. The similarity in diagnosis may in part represent the adoption of similar epidemiological and statistical models in the diagnostic approach by physicians trained in both systems. Certainly, further study is warranted to determine whether the similarities are, indeed, statistically significant. Such studies could be conducted in China or Taiwan where physicians who are trained in both systems are available. There are Western and traditional Chinese practitioners working side by side at the Min An Health Center, which is located in San Francisco's Chinatown.

The common conditions discussed in this volume include 32 disease entities: the common cold, fever due to internal injury, pneumonia, pleurisy, acute gastroenteritis, diarrhea, primary hypertension, coronary atherosclerotic heart disease, cerebrovascular accidents, nephritis, low back pain, seminal emission, impotence, insomnia, syncope, viral hepatitis, Japanese B encephalitis, epidemic parotitis (mumps), ascariasis, amenorrhea, uterine hemorrhage, dysmenorrhea, threatened abortion, agalactia, salpingitis, menopause, acute cholecystitis and cholelithiasis, acute appendicitis, thromboangitis obliterans, burns, urticaria, and Meniere's disease. For each condition, management regimens for both herbal medicines and acupuncture therapy are discussed. The author does not present his preference or indicate the superiority of one method against another. Clinical trials comparing the effectiveness of the herbal medicines against acupuncture therapy for the same condition have not been performed. Moreover, clinical trials to compare the effectiveness of the standard Western therapy against the method of choice in traditional Chinese medicine are to date lacking. Case histories are provided for the following conditions which illustrate the effectiveness of treatment by traditional Chinese medicine: the common cold, fever due to internal injury, pneumonia, acute gastroenteritis, primary hypertension, coronary atherosclerotic heart disease, cerebrovascular accidents, nephritis, impotence, syncope, viral hepatitis, Japanese B encephalitis, epidemic parotitis, ascariasis, amenorrhea, uterine hemorrhage, threatened abortion, cholecystitis and cholelithiasis, appendicitis, and thromboangitis obliterans. These case histories are very useful for beginners in traditional Chinese medicine to familiarize themselves with diagnosis and treatment approaches of the above conditions. It is hoped that case histories will be available for all conditions listed in this volume in the subsequent editions.

The use of medicinal herbs is described in detail, with a table which compiles 250 commonly prescribed medicinal herbs. These include plants, animals, and minerals. Although indications are listed, it would be interesting to conduct a controlled study to document the effectiveness of each herb for specific disease entities. Traditional Chinese medicine is noted for its prescriptions and the present volume contains a table which lists 150 of the most commonly used.

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TRANSCENDENCE OF LOSS OVER THE LIFE SPAN

By Patricia Weenolsen, PhD. 559 pp. New York, Hemisphere Publishing Corp, 1988. $70.00 (cloth)

This book attempts to examine how the individual overcomes a sense of loss and how one tries to continue on with life through the aging process. It sets forth its assertions based on a study that followed a group of women, aged 25 to 67, for an extended period of time. The women participating in this study were randomly selected by telephone from the Santa Cruz, California, area.

The book's author, Patricia Weenolsen, is a research associate and lecturer at the University of California at Santa Cruz. She received an Outstanding Faculty of the Year Award in 1986 from Seattle University, and has been involved in the field of death and dying through university teaching.

Reading about death and dying is not an easy task. Yet it is an extremely important one. This type of book may be of help by bringing forth ways in which loss has been dealt with by a group of women.

The book is divided into 12 chapters, and in addition there are four appendices and an extensive list of references. All of the chapters include a substantial number of case discussions which add to the clarity of the individual chapters. Definitions are reasonably clear, and the chapters are presented in a readable manner. Most deal with the various areas of loss. There are also two important chapters on life meaning and on time of death.

This book would be of interest to those in the health care field who feel that they need to be more knowledgeable about this area of the life cycle—death.

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COMPREHENSIVE REVIEW IN TOXICOLOGY

Second Edition. By Peter D. Bryson, MD. 696 pp, illustrated. Rockville, Md, Aspen Publishers, Inc. 1989. $137.00 (hardcover)

This textbook represents a second edition by Peter Bryson, a well-known medical toxicologist who offers clinical toxicology seminars throughout the country. Without the assistance of collaborators, he presents an effort of almost 700 pages including 54 chapters and a 60-page index. The tables which he provides are concise and very readable.

Comprehensive Review in Toxicology also contains many mnemonics which are of invaluable assistance to the student of clinical toxicology. Other notable features in this book are two appendices. One presents the therapeutic blood concentrations of selected drugs, while the second offers the volume of distribution of selected drugs. The latter is particularly helpful when one is investigating the pharmacologic features of a toxin and potential therapeutic interventions (eg, hemodialysis).

The textbook, however, does have some shortcomings. There are minor factual errors. While there are many references, a large number are review articles or very old. The book leaves out many toxins which are common (depending on geography) and essential to the teaching of clinical toxicology. Thus the discussions of toxic plants and marine toxicology are brief. There is little discussion of such toxins as fluoride, bromide, copper, and boric acid. Finally, little attention is devoted to occupational/environmental toxicology, and many toxic pesticides are incompletely discussed.

This book is best suited for nonphysicians and emergency department physicians with a budding interest in clinical toxicology. It will make a valuable addition to their libraries.

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PSYCHIATRY AND MENTAL HEALTH SCIENCE HANDBOOK

Co-edited by Claudewell S. Thomas, MD, and Jacob Jay Lindenthal, PhD, 267 pp. St Louis, Mo, Warren H. Green, Inc, 1988. $29.50 (paperback)

The biopsychosocial model of illness continues to gain acceptance and applicability. *Psychiatry and Mental Health Science Handbook* differs from many publications in this pharmacologically dominated era by focusing on the social context in which the major psychiatric illnesses develop. This emphasis on the sociocultural forces which shape behavior and influence the treatment of mental illness is the common denominator of the various chapters in this recently published book.

The book is divided into two major sections. The first, entitled "Clinicopathological Issues," summarizes the recent literature on a number of clinical entities, such as schizophrenia, organic mental disorders, substance abuse, and sleep disorders. A long chapter devoted to childhood psychiatric disorders is particularly well written and presents in a concise fashion a lot of interesting and relevant information. Most of the diagnostic categories are discussed from a sociocultural point of view, with the chapter on schizophrenia, for example, providing a nice overview of deinstitutionalization and changing community treatment approaches. The chapter on brain disorders concludes with a frankly poignant section entitled "Who Is Left When the Experts Leave?" in which mental health professionals are reminded of the emotional pain suffered by brain-diseased patients and their families. An unfortunate consequence of our advances in laboratory and imaging techniques may be a tendency to focus on tests and "making a diagnosis," while ignoring the ways in which these disorders affect the lives of our patients.

The second section, entitled "Psychosocial Issues," examines topics often omitted or shortchanged in other texts. These include homicide, rape, the changing role of privacy and confidentiality, and even the growing use of computers in mental health. The final chapter of the book, using the example of Bernard Goetz, examines how social norms of what is and what is not acceptable behavior both influenced and were influenced by the Goetz case.

Obviously, these are wide-ranging issues and constitute both the strength and the weakness of this book. Those readers seeking a concise diagnostic and treatment manual suggested by the word "handbook" in the title may be disappointed with the broad, somewhat unwieldy subject matter and the sociocultural perspective. Other readers, however, may be delighted to find a book which "stands back" and takes a fresh look at our patients and their disorders in the context of our society and treatment delivery systems.

The book is a little wordy and editorializing for a so-called "handbook," and typographical and even grammatical errors occur with irritating frequency. Despite these drawbacks, the book succeeds in reminding us that even though behavior has important biologic and intrapsychic substrates, it also is firmly rooted in the values, norms, and dynamics of society and culture. From that perspective, this book is unique and differs from most other currently available synopses.

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THE RESPONSIBLE PHYSICIAN: STANDARDS OF EXCELLENCE AND THE CRITICALLY ILL PATIENT

Edited by Stewart G. Wolf, Jr, MD, Milton W. Hamolsky, MD, Austin H. Kutscher, MD, and Karin Muraszko, MD. 102 pp. Brooklyn, NY, The Center for Thanatology Research, 1989. $14.95 (paperback)

This small softcover book (102 pages) is one of a series regularly produced by the Foundation of Thanatology. Founded in 1976, and operated by a small staff in New York City, this foundation has as its goal "scientific humanistic inquiries into death, loss, grief, and bereavement." It produces *Advances in Thanatology* (quarterly), *Archives of the Foundation of Thanatology* (quarterly), and *Thanatology Abstracts* (annual). The foundation also publishes books on this subject.

The editors of the current volume include Stewart G. Wolf, Jr, Milton W. Hamolsky, Austin H. Kutscher, and Karin Muraszko. There are three sections to the book: "Responsible Physician: Standards of Excellence," "Perspectives on the Care of the Patient," and "Philosophical Overview of the Nature of Man." There are six essays in section I, four in section II, and two in the final section. The contributors of the essays appear to be practicing physicians with few exceptions, and they include pediatricians, oncologists, a nursing instructor, psychiatrists, professors of medicine, a neurosurgical resident, a gerontologist, and two emeritus professors.

The organization and presentation of the topics are logical. Communication, physician responsibility, training, the principles of thanatology, and a physician's thoughts on dying comprise section I. Section II has essays on terminal versus critical illnesses, goals and management, hospital harassment, an excellent section on pediatric care, which differs considerably from adult care, and an essay on the place of high technology with regard to its use in the care of the elderly. Finally, section III speaks about death and the nature of man and disease and the role of the physician.

I found the book more practical than I expected it to be and cannot think of areas which were not addressed. The spiritual aspect of the care of critically and terminally ill patients was not directly addressed, but was mentioned in the pediatric essay. It may have been of value to have had some theological insights, although the tone and purpose of the book were primarily designed for and specifically addressed to the medical and ethical aspects of the seriously ill.

This volume is small, easy to read, and may well be worth possessing for more than a single reading, as we encounter, far more commonly than previously, areas where our scientific technology has gotten ahead of our artful applications in medical patient care. Ignoring this area is no longer acceptable, and all of us face these types of dilemmas nearly every day in clinical practice. It is important that we continue to search for practical and applicable solutions to these ethical problems.

Modest cost and easy readability make this an attractive and worthwhile addition to the practitioner's library.

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RATIONING OF MEDICAL CARE FOR THE CRITICALLY ILL

Edited by Martin A. Strosberg, I. Alan Fein, and James D. Carroll. 97 pp. Washington, DC, Brookings Books, 1989. $11.95 (paperback)

This book is a conference dialogue on public policy by administrators, economists, ethicists, lawyers, philosophers, physicians, politicians, and third party payers. Essentially, two sensitive and emotionally laden questions are addressed: Can cost-containment and efficiency measures obviate the need to ration medical care for the critically ill? Must or should physicians act as "society's gatekeepers"?

From his background as an economist, Henry J. Aaron readily disposes of the first question, stating that efficiency is not the answer. It is estimated that $15–$20 billion out of the annual $450 billion national health expenditures are wasted on unneeded services. Even squeezing out all the inefficiency, expenditures will still rise 1% of the Gross National Product every three or four years. Citing the British experience, Aaron notes that there is ten times more coronary artery surgery in the US; three times more renal dialysis, and much less chemotherapy for patients with cancer. In fact, it is standard policy there not to provide dialysis to people older than 55 experiencing renal failure. A British physician puts the rationale bluntly, even if unpalatably: "Oh, but you have to understand—everybody over the age of fifty-five is a bit crumbly."

Ethicist Robert Veatch thinks that the physician is the most likely person to do the rationing. He proposes replacing the Hippocratic Oath with the following warning: "I will generally work for your interests, but in the case of marginally beneficial expensive care, I will abandon you in order to serve society as their cost-containment agent."

Max Arie Weil suggests an alternative to rationing that considers the three Rs: Is care *R*ational, *R*edeeming, and *R*espectful? He ponders whether to treat a 65-year-old man with a 95% body burn, or to attempt to sustain a 250 gram infant fetus. In both situations, a patient has never survived. Risk benefit analysis is the decision pathway which leads to Weil's conclusion: ". . . if it is rational, redeeming, and respectful, the resources that we presently have will be aplenty."

Congressman Willis D. Gradison, Jr (R-OH), clarifies the "hot potato" concept of legislative sociopolitical cultural decisions. "Members of Congress will be the last people in the country to have their fingerprints on any policy that even appears to deal with the rationing of health care." Obviously, Congress will be happy to have the physicians make those decisions.

Anesthesiologist Michael A. Rie comments that the conference avoided the issue of when and how our society creates a public allocation policy. Instead, Rie and ethicist H. Tristam Englehardt opt for a formula: Intensive Care Unit Entitlement Index (ICUEI) $= {}^{PQL}/_{C}$. P is the probability of a successful outcome; Q is the quality of success; L is the length of life remaining to a patient; and C the costs required to achieve therapeutic success. Interestingly, Lindsay Robinson, a third party payer executive, pointed out the default of policy makers to cost containment goals: "Reimbursement incentives should serve as a tool to implement social policy and never as a replacement for it."

After the conference, Robert Baker, I. Alan Fein, Martin A. Strosberg, and Weil came up with proposals and subsequent actions to reform the health care system. Possibly, the reforms could forestall or even preempt the need for explicit rationing of intensive care medicine.

They first suggest that admission criteria should be tightened to restrict care to patients likely to benefit while excluding those for whom no ultimate benefit can be predicted, where survival is extremely improbable, and where iatrogenic risks exceed potential benefits. Second, it is proposed that consent procedures be reformed, allowing for advance directives in specific situations; a living will measure. Third, the authors urge the medical community to provide leadership regarding the legal aspects of defining concepts of acceptable medical practice to yield societal consensus. As lawyer Barbara R. Grumet commented during the conference, the legal system relies upon medical peer group standards for acceptable care. Finally, the authors call upon a number of medical organizations to adopt the concept of prognostic admissions policies. Those prognostic estimates could be used to develop criteria on admissions.

In addition to all of the above, John S. Morris discusses the cultural issues, William A. Knaus reviews explicit intensive care unit (ICU) allocation policies, Baker reviews the background of do-not-resuscitate orders, and Daniel Teres looks at ICU triage and anticipates that civilian triage will be common in the future.

While the arguments about the rationing of medical care for the critically ill are not new, this 97-page book does focus in on the ICU utilization issue. In easily comprehensible language, the points are made for the reader's consideration. Some of the quotes and concepts may not sit easily with health care providers, especially physicians. Realizing that these rationing decisions may actually come to fruition provides a compelling reason for physicians to prepare themselves for the social, economic, and political debates that are sure to continue. Reading this compact book is a solid beginning.

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Sustained-release nicotinic acid carries increased risk of hepatic failure

Nicotinic acid is a common and effective treatment for hypercholesterolemia. It is widely available without a prescription, making it important for both physicians and laypersons to be aware of the associated risks. Doses of greater than 3 g/day increase the risk of hepatotoxic complications. Mullin et al described a patient who had taken nicotinic acid for more than a year without side effects; on switching to sustained-release nicotinic acid, he experienced fulminant hepatic failure (*Ann Intern Med* 1989; 111:253–255).

After undergoing coronary angioplasty, a 44-year-old man began therapy with crystalline nicotinic acid for hypercholesterolemia. He tolerated well a dose of 6 g/day, and more than a year later, his liver enzyme levels were within normal limits. Shortly thereafter, the patient switched to 6 g/day of sustained-release nicotinic acid, which he had bought from a health food store. Within three days, symptoms including nausea, vomiting, abdominal pain, low-grade fever, dark urine, and gingival bleeding developed.

Within a 24-hour period, hepatic failure developed, at which time the patient was comatose and unresponsive to deep pain. He recovered following successful liver transplantation.

The authors presumed that the "fulminant hepatic failure resulted from use of sustained-release nicotinic acid." They based this conclusion on the patient's lack of liver dysfunction while taking crystalline nicotinic acid, the short amount of time between his first taking sustained-release nicotinic acid and the onset of hepatic failure, and the lack of findings to substantiate a viral or toxicologic cause.

Other authors have reported "a higher frequency and degree of hepatocellular injury with sustained-release forms of nicotinic acid without additional therapeutic advantage, compared with crystalline preparations." Mullin and colleagues suggested close monitoring of patients taking sustained-release forms of nicotinic acid.

Metastatic Crohn's disease

Crohn's disease, primarily a gastrointestinal disorder, can produce extraintestinal involvement. "Metastatic" Crohn's disease is a term used to describe "extraintestinal involvement not due to direct extension of the disease" (*West J Med* 1989; 151:203–205). The clinical and histologic presentation of the skin in metastatic Crohn's disease is similar to that seen in sarcoidosis. McGillis and Huntley described a patient with granulomatous lesions of the skin initially diagnosed as sarcoidosis, but later determined to be metastatic Crohn's disease.

The 62-year-old patient presented with an erythematous, scaly, nummular patch on his left lower leg. Six months previously, symptoms of malaise, diarrhea, vomiting, and a 20-lb weight loss led to a diagnosis of Crohn's disease, based on the finding of noncaseating granulomata on endoscopic biopsy. Therapy at that time with prednisone, 30 mg/day, led to complete resolution of all gastrointestinal symptoms.

Mid-strength topical steroid therapy for the skin lesions that developed later was ineffective. The lesions progressed, thickened, and ultimately formed ulcerations and crusting. Skin biopsies led to a finding of sarcoidal granulomas, based on which an initial diagnosis of sarcoidosis was made. However, no systemic sarcoidal involvement was found, and the patient exhibited no adenopathy and no evidence of tuberculosis. "Because of the established diagnosis of Crohn's disease involving his bowel and the lack of involvement in other target organs typical for sarcoidosis, his cutaneous reaction seemed to best fit into the category of 'metastatic' Crohn's disease." The skin lesions cleared with oral prednisone.

Differentiating between sarcoidosis and metastatic Crohn's disease requires an evaluation of extracutaneous involvement. Target tissues for sarcoidal infiltration include the lungs, skin, lymph nodes, and eyes; whereas gastrointestinal involvement is uncommon in sarcoidosis. The authors note that about 44% of patients with Crohn's disease exhibit skin disorders, and these are most prevalent in patients with large bowel and colon involvement.

Dose-response relationship demonstrated between cigarette smoking and histologic changes in the urinary bladder

Auerbach and Garfinkel analyzed urinary bladder epithelium isolated from autopsy samples (*Cancer* 1989; 64:983–987). They documented all histologic abnormalities suggestive of preneoplastic alterations, and compared these findings to the smoking habits and artificial sweetener use of the patients. Evidence of a dose-response relationship of cigarette smoking and histologic changes in the urinary bladder supports a similar relationship previously reported for smoking and cancer of the urinary bladder.

The predominant histopathologic finding was atypical nuclei in the epithelial cells; the number of cell rows in the epithelium was another common alteration.

The authors found that 85.3% of tissue sections from nonsmokers had no cells with atypical nuclei. "The percentage of sections having cells with 50% or more atypical nuclei increased from 4.3% in those who never smoked to 88.4% in subjects who smoked 40 or more cigarettes per day." The percentage increased in direct proportion to the number of cigarettes smoked per day. Eighty-three percent of sections from persons who had never smoked had a normal number of basal cells in the urinary bladder epithelium. "The number of cell rows increased with the number of cigarettes smoked per day." In cigarette smokers who no longer smoked, as well as pipe and/or cigar smokers, the percentages were lower than those for persons who smoked cigarettes up until their death, but were higher than those in nonsmokers.

The histologic abnormalities included hyperplastic changes, squamous cell metaplasia, carcinoma in situ,

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NEWS BRIEFS are compiled and written by Vicki Glaser.

cystitis cystica, and acute or chronic inflammation.

Artificial sweetener use showed no correlation with atypical nuclei or the number of cell rows.

One hospital's experience with a total ban on smoking

At The Johns Hopkins Medical Institutions (Baltimore, Md), people smoking—both staff members and visitors—is a common sight in most public areas. Given the acknowledged health risk of smoking, as well as passive smoking, it seems incongruous for smoking to occur so freely in a health care institution.

As the first phase of a no-smoking policy that began in 1987 at these institutions, administrators decided to implement a total ban on smoking in The Johns Hopkins Children's Medical and Surgical Center, a self-contained pediatric hospital within the Johns Hopkins complex. An evaluation of the no-smoking program was reported in a recent issue of *JAMA* (1989; 262;799–802).

Prior to this new policy, smoking was permitted in designated lounges, although the authors observed "marked noncompliance among both visitors and staff, with highly visible smoking occurring throughout the center." Six months prior to the ban on smoking, administrators began a program aimed at preparing employees and the environment for the change in public policy. An advisory committee comprised of employees participated in the preparatory program, which included an "awareness-arousing survey of smoking behavior and attitudes of employees," newsletters outlining the policy and offering information on smoking cessation options, "Health Awareness Days" with free health screening and counseling, and media exposure of the new policy in the local press and in publications within the medical institutions. One month prior to initiation of the ban and thereafter, parents of patients received a written statement explaining the no-smoking policy as part of the admissions procedure. A press conference marked the first day of the ban, accompanied by the placement of no-smoking signs and the availability of "quit kits" for parents of patients.

The authors' evaluation of the smoking policy spanned a period of from six months before the ban to six months after. Their findings were based on a survey, unobtrusive observations of smoking activity in the hospital, cigarette butt counts in ashtrays, and environmental nicotine vapor concentrations. The survey revealed that while 82% of smokers reported smoking during work hours six months before the ban, only 43% did so six months after the ban. Observations two weeks prior to the ban found 53% of staff and visitors smoking in the hospital. Six months after the ban, no smoking was reported during a comparable observation time. There were 83 times fewer cigarette butts found during the daytime after the ban than before the ban, with a 44-fold decrease found in overnight counts.

Although an insignificant number of employees either stopped smoking or smoked fewer cigarettes as a result of this ban on smoking in the workplace, most smokers complied with the ban and no longer smoked at the hospital. The authors concluded that "a total ban on smoking in a hospital is feasible and effective in virtually eliminating public smoking."

Frequency of work-related carpal tunnel syndrome may be underestimated

Carpal tunnel syndrome (CTS) produces "pain, numbness, and weakness in the median nerve distribution of the hand as a result of compression or irritation of the median nerve as it passes through the carpal tunnel in the wrist." (*MMWR* 1989; 38:485–489). Work-related CTS results from continual trauma to the hand and wrist, such as butchers, grocery checkers, typists, musicians, and carpenters might experience.

A survey of health care practitioners by the California Occupational Health Program revealed that 4.7% of cases of carpal tunnel syndrome may have been work related, yet only a small percentage of these cases were reported to the state. Physicians must report all cases of suspected occupational injury or illness to the state, according to the California Doctor's First Report program.

Recognition of work-related CTS is essential for effective treatment. Unless the patient's work activities are evaluated and redesigned, CTS can progress to increasing discomfort, impaired hand function, and disability. Depending on the severity of the condition, treatment can range from wrist immobilization and nonsteroidal anti-inflammatory therapy to surgery.

Occupational factors that contribute to carpal tunnel syndrome include the following: "1) repetitive movements of the hand, 2) forceful grasping or pinching of tools or other objects, 3) awkward positions of the hand and wrist, 4) direct pressure over the carpal tunnel, and 5) use of vibrating hand-held tools."

FAX machines: standard medical equipment?

The ability to have nearly instantaneous access to patient information such as medical records and test results makes sense for any physician, and especially those involved in any aspect of emergency medical care. As a cardiologist, Richard S. Ader, MD, has found that "most of cardiology is visual. I receive frequent calls from emergency rooms about electrocardiograms and from nurses in the evening about rhythm strips and patterns on the monitor." In a recent letter to the *New England Journal of Medicine* (1989; 320:1629), Ader explains how facsimile or FAX machines have improved his access to critical data and, thereby, enhanced patient care.

Ader uses facsimile machines to create a visual communications network between the coronary care units of three hospitals and his home. He can now view electrocardiograms and rhythm strips via the telephone 24 hours a day. Coronary care units and emergency rooms of most hospitals could benefit from having facsimile machines as standard equipment.

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

A. Leonard Abrams, MD, New York. Died July 30, 1989; age 66. Dr Abrams was a 1948 graduate of New York University School of Medicine. He was a Diplomate of the American Board of Psychiatry and Neurology and a Fellow of the American Psychiatric Association. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Morris Birnbaum, MD, Forest Hills. Died April 25, 1989; age 82. Dr Birnbaum was a 1931 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the American Academy of Family Physicians, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Harold Frederick Buckbee, MD, Dolgeville. Died February 1, 1989; age 85. Dr Buckbee was a 1930 graduate of the Albany Medical College of Union University. He was a member of the Medical Society of the County of Herkimer and the Medical Society of the State of New York.

Benjamin H. Budoff, MD, Boca Raton, Florida. Died February 9, 1989; age 72. Dr Budoff held a Registrable Qualification Granted by the Scottish Conjoint Board in 1942. He was a Diplomate of the American Board of Otolaryngology. His memberships included the New York Academy of Medicine, the American Academy of Otolaryngology, the Pan American Medical Association, the Nassau County Medical Society, and the Medical Society of the State of New York.

Giovanni Capozzi, MD, Queens Village. Died July 29, 1989; age 66. Dr Capozzi was a 1949 graduate of the Facolta di Medicina e Chirurgia dell'Universita di Napoli, Naples, Italy. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Roland Benedict Carr, MD, Naples, Florida. Died December 19, 1988; age 88. Dr Carr was a 1924 graduate of the State University of New York at Buffalo School of Medicine. He was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

William Gaston Chorba, MD, Bronx. Died July 6, 1989; age 87. Dr Chorba was a 1925 graduate of Columbia University College of Physicians and Surgeons. He was a Fellow of the American College of Surgeons. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Hugh Mason Hicks, MD, New York. Died July 13, 1989; age 85. Dr Hicks was a 1926 graduate of Columbia University College of Physicians and Surgeons. He was a Diplomate of the American Board of Internal Medicine. He was a member of the New York Academy of Medicine, the American Geriatrics Society, the New York County Medical Society, and the Medical Society of the State of New York.

Jed Hotchkiss Irvine, MD, New York. Died July 6, 1989; age 85. Dr Irvine was a 1929 graduate of the University of Virginia School of Medicine. He was a Diplomate of the American Board of Internal Medicine and a Fellow of the American College of Physicians. His memberships included the New York Academy of Medicine, the American Rheumatism Association, the New York County Medical Society, and the Medical Society of the State of New York.

Mildred M. Wicker Jackson, MD, Hemlock. Died May 17, 1989; age 91. Dr Jackson was a 1923 graduate of the Cornell University Medical College. She was a Diplomate of the American Board of Pediatrics and a Fellow of the American Academy of Pediatrics. She was a member of the Medical Society of the County of Livingston and the Medical Society of the State of New York.

James H. McDonough, MD, Rome. Died March 4, 1988; age 79. Dr McDonough was a 1942 graduate of the University of Pennsylvania School of Medicine. He was a Diplomate of the American Board of Preventive Medicine, Inc. His memberships included the New York Academy of Medicine, the American Academy of Occupational Medicine, the American Occupational Medical Association, the Medical Society of the County of Oneida, and the Medical Society of the State of New York.

Salvatore P. Squitieri, MD, Manchester, Maryland. Died April 18, 1989; age 81. Dr Squitieri was a 1934 graduate of the St Louis University School of Medicine. He was a Member of the American Academy of Family Physicians, the Bronx County Medical Society, and the Medical Society of the State of New York.

John Henry Sullivan, MD, Canastota. Died July 5, 1989; age 82. Dr Sullivan was a 1933 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a member of the Medical Society of the County of Sullivan and the Medical Society of the State of New York.

Rudolph E. Vandeveer, MD, Winter Park, Florida. Died June 25, 1989; age 83. Dr Vandeveer was a 1932 graduate of the Yale University School of Medicine. He was a Diplomate of the American Board of Pediatrics and a Fellow of the American College of Allergists and the American Academy of Pediatrics. He was a member of the New York Academy of Medicine, the Medical Society of the County of Oneida, and the Medical Society of the State of New York.

Jack B. Weissman, MD, New York. Died June 28, 1989; age 44. Dr Weissman was a 1970 graduate of Harvard Medical School. He was a Diplomate of the American Board of Internal Medicine and a Fellow of the American College of Physicians. He was a member of the New York Academy of Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Charles A. Weymuller, MD, Albuquerque, New Mexico. Died July 22, 1989; age 93. Dr Weymuller was a 1920 graduate of the University of Nebraska College of Medicine. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Harry Zimmerman, MD, Margate, Florida. Died June 27, 1989; age 89. Dr Zimmerman was a 1926 graduate of the State University of New York Health Science Center at Brooklyn. He was a Qualified Fellow of the International College of Surgeons. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Harold Zolan, MD, Massapequa Park. Died July 6, 1989; age 72. Dr Zolan was a 1943 graduate of the Chicago Medical School. He was a Diplomate of the American Board of Psychiatry and Neurology and a member of the American Psychiatric Association, the Nassau County Medical Society, and the Medical Society of the State of New York.

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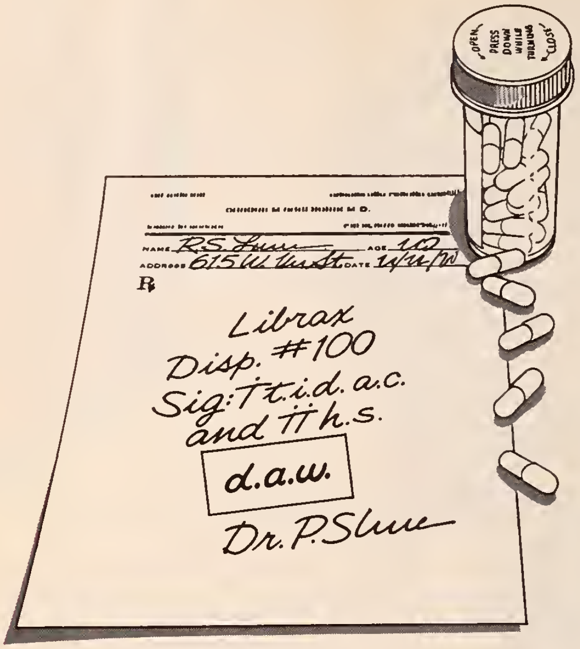

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Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established. Inform patients to consult physician before increasing dose or abruptly discontinuing this drug.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

Drug Abuse and Dependence: Withdrawal symptoms similar to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of chlordiazepoxide; more severe seen after excessive doses over extended periods; milder after taking continuously at therapeutic levels for several months. After extended therapy, avoid abrupt discontinuation and taper dosage. Carefully supervise addiction-prone individuals because of predisposition to habituation and dependence.

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(*continued from p 8A*)

College of Obstetricians and Gynecologists, 409 12th St, SW, Washington, DC 10024-2188. Tel: (800) 533-8811.

DISTRICT OF COLUMBIA

Nov 5–8. **83rd Annual Scientific Assembly of the Southern Medical Association.** 30 Cat I Credits. Washington DC Convention Center. Contact: Education Department, Southern Medical Association, 35 Lakeshore Dr, PO Box 190088, Birmingham, AL 35219-0088. Tel: (205) 945-1840.

Nov 19–21. **Fourth National Forum on AIDS and Hepatitis B.** The Washington Hilton. Contact: American Society for Microbiology, 1913 I Street, NW, Washington, DC 20006. Tel: (202) 833-9680.

FLORIDA

Nov 2–5. **Flexible Fiberoptic Sigmoidoscopy and Naso-pharyngo-laryngoscopy in the Primary Care Physician's Office.** Grosvenor Resort, Lake Buena Vista. Contact: Seminars & Symposia, Inc, 132 E 70th St, New York, NY 10021. Tel: (212) 517-7520.

Nov 8–12. **30th Workshop in Electrocardiography.** Diplomat Hotel & Golf Resort, Hollywood. Contact: Anne Criss, Rogers Heart Foundation, 1200 7th Ave, N, St Petersburg, FL 33705. Tel: (813) 894-0790.

HAWAII

Nov 6–10. **Diagnostic Radiology.** 24 Cat I Credits. Maui Marriott, Maui. Contact: Radiology Postgraduate Education, Room C-324, University of California School of Medicine, San Francisco, CA 94143. Tel: (415) 476-5731.

Nov 30–Dec 4. **AMA-Hospital Medical Staff Section 14th Assembly Meeting.** Sheraton Waikiki Hotel, Honolulu. Contact: Dept of Hospital Medical Staff Services, American Medical Association, 535 N Dearborn St, Chicago, Ill 60610. Tel: (312) 645-4754.

ILLINOIS

Nov 16–18. **Critical Care in Obstetrics and Gynecology.** 16 Cat I Credits. Fairmont Hotel, Chicago. Contact: The American College of Obstetricians and Gynecologists, 409 12th St, SW, Washington, DC 10024-2188. Tel: (800) 533-8811.

MASSACHUSETTS

Nov 1–2. **Lasers in Gynecology.** 23 Cat I Credits. University of Massachusetts Medical Center, Worcester. Contact: The American College of Obstetricians and Gynecologists, 409 12th St, SW, Washington, DC 10024-2188. Tel: (800) 533-8811.

NEVADA

Nov 30–Dec 3. **Nebraska Obstetrics and Gynecologic Society.** Las Vegas. Contact: Brenda C. Ram, Continuing Education, University of Nebraska Medical Center, 42nd & Dewey Ave, Omaha, NE 68105. Tel: (402) 559-4152.

PENNSYLVANIA

Nov 27–29. **The Biological Actions of**

(*continued on p 16A*)

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References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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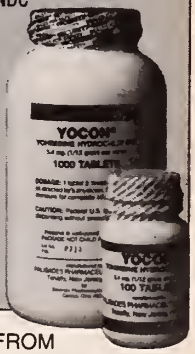

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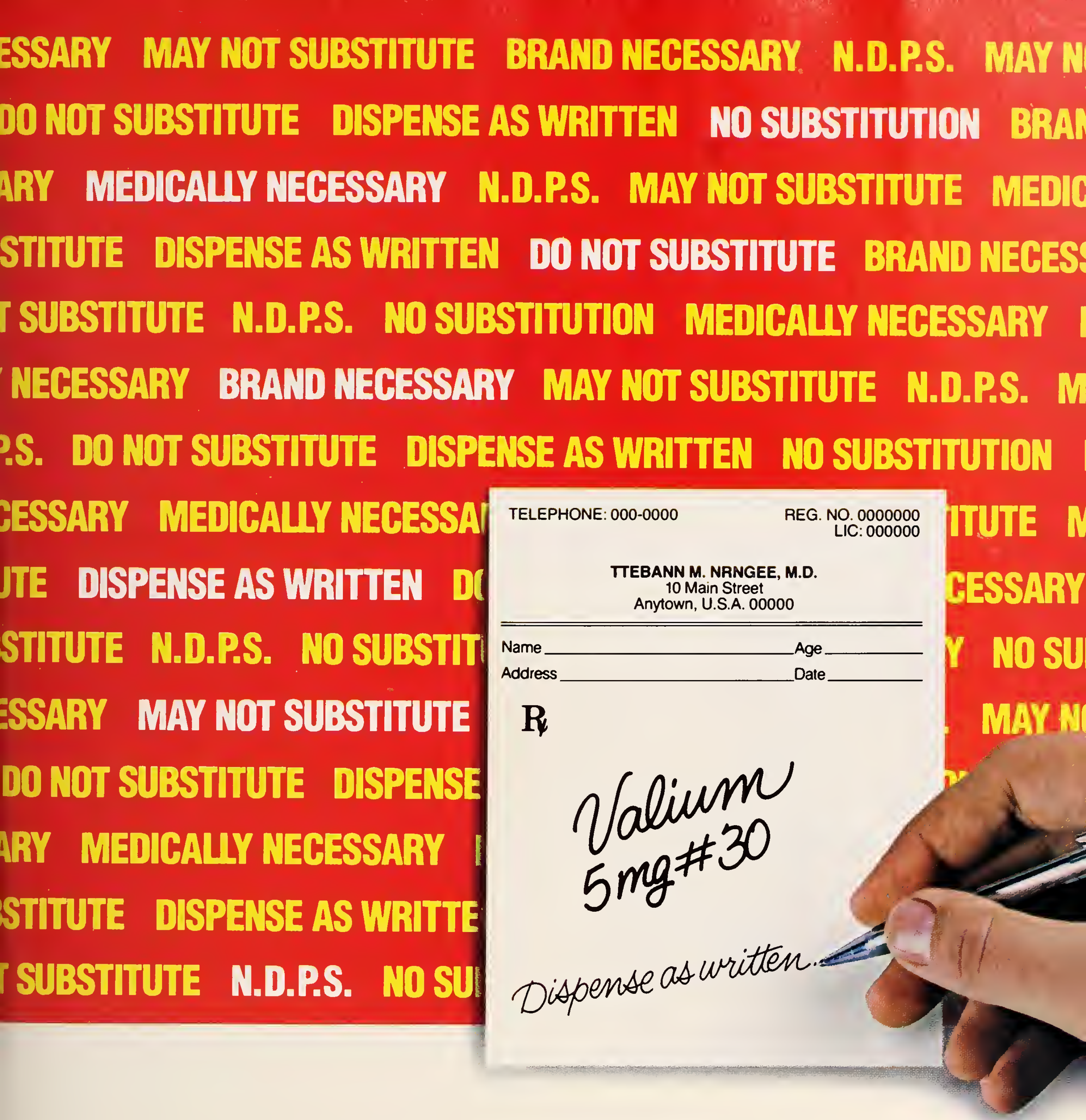
Index to Advertisers

AMA Advisers	15A
Blanksteen Companies	12A
Classified Advertising	6A, 12A, 13A, 14A
Cliggott Publishing	5A
Knoll Pharmaceuticals	7A, 8A
Eli Lilly and Company	2nd Cover
Marine Midland Bank	1A
Merck, Sharp & Dohme	3A, 4A
Palisades Pharmaceuticals	13A
Roche Laboratories	9A, 10A, 3rd & 4th Cover
Shearson, Lehman, Hutton	6A
U.S. Air Force	11A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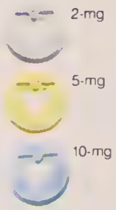

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Contents

COMMENTARIES

The elimination of the requirement for medical libraries in hospitals 601
PETER B. FARNSWORTH, MD

The role of community hospitals in the detection of occult breast cancer 602
NED D. RODES, MD

The rural health care challenge in New York State 603
THOMAS C. ROSENTHAL, MD

SPECIAL REPORT

New York State's health care system: Selected major problems and recommended solutions 606

RESEARCH PAPER

Occult breast cancer in two community hospitals in New York State 612
MUHAMMAD A. HENA, MD; ANDREW C. WARHIET, MD; JOHN C. CROWTHER, MD

REVIEW ARTICLE

Vascular insufficiency: A significant factor in impotence 614
ADRIAN W. ZORGNIOTTI, MD

SPECIAL ARTICLE

A New York initiative in medical education 618
JOHN E. VERBY, MD; MACARAN A. BAIRD, MD; L. THOMAS WOLFF, MD; PETER G. BEATTY, MSEd

HISTORY

Dietary changes and the decline of scurvy and tuberculosis in 19th century Europe 621
VINCENT J. KNAPP, PhD

CASE REPORTS

Meningitis following bacteremia with *Streptococcus sanguis* 625
ROBERT H. K. ENG, MD; ANTHONY J. MANGIA, MD; SHARON M. SMITH, MD; LAWRENCE F. MAHDI, MD; KENNETH STERNBERG, MD

Determination of cerebral blood flow in an arteriovenous malformation using SPECT scanning 627
BRUCE ROSENBLUM, MD; IRA NOVICH, MD; ALLEN ROTHMAN, MD

Treatment of dolichoectasia of the middle cerebral artery with bypass and exclusion 629
R. A. DE LOS REYES, MD; F. HARRISON BOEHM, JR, MD; MARK A. SPATOLA, MD; JAMES T. GOODRICH, MD; FRANKLIN G. MOSER, MD; JOSEFINA F. LLENA, MD; ALFRED J. SPIRO, MD

LETTERS TO THE EDITOR

Omental infarction in pregnancy 634
MELVIN BERLIN, MD

Thallium stress tests and the White House security system 634
BERNARD GITLER, MD; IRA S. NOVICH, MD; ISAIAH J. SELIGMAN, MD

Primary meningococcal conjunctivitis 634
JOHN P. CURRAN, MD; JORGE R. NUNEZ, MD; ERNEST VISCONTI, MD

Caring and serving 635
RANDALL D. BLOOMFIELD, MD

LEADS FROM EPIDEMIOLOGY NOTES 636
BOOK REVIEWS 640
NEWS BRIEFS 644
OBITUARIES 646
MEDICAL MEETINGS AND LECTURES 6A

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If hyperkalemia is suspected, obtain an ECG and monitor serum potassium. If hyperkalemia develops, discontinue 'Dyazide' and initiate thiazide therapy if needed Persistent hyperkalemia may require dialysis Monitor serum electrolytes frequently in patients with mild renal dysfunction and in diabetic patients. In patients who may develop respiratory or metabolic acidosis, monitor serum electrolytes and acid/base balance frequently

Precautions *The bioavailability of the hydrochlorothiazide and triamterene components of 'Dyazide' is about 50% of the maximum obtainable with oral therapy. Theoretically, a patient transferred from therapy with hydrochlorothiazide with or without triamterene might show an increase in blood pressure, fluid retention, or change in serum potassium. Extensive clinical experience with 'Dyazide', however, suggests that these conditions have not been commonly observed in clinical practice. (See CLINICAL PHARMACOLOGY.) Use thiazides cautiously in patients with impaired hepatic function. They can precipitate coma in patients with severe liver disease, potassium depletion induced by the thiazide may be important in this connection; administer 'Dyazide' cautiously and be alert for such early signs of impending coma as confusion, drowsiness and tremor, if mental confusion increases, discontinue 'Dyazide' for a few days, attention must be given to other factors that may precipitate hepatic coma, such as blood in the gastrointestinal tract or preexisting potassium depletion. If patients develop hypokalemia, which is uncommon with 'Dyazide', increase potassium intake (i.e., with supplements or potassium-rich foods). If repeat determinations show serum potassium concentrations below 3 0 mEq/L, discontinue 'Dyazide' and initiate potassium chloride supplementation. Institute corrective measures cautiously and monitor serum potassium concentrations frequently, especially in patients receiving digitalis or those with a history of cardiac arrhythmias. Diuretics may aggravate existing electrolyte imbalances, especially at high dosages or in patients on salt-restricted diets. Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Chloride replacement may be required in the treatment of metabolic acidosis. If dilutional hyponatremia develops, restrict water intake. In actual salt depletion, initiate sodium chloride replacement. Use 'Dyazide' cautiously in patients with a history of renal stone formation.*

If hyperkalemia develops when treating for hypokalemia, take corrective measures Also discontinue 'Dyazide' and, if appropriate, substitute a thiazide diuretic until potassium levels return to normal Do periodic BUN and serum creatinine determinations, especially in the elderly and in patients with suspected or confirmed renal insufficiency Serum PBI levels may decrease without signs of thyroid disturbance Discontinue thiazides before conducting parathyroid function tests.

Angiotensin-converting enzyme (ACE) inhibitors can elevate serum potassium, use with caution with 'Dyazide' Concurrent use with chlorpropamide may increase the risk of severe hyponatremia. A few occurrences of acute renal failure have been reported in patients on 'Dyazide' when treated with indomethacin Therefore, caution is advised in administering nonsteroidal anti-inflammatory agents with 'Dyazide' Diuretics reduce renal clearance of lithium and increase the risk of lithium toxicity. Clinically insignificant reductions in arterial responsiveness to norepinephrine have been reported Thiazides have also been shown to increase the paralyzing effect of nondepolarizing muscle relaxants such as tubocurarine; therefore use cautiously in patients undergoing surgery Monitor electrolytes in patients taking amphotericin B, corticosteroids or corticotropin concomitantly Thiazides may potentiate the action of other antihypertensive drugs. The effects of oral anticoagulants may be decreased when used concurrently with hydrochlorothiazide, dosage adjustments may be needed 'Dyazide' may raise the level of blood uric acid, dosage adjustments of antigout medication may be needed to control hyperuricemia and gout. The following agents given with triamterene may promote serum potassium accumulation and possibly result in hyperkalemia, especially in patients with renal insufficiency: blood from blood bank (may contain up to 30 mEq of potassium per liter of plasma or up to 65 mEq of potassium per liter of whole blood when stored for more than 10 days), low-salt milk (may contain up to 60 mEq of potassium per liter); potassium-containing medications (such as parenteral penicillin G potassium), and salt substitutes (most contain substantial amounts of potassium). Exchange resins, such as sodium polystyrene sulfonate, whether administered orally or rectally, reduce serum potassium concentrations by sodium replacement of the potassium, fluid retention may occur in some patients because of the increased sodium intake Chronic or overuse of laxatives may reduce serum potassium concentrations by promoting excessive potassium loss from the intestinal tract; laxatives may interfere with the potassium-retaining effects of triamterene. The effectiveness of methenamine may be decreased when used concurrently with hydrochlorothiazide because of alkalinization of the urine 'Dyazide' will interfere with the fluorescent measurement of quinidine

There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy only if clearly needed Thiazides and triamterene cross the placental barrier and appear in cord blood The use of thiazides in pregnancy requires weighing the anticipated benefit against possible hazards, including fetal or neonatal jaundice, pancreatitis, thrombocytopenia, and possibly other adverse reactions which have occurred in the adult Thiazides appear, and triamterene may appear, in breast milk. If use of the drug is essential, the patient should stop nursing Safety and effectiveness in children have not been established.

Adverse Reactions *The serious adverse effects associated with 'Dyazide' have commonly occurred in less than 0.1% of patients treated with this product. Anaphylaxis, rash, urticaria, photosensitivity, cardiac arrhythmias, postural hypotension, diabetes mellitus, hyperkalemia, hyperglycemia, glycosuria, hyperuricemia, hypokalemia, hyponatremia, acidosis, hypochloremia, jaundice and/or liver enzyme abnormalities, pancreatitis, nausea and vomiting, diarrhea, constipation, abdominal pain, acute renal failure, interstitial nephritis, renal stones composed primarily of triamterene, elevated BUN and serum creatinine, abnormal urinary sediment, leukopenia, thrombocytopenia and purpura, megaloblastic anemia, muscle cramps, weakness, fatigue, dizziness, headache, dry mouth, impotence, sialadenitis Thiazides alone have been shown to cause the following additional adverse reactions. paresthesias, vertigo, xanthopsia, transient blurred vision, allergic pneumonitis, pulmonary edema, respiratory distress, necrotizing vasculitis, exacerbation of lupus, aplastic anemia, agranulocytosis, hemolytic anemia In neonates and infants: thrombocytopenia and pancreatitis—rarely, in newborns whose mothers have received thiazides during pregnancy*

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MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices. Information must be submitted at least three months prior to the event.*

DECEMBER 1989

AROUND THE STATE

BRONX

Dec 6. **Eyewitness Testimony.** Lehman College. Contact: Mark L. Taff, MD, President, New York Society of Forensic Sciences, 130 Gold Pl, Malverne, NY 11565. Tel: (516) 887-4691.

BUFFALO

Dec 1. **Current Aspects of Treatment of Soft Tissue Sarcoma.** Roswell Park Memorial Institute. Contact: Gayle Bersani, RN, Coordinator of Continuing Education Programs, Education Dept, Roswell Park Memorial Institute, Elm and Carlton Sts, Buffalo, NY 14263. Tel: (716) 845-2339.

MANHATTAN

Dec 1–2. **Genes, Brain and Behavior.** Roosevelt Hotel. Contact: Ivan Bodis-Wollner, MD, Secretary, Treasurer, Association for Research in Nervous and Mental Disease, Inc, Box 1052, Mt Sinai Hospital, 1 Gustave Levy Pl, New York, NY 10029. Tel: (212) 348-8133.

Dec 4–7. **Neurosurgery NYC/1989.** Grand Hyatt. *Dec 6–10.* **Board Review in Geriatric Medicine.** 29 ½ Cat I Credits. Contact: NYU Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

Dec 5. **Management of Patients with High-risk Ventricular Arrhythmias.** 2 Cat I Credits. Helmsley Hotel. Contact: New York Cardiological Society, Inc, 681 Lexington Ave, Suite 310, New York, NY 10022. Tel: (212) 888-5595.

Dec 7–8. **Update in Gastroenterology and Hepatology.** 15 Cat I Credits. College of Physicians and Surgeons. Contact: Columbia University, Continuing Education Center, 630 W 168th St, New York, NY 10032. Tel: (212) 305-3682.

Dec 8. **Clinical Nutrition—Update for the 90s.** 7 Cat I Credits. Grand Hyatt Hotel. Contact: Ann J. Boehme, CMP, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

Dec 9. **Oculoplastic Workshop.** Manhattan Eye, Ear & Throat Hospital. Contact: Francine Leinhardt, Course Coordinator, Manhattan Eye, Ear & Throat Hospital, 210 E 64th St, New York, NY 10021. Tel: (212) 838-9200, ext 1776.

Dec 9–10. **Update in Urological Surgery.** 12 Cat I Credits. NYU Medical Center. Contact: NYU Medical Center, Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

SYRACUSE

Dec 2–3. **40th Annual Postgraduate Review Course in Ophthalmology.** Hotel Syracuse. Contact: Office of Continuing Education, SUNY Health Science Center at Syracuse, 750 E Adams St, Syracuse, NY 13210. Tel: (315) 473-4606.

AROUND THE NATION

ALABAMA

Dec 8–10. **Plastic Surgery of the Nose and Face.** 30 Cat I Credits. Birmingham. Contact: American Academy of Facial Plastic and Reconstructive Surgery, Inc, 1101 Vermont Ave, NW, Suite 404, Washington, DC 20005. Tel: (202) 842-4500.

CALIFORNIA

Dec 8–10. **Advanced Seminars in Diagnostic Imaging.** 20 Cat I Credits. Ritz-Carlton Hotel, Laguna Niguel. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077-1925. Tel: (404) 641-9773.

Dec 9–10. **Stabilization and Manage-**

(*continued on p 16A*)

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COMMENTARIES

The elimination of the requirement for medical libraries in hospitals

It can be accurately stated that the following quotation of Justice Brandeis characterizes the physician perception of governmental intrusion into the practice of medicine: "Experience should teach us to be most on guard to protect liberty when the Government's purposes are beneficent . . . .The greatest dangers to liberty lurk in insidious encroachment by men of zeal, well-meaning but without understanding."[1]

This insidious encroachment without understanding is perhaps most recently exemplified by the decision of the Health Care Financing Administration (HCFA) followed by the New York State Department of Health (DOH) to eliminate the requirement (minimal standard) that hospitals maintain a medical library in order to be eligible for federal funding. The Joint Commission on Accreditation of Healthcare Organizations (JCAHO) has with understanding maintained the standard of hospital medical libraries and considers them relevant to the quality of care.

The reasoning behind the HCFA and DOH decision to eliminate hospital medical libraries is astounding and bizarre. In the *Federal Register* on June 17, 1986, HCFA is quoted as follows: "Medical information impacts on quality of care only if practitioners and staff use it. The mere existence of this information with no parallel requirement for staff to use it gives no assurance that quality of care is enhanced."[2] The DOH concurred. This concurrence is curious in light of the fact that the recently enacted New York State law designed to improve quality of care requires hospitals to provide "Continuing education programs for medical, dental and podiatric staff in their areas of specialty"[3] but does not have a parallel requirement for attendance.

The following are quotations from a DOH spokesman on the issue of hospital medical libraries:

> For many years this Department has operated an active hospital surveillance and enforcement program . . . .Our enforcement process, in all the years we had a medical library requirement in regulation, was never able to link adequacy of medical library services to any of the many specific problems found with delivery of patient care.[4]

In addition, the DOH reasoned:

> From our regulatory perspective . . . we have not been able to determine, for example, that lack of a pertinent collection or article resulted in an instance of poor patient care . . . .It is because of this lack of a demonstrable cause and effect relationship between delivery of acceptable patient care and services and operation of a medical library, that we elected to drop the requirement . . .

And the bottom line reasoning: "our experience has told us [the regulation] was not useful as a regulatory and enforcement tool and which may mandate expenditure of hospital resources to the detriment of more pressing hospital priorities . . . ."[4] It has not escaped suspicion that this is a perverse experiment to obtain data showing that lack of hospital medical libraries does indeed impact adversely on the quality of patient care.

How could this change in the regulations have occurred without an outcry from the medical community? Quite simply—the medical community has no input and must consequently respond after the regulations are set in stone by those health planners who do not understand and offer such baffling alternatives as that suggested to the Medical Library Association. The DOH spokesman advised: "Rather than continuing to petition this Department [DOH] to readopt a section of regulations . . . we have urged you and your members to capitalize on your access to hospital boards, hospital administrators and hospital professional staffs and become partners in hospital solutions to care delivery problems."[4]

Most medical educators would readily concede that medical journals and books are the primary source of continuing medical education (CME) for physicians. No understanding person would deny that CME is an important component of competency nor deny that competency is related to quality of care. With the ever escalating cost of journals and text books, many physicians have cancelled their subscriptions or purchases and are turning to hospital medical libraries, which now are deemed by those who do not understand (or practice) medicine to be unrelated to quality care. The enormous and essential contributions of medical libraries to undergraduate and graduate education are ignored as well as the libraries' resource and reference functions.

This very regressive change in hospital regulations (minimal standards) should arouse a vigorous response from hospital medical staffs throughout the state. The loss of hospital medical libraries will adversely impact on the quality of care, and, in the eyes of those without understanding, the physician will be held responsible.

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1. *Olmstead v US*, 277 US 438, 479 (1928) (Brandeis, J, dissenting).
2. 51 *Fed Reg* 22,033–22,034 (1986).
3. Public Health Law § 2805-j 1.(h).
4. Letter from David H. Philips, Jr, Bureau of Standards Development, NYSDOH, to Tom Flemming, Chair, Upstate New York and Ontario Chapter, Medical Library Association, July 11, 1989.

The role of community hospitals in the detection of occult breast cancer

The experience with occult breast cancer in two community hospitals, presented in this issue of the *Journal*, is noteworthy and deserves attention.[1] As the authors of that report mention, most published reports on this experience come from large-volume screening programs for breast cancer.[2–4] Beginning with the oft-quoted Health Insurance Plan of Greater New York City directed by Dr Strax in 1963, and continuing through the nationwide (NCI-ACS) program involving more than 280,000 women that began in 1973, the detection of occult breast cancer has become commonplace.

Worldwide, programs in the Netherlands, Sweden, Italy, Finland, the United Kingdom, Canada, and the United States have matured and produced substantial, reliable, and encouraging data.[5–7] The world concensus is that mammography detects potentially lethal[8] breast cancer at a stage in which treatment is effective in prolonging disease-free survival. The age groups above 50 years show the clearest effect, but evidence of similarly improved survival for women between 40 years and 50 years is persuasive.[6,7]

Now we are learning of the experiences of community hospitals, at which most cancers are detected, diagnosed, and treated. In the study by Hena et al,[1] the "predictive value" (malignancy rate) of mammographically detected abnormalities requiring biopsy was about one out of five (21.6%). This is the critical figure. It is a measure of program efficiency. By definition, this is the proportion of people who have breast cancer to all persons screened as "positive" for breast cancer. The number of patients with cancer are, of course, those determined by subsequent diagnostic evaluation, ie, biopsy and histologic diagnosis.[9]

In the Breast Cancer Detection Demonstration Project #25 in Columbia, Mo, the predictive value was one cancer detected out of five biopsies the first year (1974). By the fifth and final year (1980), the ratio was one cancer detected in three biopsies (unpublished data). In North America, about one in five is often reported, but in Sweden the rate is said to be one in two.[10] This ratio may be too low—some borderline lesions may be permitted to grow. Watching occult breast cancer grow is, theoretically at least, not desirable.

The critical factors in obtaining acceptable predictive values are quality of the mammogram and the ability of the mammographer to perceive and critically analyze the abnormalities displayed by the image. The American College of Radiology has vigorously addressed the matter through its Mammography Accreditation Program. All diagnostic radiologists who are increasingly involved in mammographic interpretations would derive great benefit from attending one of the mammographic symposia. These are presented by an outstanding faculty chaired by Dr Robert McLlend. It is a splendid educational effort. We anticipate a growing number of accredited hospitals and other facilities.

My hope is that the experience reported in the *Journal* by Hena et al[1] in the two community hospitals in Albany, NY, will be replicated wherever possible. In-depth mammographic screening in this country can then become a reality. It must also be appreciated that responsible screening for breast cancer carries some risks and potential problems.[11]

An open attitude should prevail regarding related matters such as sensitivity and specificity, screening intervals, optimal views, xeromammography vs film mammography, identification of high-risk subgroups, and age limits. The fact remains that mammographic screening is reducing breast cancer mortality worldwide.[4,12]

Further good news, demonstrated in the report by Hena et al,[1] is that such a program can be consistently and reliably implemented in community hospitals.[1] Success depends on the availability of state-of-the-art mammography and close cooperation between radiologist, surgeon, and pathologist. This team approach is reflected in the good results reported by Hena et al.[1]

Finally, this report suggests to me that cooperating hospitals might accomplish in this country what the European Group for Breast Cancer Screening proposes for their area of the world: "to establish contacts between breast screening projects, to encourage the interchange of information, to work for the standardization of terminology, methods of examination and diagnostic criteria and to promote the advance of research in this field."[12]

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The rural health care challenge in New York State

Those living outside of New York State often think all of New York resembles New York City's Fifth Avenue. Those of us who are New Yorkers appreciate the rich rural heritage we have. However, even in our own state, rural areas and issues are underrepresented in the various regulatory agencies and in the minds of many who address the health concerns of New York citizens. In the last two decades the physician/patient ratio in urban areas has approached 1:600, while for rural New York it has remained stubbornly close to 1:1,200. Rural hospitals are struggling to meet the needs of their communities. It seems appropriate to review the status of rural health care in New York State and to suggest strategies that may contribute to the support and enhancement of the rural health care system.

Three million people live in areas of New York State with fewer than 150 persons per square mile. This is the sixth largest rural population in the United States. These rural areas have more elderly citizens below federal poverty thresholds, lower median incomes, and a more culturally homogeneous population than urban areas. Teenage pregnancy and low birth weights are more prevalent. Death rates from accidents are higher in rural areas. Rural areas have shortages of primary care physicians, medical specialists, dentists, and social workers as well as nurses, physical therapists, and occupational therapists. There are few home health care programs and a greater reliance on rural hospitals as institutions for a wide range of health related care. These rural hospitals have a generally shorter length of stay and lower occupancy rate than urban hospitals. They deal with a less complex case mix and lower intensity of care.[1,2] As the sole community provider, the rural hospital is more likely to maintain basic medical services such as emergency rooms, obstetrical units, pediatric units, nurseries, and skilled nursing facilities. Long distances, travel time, and weather problems add to resource accessibility problems.[3] In many cases the rural hospital is the major employer in the county and is more likely to be owned by a municipality.[4] In spite of these extra pressures, the per diem reimbursement of rural hospitals is significantly less than that of urban hospitals of similar size.[5]

Rural physicians see 9% more patients per week and work 6% more hours per week. Waiting time to appointment is 0.2 days longer than in urban practices.[6] Income for rural physicians lags behind their urban counterparts in spite of higher volume and longer hours.

The two key issues of the rural health care challenge in New York State are that the rural hospital is the focus of rural health care and that professional recruitment of medical personnel is an acute problem.

A brief history of government involvement in rural health care is revealing. The Hill-Burton Program of 1949 stimulated the construction of rural hospitals. The Comprehensive Health Planning Act of 1966 encouraged the development of a health planning system throughout the United States. The focus of these two programs in rural areas was on developing primary care services. In 1974, the National Health Planning and Resource Development Act began to change the focus to cost containment, with health systems agencies evolving from the old planning boards. Through the certificate of need process, the health systems agencies and the New York State Department of Health (DOH) regulated the expansion of new technologies. Health systems agencies were successful in modulating unnecessary expansion of new, expensive technology. This resulted in greater reliance on and competition with major urban hospitals. Patients who required the higher technologies and physicians and nurses who used them were lost to the urban centers. This regionalized approach had good intent but rural hospitals had more difficulty recruiting and retaining manpower at the same time that consumers were looking to the hospital to provide expanded services. Almost unwittingly these policies favored large urban institutions and ignored small communities and their institutions. The inflexibility of other regulations also created hardships unrelated to quality for many rural communities and suppressed creativity.[7] As the focus of health care expertise, the rural hospital is bound to a greater role in the community, different from the urban institution, but in competition with it.

Financially healthy, credible, and innovative rural institutions will attract more manpower but the other task of increasing the numbers of physicians, nurses, and ancillary professionals who will consider rural practice must be addressed through the educational system. Several studies on physician manpower define the issues in career location choices. While most graduates who go to rural areas are from rural areas, the actual decision about practice location is made in residency.[8] Economic considerations and spousal area of origin are often more compelling than personal preferences.[9–11] Experience in rural areas during training and a faculty that includes rural practitioners seem to be effective ways to influence career choice.[12–14] It has also been shown that graduates of primary care residencies are more likely to choose rural practice. Twenty-four percent of family practice graduates in New York State have chosen rural practice versus 3% of all other residency graduates (R. Golden, unpublished data, February 1988). There is also evidence that practice location is more dependent on the perceived financial health of local institutions than is generally recognized.[15]

Inertia is another important problem in recruiting to rural areas. A qualified graduate of a residency program is often wooed by local physician groups who become acquainted with the resident during his or her training. Accepting a job offer close to the teaching hospital means no urgency to move a household, the ability to have the children stay in their schools, and fewer difficulties obtaining staff privileges. These are powerful forces for starting practice near the area of training, usually an urban one.

Seven hundred Texas physicians were asked to rank personal and professional variables for selection of practice site. The most important personal variables were: 1) acceptability of site to spouse, 2) quality of education for children, and 3) the overall life style of the area. The important professional variables were: 1) adequate hospital facilities, 2) availability of contact with physician colleagues, and 3) availability of consultants.[16] If these are the issues of rural health recruitment then what strategies might reverse the trend of the last two decades? Again, one should look at rural health care institutions first and review what is being done in New York State.

The New York State Legislative Commission for Rural Resources, created in 1982, has compiled a Rural Health Resource Guide to help in the planning of rural health service systems.[17,18] This commission was established to examine the impact of rural resources upon the state economy, review existing laws and regulations as they pertain to rural resources, assess the effectiveness of existing programs, and make recommendations to the legislature for action to enhance and protect the state's rural resources.

In 1987 a Task Force on Rural Health Strategies was commissioned by the DOH and the Commission for Rural Resources. This task force presented its report in November 1987. Among its recommendations was the establishment of a Rural Health Council to review regulatory and reimbursement policies that impact on rural health care delivery in New York State. This council is charged with developing programs to enhance and coordinate the current delivery system and improve access to medical care within an appropriately regulated environment. The council held its first meeting in July 1988.

The DOH has also extended a request for proposals regarding the establishment of "swing beds" which will allow a rural hospital temporarily to designate some beds as skilled nursing beds when there is a local problem placing patients from the acute care setting into a long term care center.[19] This and other efforts at flexibility will enhance the rural hospital's ability to care for the community.

Rural health networks are a regional way to enhance the rural hospital environment. In 1988, a Western New York Rural Health Cooperative and an Upper Hudson Primary Care Consortium received support from the Robert Wood Johnson Foundation to organize regional networks of hospitals, nursing homes, and other rural providers as well as universities. These networks will initially address quality assurance and physician credentialling issues as well as coordinate hospital bed consolidation and develop strategies for alternative services at institutions which may no longer be viable as acute care general hospitals. Politically, networking potentiates the rural area's leverage with regional referral centers and regulatory agencies.

At the federal level there are also encouraging signs. The Department of Health and Human Services has formed a new Office of Rural Health to serve as an advocate of rural health institutions and hospitals.[20] The US Public Health Service recently issued a request for proposals to establish rural health research centers. Two organizations in New York State have applied for this grant.

Rural communities need to encourage their own youth to consider careers in health care and medical schools need to consider selective admissions of rural students.[21] These students need to have training opportunities in rural areas to reaffirm their intentions[22] and rural communities must consider loan repayment programs[23] plus the provision of moving expenses and incentives to bring a graduate into rural practice. The education system needs to encourage the primary care specialties by selective admissions and role enhancement during training.

There are opportunities in rural areas that universities have inadequately recognized. Faculty practices can be located in rural areas and can have a major role in teaching and research.[24] The rural professors can provide role models and legitimize career goals of students interested in rural practice. The university should also recognize the wealth of research needed and possible in rural communities[25] that includes population demographics, health care delivery problems, and clinical questions.[26,27] The stability of rural communities provides an excellent laboratory for longitudinal studies. Teaching affiliation agreements with rural hospitals enhance both the university and the hospital.[24]

While there is much yet to be done to refine rural health care delivery, the situation is far from hopeless. Rural New York State now has the ear of legislators and regulatory agencies. Further rigorous studies need to be done in the areas of need assessment and solution effectiveness to ensure responsible change. Rural communities must look to themselves for resolution of manpower shortages by grassroots efforts to encourage health careers. Plans must be developed to facilitate the return of graduates to rural communities. Rural New York State has much to offer as a life style. The positives need to be emphasized as we work for the general improvement of health services for our rural citizens in New York State.

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SPECIAL REPORT

New York State's health care system*

Selected major problems and recommended solutions

Many serious substantive problems currently beset the health care system in New York State. Some of these are general in nature while others are quite specific. Only a selected few will be presented for discussion here. Before outlining these selected substantive issues, however, it is appropriate to articulate what we believe to be the core problem which exists between government and all segments of the health care system. This is a problem of process. In the view of the Medical Society of the State of New York (MSSNY), major process changes are essential if we are to resolve the substantive issues now confronting us and those which are certain to arise in future years. In Part I we will attempt to articulate this dimension of the problem and make specific suggestions to resolve it. In Part II we will identify three major substantive issues and set forth our proposals concerning each.

PART I

THE DISINVOLVEMENT OF THE PROVIDER IN THE HEALTH POLICY FORMULATION PROCESS

(a) Problem

It is beyond argument that health policy in New York State during the 1980s has been formulated and implemented without significant input from the provider community. We speak here not only of licensed physicians. Other licensed individual providers, most notably nurses, have been effectively excluded and have publicly and repeatedly decried that fact.

Similarly, the recognized representatives of the institutional segment of our health care system at every level have publicly stated that government in New York has increasingly excluded them from any significant role in the determination of basic health policy objectives. Other nonprovider groups, including consumers, have also had, in our view, an involvement more formalistic than real. Even the legislative branch of government has repeatedly expressed dismay at the extent to which the regulator in this field has preempted, through extremely broad and expansive regulatory initiatives, the policy formulation function normally reserved to elected officials.

Only a short time ago the Administrative Regulations Review Commission, compring senators and assemblypersons, both Democrats and Republicans, openly discussed the possibility of enacting a statutory moratorium on Department of Health regulations as an extreme but necessary response to what was termed a "grossly excessive" regulatory environment.

This phenomenon of noninvolvement has been exacerbated, furthermore, by a clearly adversarial bias which the regulator has assumed toward the regulated. To our knowledge this regulatory attitude is virtually unique to New York State. Within our state, furthermore, it would appear to be unique to the health field.

The results of these dual phenomena of disinvolvement and adversarialism are obvious: Our health care system is not adequately serving the health care needs of our people. This is a fact.

To some extent the problems of our system are the problems of limited resources. We can do relatively little about resources available at the state level.

We are cognizant of the growing aged population and the increasing demands which are made by that population on the system. We are cognizant of the increasing costs of health care that come not from simple inflation but from the inherently complex technologies which everyday are becoming increasingly more sophisticated and, thus, more expensive. We are cognizant of the overutilization problems inherent in the unique way health care is financed in our country and the vast differences between that form of financing and the way Americans purchase virtually all other products and services. We are cognizant of the overutilization problems which derive from the inherent nature of the health system's "product" and our human attitudes toward that product. We are cognizant also of problems in New York State with respect to acquired immunodeficiency syndrome (AIDS) and drug addiction. Obviously, these problems are not caused in any primary sense by the Department of Health.

To a great extent, however, our system's problems are the direct result of grossly inefficient use of those resources which are available. This dimension of the problem is inexcusable. And it is this dimension of the problem about which we can do a great deal.

The extent to which this state is failing to deal with these problems efficiently is a failure for which the Department of Health, as the principal entity formulating health policy in New York State, must be held accountable. Our failure to deal with many of these problems has resulted—to a substantial degree—from the Department of Health's total usurpation of the health policy formulation and implementation functions. This usurpation of control is, therefore, both a principal cause of the system's problems and the justification for holding the Department of Health principally accountable for the system's failures.

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*On August 18, 1989, the leadership of the Medical Society of the State of New York (MSSNY) met with Governor Mario M. Cuomo at the State Capitol in Albany to discuss the problems confronting New York State's health care delivery system. The major problems and recommended solutions were outlined in this document which was presented to the governor. The MSSNY delegation was led by David M. Benford, MD, President; and included Robert A. Mayers, MD, President-elect; Charles D. Sherman, MD, Immediate Past-President; Charles N. Aswad, MD, Vice-President; Stuart I. Orsher, MD, Chairman, MSSNY's State Legislation Committee; Donald F. Foy, Executive Vice-President; Gerard L. Conway, Esq, Director, MSSNY's Division of Governmental Affairs; and Steven B. Weingarten, Esq, Counsel. In addition to Governor Cuomo and several key staff persons, Lieutenant Governor Stan Lundine and Health Commissioner David Axelrod, MD, were also present.

The health system in New York State has traditionally been highly regulated, but recent growth in the dominant role of the Department of Health has been extreme even for our state. Unfortunately, it has directly coincided with and has even been accelerated by the dramatic diminution of the involvement of the federal government in the area of health planning, an involvement which ended entirely in 1986 with the enactment of Public Health Law 93-641.

The federal disinvolvement, of course, reflected the major changes in public policy which have occurred during the decade of the 1980s and which were characterized by a move to deregulation generally. In the health field particularly, the federal government for most of this decade pursued a policy which relied increasingly on traditional market incentives and which sought to moderate cost increases through the basic forces operative in a competitive free market economy.

As a direct consequence of the termination of the federal health planning program, each individual state was given the authority to formulate its own health planning system. Almost all of the individual states reflected the federal policy of deregulation and most states enacted health planning legislation expressly disavowing elaborate regulatory requirements.

This was not the case in New York State, however, where we proceeded in precisely the opposite direction. Although for over a generation New York had clearly been regarded as one of the nation's most regulated systems, even more bureaucratic controls were added on.

Whatever the specific objective of any given regulation, the general purpose and the overall effect of the aggregate have been clear—more and more centralization of power. Department of Health control of the institutional segment of our system is absolute and includes everything from the certificate of need process through reimbursement and the newly initiated 405 regulations.

Acquisition of similar complete domination of the physician sector is the obvious objective of a whole host of Department of Health proposals. Office of Professional Medical Conduct expansions and the aggressive efforts to exclude the Board of Regents from the professional conduct process; proposals relative to graduate medical education funding; hospital medical staff controls; incident reporting and the newly initiated hospital surveillance process; prescription writing regulations which are principally directed not to quality monitoring but to the alteration of clinical practice; physician recertification; proposals to authorize regulatory alteration of statutorily established scopes of practice of the allied health professions; and initiatives which would arrogate to the Department of Health powers traditionally vested in the Insurance and Social Services Department—all these are cause for concern.

The direct results of such initiatives are obvious:

- A grossly inadequate and misshapen institutional system which is teetering on the verge of total financial collapse;
- Shortages in almost every area of allied health personnel, most particularly in the nursing area;
- A system that has largely abandoned the poor and the uninsured;
- Businesses that threaten to leave our state because health care is too costly and its quality is increasingly questionable.

In summary, our problem is simply stated: *We have a health system once preeminent in the nation and the world which is collapsing.*

(b) Solution

Two years ago the legislature, through the Council on Health Care Financing (CHCF), began a thorough evaluation of our state health planning system. Its report was transmitted to Governor Mario M. Cuomo, Senator Ralph Marino, and Assemblyman Mel Miller in February of this year. It contains a careful documentation of serious process problems and presents a compelling argument for fundamental change in the existing system.

The views of organized medicine were presented to the CHCF and we were generally pleased at the extent to which our views were reflected in the comments of most other segments of the system and are themselves reflected in the CHCF recommendations. For our present purposes we wish to comment only on the state planning structure as it was addressed by the CHCF rather than regional planning agencies.

We hasten to point out, however, our view that the development of regional planning along the general lines set forth in the CHCF's recommendations is appropriate and necessary and should be pursued. In this regard we are cognizant that earlier this year the governor signed Chapter 554 relative to the composition and responsibilities of regional health system agencies, a bill which we believe to be consistent in its general purpose with our recommendations.

We would urge consideration of legislation which would place into effect a structure with the following characteristics which we believe to be appropriate and necessary to an efficient health planning process at the state level:

1. A single body (proposed council) should be established and charged with all aspects of state health planning and system development (Fig 1).
2. The proposed council would have a large representational membership including both institutional and individual providers as well as insurance carriers, business, labor, and consumer representatives.
3. Members representing a particular interest would be nominated by the established organizational representative of that particular group (eg, nurses would be nominated by the New York State Nurses Association, hospital representatives nominated by

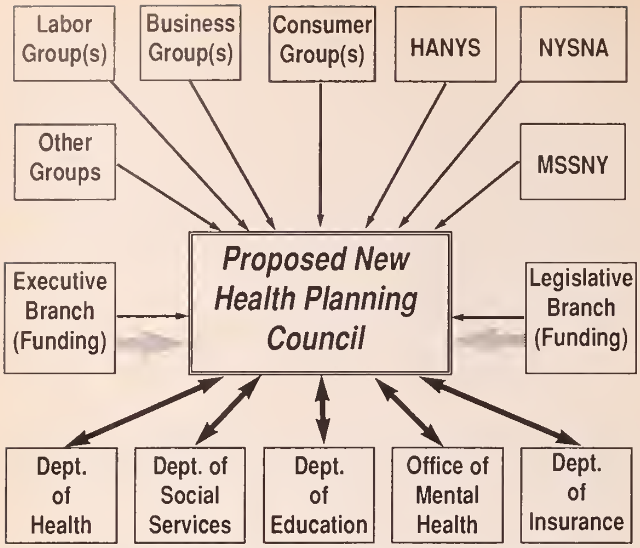

Figure 1. Proposed new Health Planning Council. Single-headed arrows show voting representation only. Double-headed arrows show representation and oversight. Thick gray arrows show separate direct funding.

the Hospital Association of New York State, physicians nominated by the Medical Society of the State of New York, etc). This extremely important provision will serve as the cornerstone of meaningful broad-based participation.

4. The proposed council would be provided adequate *independent professional staff* including a director or executive secretary, and most importantly, be funded separately from the Department of Health. It would be granted immediate access to all existing Department of Health data and data generating capability.

The proposed council would have the principal responsibility for developing a state health plan as well as coordinating approved regional health plans developed by regional planning agencies. All regulations proposed by the Department of Health including the State Hospital Code, regulations relative to fiscal and reimbursement policies, regulations dealing with the certificate of need (CON) process, quality assurance, incident reporting, professional conduct of individual health care providers, issues involving the continuing education and periodic certification of all providers, and the permissible functions of each class of individual providers would be among the areas of proposed council responsibility.

Overall, the proposed council would have the ultimate responsibility to monitor and assure the coordination of policy and regulation of health matters including coordination between all relevant state agencies—most notably the Department of Health, the Department of Social Services, the Office of Mental Health, the Education Department, and the health insurance and professional liability insurance operations of the Insurance Department.

We recognize, as did the Council on Health Care Financing, the far-reaching nature of these proposals in regard to process change. We are convinced, however, that the dimensions of the problems as they now exist compel such fundamental change.

PART II

MAJOR SUBSTANTIVE HEALTH CARE POLICY ISSUES

Partly as a result of the process inadequacies addressed in Part I and partly because of difficulties inherent in the issues themselves, New York State is currently experiencing many specific substantive problems in its health care system. Although this is not the time to discuss in detail each of these problems, we must acknowledge them as appropriate issues to be addressed in future meetings between the governor's office and all appropriately involved groups. In our view, important examples of these problems include, not necessarily in order of priority, the following:

- **Access problems.** We perceive these to be primarily financial problems in terms of their causation, particularly with respect to the uninsured and the poor. These are especially costly in their consequences in the specific areas of prenatal and pediatric care as well as the general areas of primary and preventive care;
- **Long term care and the needs of the aging.** Also a cost problem but it also includes serious access difficulties which are caused by structural inadequacies in the system;
- **Serious system structure problems for the mentally ill.** This problem has already impacted not only on the quality of care given in this area, but also on the problem of the homeless, especially in New York City;
- **Serious health manpower problems.** These occur particularly in the area of nursing services but soon will occur—and occur suddenly—in the other areas, including the area of physician services, where we are already seeing fewer and fewer medical school applicants as well as applicants with significantly lower levels of academic achievement;
- **Increasingly serious problems in filling available residencies** with top flight graduates;
- **Medical liability premiums** which remain at the very highest national levels;
- **Serious rural health care problems;**
- **Growing gaps** between limited resources and advanced technology and between system services and consumer demand;
- **Misallocation of limited resources.** This problem can only be addressed after a thorough analysis of system resources;
- **A negatively oriented hospital survey process.** This seems to be based on excessively rigid practice pattern evaluation protocols rather than on flexible, positively oriented quality assurance objectives and processes;
- **Overly aggressive and often misdirected stop-gap cost containment measures.** These are especially troublesome because they are often initiated without full coordination between and among all aspects of the delivery system;
- **Acute system stresses** caused by the problems of AIDS and drug abuse.

This is not a complete list of the many substantive problems in New York State. It is, however, a list useful as the basis for developing agendas for future in-depth discussions. The MSSNY would welcome the opportunity to work with government representatives in formulating an appropriate plan pursuant to which such in-depth discussions can be undertaken in a fully coordinated manner.

In fact, such discussions would bring us again to that position we feel we must have in order to serve all New Yorkers—that of an active and equal partner.

THREE OPPORTUNITIES FOR IMMEDIATE COOPERATION

There are three substantive matters that so affect the current medical climate in New York State that we must take this opportunity to discuss them, albeit briefly.

*UNY*Care*

We raise as our first substantive issue the Department of Health's UNY*Care proposal which, while principally addressed to New York State's uninsured population, would radically restructure the health insurance system in New York State. Although here raised as a substantive issue of overwhelming importance, we are compelled to make two threshold observations which we believe demonstrate the general problem of process addressed in Part I of this document.

First, recognizing that the existing written document describes itself as "Draft and Confidential," it nevertheless shocks us that such an initiative progressed even to that point without any input whatsoever from organized medicine. We are informed also that neither assistance nor advice was sought from any of the other major provider groups.

Second, the "Executive Summary" as originally drawn reflects a vision of New York State's health care system which can only be a product of a public relations mindset. Certainly it is not a realistic evaluation of the current health care system in New York. Thus, over and over again we find gratuitous, self-congratulatory references to New York State as "a leader in health policy"; to "our evident achievements"; to our "commitment to the poor" (who, in our opinion, do not presently have reasonable access to quality health care); to the assertion that the state's Medicaid program in the mid-1970s was "the most generous Medicaid program in the nation" (certainly not with respect to physician services, where New York State ranks at almost the very bottom in the nation in terms of physician payments, a fact which effectively denies the system's intended beneficiaries efficient access to physician care).

To initiate a major health system analysis with such a positive assessment of the current state of the system when, in fact, that system is in very real jeopardy casts the credibility of the entire document into immediate and serious question.

Moving to the document's substance, we believe that certain of the provisions contained in the proposal are laudable, at least as stated objectives. These include:

- expansion of public programs to cover children from birth to age 17 and all persons under 100% of poverty, as well as providing subsidies for persons between 100% and 200% of poverty;
- a basic benefits package paralleling the Medicare program which would eliminate current health insurance coverage gaps and meet the needs of the general population;
- primary care coverage encompassing prevention and maintenance health care services;
- coverage of the $540 Medicare Part A deductible for beneficiaries below 200% of poverty;
- the availability of long term care coverage;
- the efficiency of a uniform "one-payor" reimbursement system for all medical services.

These are worthwhile objectives and, subject to review of program specifics, we would welcome the opportunity of working together with government in the implementation process.

This, of course, is not to suggest that the proposal as we now understand it is not without areas of possible controversy. For example, while a uniform "one-payor" reimbursement structure has great appeal in terms of efficiency, the specific reimbursement negotiation process must be carefully constructed in order to guard against excesses which could drive away business and government support, and/or inadequacies which would seriously discourage provider involvement.

Perhaps most importantly, the state will be the sole actor, playing a larger role in controlling health care delivery in New York State. This, of course, raises all the concerns referred to earlier relative to the current "process" inequities. The state, for example, will develop a budget for UNY*Care and determine revenue needs, as well as manage program funding and expenditures. Frankly, our experience in the area of Medicaid seems to us to show that the State of New York has not done very well in this area.

A more complete analysis of the draft UNY*Care proposal was prepared by the MSSNY's Division of Socio-Medical Economics and published in the October 1989 issue of the *New York State Journal of Medicine.* Public hearings on this proposal are expected to be held around the state over the next few months. In regard to these hearings, the information which we have received, admittedly unofficial, indicates that a committee to hear this matter already has been appointed. The MSSNY regrets that no representative of organized medicine or any other provider group has been asked to serve on the committee.

UNY*Care or any other major health care insurance or funding expansion must be preceded by a careful examination of the system to be funded. Careful choices concerning the services to be funded must be considered and made lest we simply perpetuate existing system inefficiencies and skewed priorities. Preferential funding of institutional care in lieu of less expensive alternative care which is clinically acceptable is fiscally irresponsible. We all know that, more and more, health insurance coverage determines the shape of the clinical system.

We must not just articulate this phenomenon. We must design a program responsive to it, recognizing that dramatic system restructuring may result from such a process of insurance benefit reevaluation.

The medical community and all other providers must be extensively involved in the shaping of the benefit program, a process which can be rational and prudent only when pursued against a standard of defined available resources.

The MSSNY respectfully suggests that the expertise of organized medicine is essential to the formulation of an initative of this magnitude. We must be fully involved in the process. An opportunity to speak for 20 minutes at a public hearing to such a complex initiative is not meaningful input.

Despite our acute disappointment at not having been involved to this point, we nevertheless stand ready to work with New York State government in pursuing this objective.

Medicaid

In 1987 the respected Public Citizen Health Research Group issued a report entitled "Poor Health for Poor Americans: A Ranking of State Medicaid Programs." That report contained the following statement: *"New York fails its needy population primarily by paying physicians at such absurdly low rates that few will treat Medicaid patients"* [emphasis supplied].

This administration has put forward several laudable initiatives involving both expanded service coverages and eligibility levels. With the single exception of obstetrical care, however, there have been no significant initiatives to correct the abysmal provider payment levels to physicians. A variety of demonstration initiatives (particularly in the area of what has come to be called "managed care") are proving less than successful. We will continue to work to make these programs viable, but we all must accept the basic fact that Medicaid represents a clear example of misdirected resources. The program's priorities, evident in and determined by its reimbursement structure, guarantee its failure.

The Medical Society of the State of New York applauds our governor's efforts to bring needed medical care within the reach of greater numbers of needy New York State residents, particularly our children. We are concerned, however, that the system of medical care which awaits those potential new Medicaid patients is one that is far less desirable than that generally available to the rest of the population of New York State.

The chief reason for this, furthermore, is the lack of access which significant portions of the Medicaid population have to the services of office-based physicians. This situation exists despite conclusive evidence that 1) services in that setting are desired by large numbers of Medicaid patients; and 2) that overall Medicaid costs in that setting are lower than the cost of ambulatory care from institutional providers.

The Medical Society of the State of New York strongly believes that it is in the collective interest of the State of New York and its practicing physicians to form a partnership aimed at enhancing access to care for Medicaid recipients. Such a partnership would not only result in more accessible care, but also in higher quality and more cost-effective care for the Medicaid population. It would also ease the crunch on hospital emergency rooms to a large extent.

The Medical Society of the State of New York recommends, as an initial step, the establishment of a state commission to examine issues related to physician participation in the Medicaid program. This commission should make recommendations to the governor and the legislature by April 1, 1990, regarding statutory and regulatory actions which should be taken to assure that there is sufficient participation to meet the health care needs of the state's Medicaid population.

The commission's membership, to be appointed by the governor and the legislative leaders, should include representatives from the executive and legislative branches of state government; practicing physicians nominated by organized medicine; local social services district representatives; Medicaid recipients; and other interested parties such as advocacy group representatives. Like the new Health Planning Council suggested earlier, this commission's staff should function independently and be responsible directly to the commission's membership.

Quality Assurance

"Quality assurance" as an appropriate and necessary objective is an inarguable tenet of any health system. Any delivery system must have quality control mechanisms as essential structural components. The number and variety of quality control mechanisms in place in the field of health care are unparalleled in any other profession.

This is how it should be. We wish to state clearly and unequivocally that the Medical Society of the State of New York and the physicians of New York are committed to the objective of assuring medical care of the highest quality to the greatest extent humanly possible. Our purpose in raising the quality assurance issue at this time is to bring to light our serious concerns over inappropriate utilization of the quality assurance goal and the mechanisms put into place purportedly to achieve that goal.

For more than a decade the New York State Department of Health has utilized the extremely serious medical liability situation in New York State to assume an ever increasing role in the quality assurance process. This increase of quality assurance regulations is linked to liability bills even though evidence has proven that there is only a tenuous connection between malpractice claims and poor quality care. Nevertheless, the various medical liability enactments over the last few years have inevitably contained sections granting to the Department of Health more and more regulatory control and power, allegedly to enhance the pursuit of the general goal of quality assurance.

Initiatives undertaken pursuant to these new powers, however, have in our judgment often been directed toward objectives unrelated to legitimate quality enhancements. Most distressingly, they have sometimes served as vehicles through which questionable clinical judgments have been imposed on practitioners, which results in *forced* medical practice alternatives unrelated or even contrary to quality enhancement. Secondly, the quality assurance processes heretofore established by the Department of Health have been characterized by a focus on the identification and punishment of disquality rather than on the implementation of procedural incentives directed to service quality enhancement.

The surveillance procedures which have been implemented in the new hospital survey process were developed once again with little or no provider input. As implemented, they are neither fair nor are they likely to improve the quality of the clinical care provided to patients. As a matter of fact, because of substantial resource diversion, they more often than not represent cumbersome, time consuming intrusions into the care-giving process and as conducted they detract from, rather than enhance, that process.

We have repeatedly attempted to point out the surveillance process deficiencies. Notice problems, staff diversion problems, lack of communication between the reviewers and the person being reviewed, inadequate exit interviews, and the negative bias of the reviewer are all aspects of the process that have extremely deleterious effects on the physician-patient relationship and on public confidence in the system. This is particularly true in view of the wide publicity which the Department of Health has seen fit to give to the surveillance process and its outcomes. All of these deficiencies far outweigh any benefits reasonably to be expected.

We are deeply concerned, therefore, that a whole host of purported quality assurance mechanisms, from triplicate prescriptions to the new survey process, are being seriously misutilized and misdirected to serve entirely different purposes.

Consequently, it is not surprising that we react as a profession with great trepidation when we hear the Department of Health announce initiatives in such areas as graduate medical education, continuing medical education, relicensure, and the development of practice parameters. We do not dispute the need to work to secure improvements in these areas. We are deeply concerned, however, that this work is not being done in the manner necessary to achieve legitimate and appropriate results.

Once again, the solution to the problem is two-fold:

- There must be a diminution in the adversarial attitude of the regulator as it pursues agreed upon objectives in these areas; and
- There must be meaningful involvement of the profession in the quality assurance structure.

Absent both of these elements, the current Health Department programs aimed at quality assurance will not achieve their basic purposes.

CONCLUSION

The MSSNY has attempted in this document to identify some of the major problems which exist in our current New York health care delivery system. We have attempted also to offer for consideration possible solutions to these problems. One thing, we believe, is clear—we must cooperate.

The MSSNY and the state government have worked together in the past. During the session just completed, we cooperated in several major gubernatorial initiatives. These included the governor's prenatal care legislation and what is clearly the most significant antismoking law in the United States. In years gone by, extensive cooperation between the MSSNY and the administration scored major legislative successes, including the first mandatory seat belt law in the United States. We believe that our involvement helped in achieving these goals. We have worked with government in many other public health areas.

Only by working with each other and with all other involved interests can we achieve what must be our shared objective—a health care system of which New York can be proud and to which all of America can look for guidance.

Occult breast cancer in two community hospitals in New York State

MUHAMMAD A. HENA, MD; ANDREW C. WARHIET, MD; JOHN C. CROWTHER, MD

ABSTRACT. The histopathologic results of 360 consecutive preoperative needle localizations of nonpalpable breast lesions performed at two community hospitals in New York State were analyzed for comparison with national data. Seventy-eight (21.6%) samples were malignant lesions. Sixty of the 78 (76.4%) were infiltrative lesions, and 18 (23.6%) were in situ carcinomas. Overall, axillary lymph node metastases were noted in five (8.3%) samples. We conclude that needle localization permits detection of occult carcinoma at an early stage and that detection of occult carcinoma in patients seen in a community hospital corresponds well with published national data.

(NY State J Med 1989; 89:612–613)

The routine use of screening mammography in women to detect early breast cancer has increased over the past two decades. This approach detects carcinoma at an early stage and has led to better five- and ten-year survival rates.[1] Consequently, screening mammography in women has been widely accepted and is now being frequently used. More nonpalpable lesions are now being detected and removed. Needle placement in the area of concern followed by excision and confirmation by radiographic studies permits preservation of the breast architecture.[2] The sensitivity of mammography in detecting cancer in the asymptomatic patient is reported to range from 81% to 96%.[3–5] The malignancy rate in occult lesions has been reported to be between 10% and 25%, with axillary metastasis in 6–17%.[6–11] We undertook a study of preoperative needle localization of breast lesions at two New York State community hospitals to determine breast cancer detection rates with this technique.

METHODS

According to the American Cancer Society guidelines for breast cancer screening, women with suspicious nonpalpable breast lesions should undergo a needle localization procedure. A consecutive series of 360 such procedures were performed from July 1985 through July 1987, in two community hospitals in New York State (St Peter's Hospital and Albany Memorial Hospital, Albany, NY). The ages of these patients ranged from 35 years to 82 years. The lesions were determined by radiologists to be potentially malignant and thus required biopsy and histologic study. Radiographic findings varied including microcalcifications, ill-defined masses, and distinct masses. None of the lesions were clinically palpable by the operating surgeon. The localizations were carried out by a radiologist immediately prior to surgical excision. Radiographic confirmation of the specimen was obtained prior to pathologic evaluation. Radiographically unconfirmed specimens were excluded from the study.

RESULTS

Histopathologic interpretation revealed 78 (21.6%) lesions as being malignant and 282 (78.4%) as benign (Table I). Of the malignant group 60 (76.4%) were infiltrative carcinomas and 18 (23.6%) were carcinomas in situ (Table II). From July 1985 through July 1987 at St Peter's Hospital, 261 such procedures were performed. Fifty-five (21.1%) revealed malignancy, and 206 (78.9%) were benign. Forty-five (81.9%) were infiltrative, and ten (18.1%) were in situ carcinomas. At Albany Memorial Hospital, 99 procedures were performed during the same period. Twenty-three (23.3%) revealed malignancy, and 76 (76.7%) were reported as benign. Fifteen (65.3%) were infiltrative, and eight (34.7%) were carcinomas in situ (Tables I, II). All 60 patients with invasive carcinomas underwent either lymph node sampling or lymph node dissection. Five (8.3%) were found to have lymph node metastases. All the patients who did not undergo mastectomy or lumpectomy had follow-up mammography to assure capture of the target.

DISCUSSION

It is estimated that in the United States in 1989 there will have been 142,000 new cases of breast cancer detected and 43,300 deaths attributed to the disease.[12] Breast cancer is the second leading cause of death in females, and a majority of these breast cancers will be diagnosed and treated in community hospitals. To our knowledge, the results of the detection of breast cancer in community hospitals in the United States has not been adequately analyzed and reported. We analyzed our data to determine how community hospitals would compare with the national average and found them to be within comparable range.

Screening mammography can reveal the presence of occult breast lesions; usually masses or microcalcification

TABLE I. Benign and Malignant Occult Breast Lesions Found at Two Community Hospitals

Institution	Total	Benign	Malignant
St Peter's Hospital	261	206 (78.9%)	55 (21.1%)
Albany Memorial Hospital	99	76 (76.7%)	23 (23.3%)
Total	360	282 (78.4%)	78 (21.6%)

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TABLE II. Invasive and In Situ Occult Breast Cancer at Two Community Hospitals

Institution	Malignant	In situ	Invasive
St Peter's Hospital	55	10 (18.1%)	45 (81.9%)
Albany Memorial Hospital	23	8 (34.7%)	15 (65.3%)
Total	78	18 (23.6%)	60 (76.4%)

suggest the presence of cancer. Thus, the role of early detection by mammography has been emphasized.[7] In nonpalpable mammographic lesions it may not be possible to obtain a diagnosis by needle biopsy. Consequently, needle localization followed by excisional biopsy has been the acceptable diagnostic procedure for such lesions. Published reports have documented a 10–25% yield of occult malignancies in nonpalpable lesions, with nodal metastasis of 6–17%, as compared to 25% lymph node metastasis of lesions less than 1 cm in diameter.[6,14] In the series presented here, 91.7% of the malignancies were stage I. The prognosis for these patients should be excellent.

The treatment of breast cancer, however, remains controversial. More patients with stage I disease are being encountered. Moderate improvement in the long term survival (83.7% ten-year survival) of breast cancer patients has occurred during the past three decades, particularly with the recent increase in use of and patient compliance with screening mammography.[14]

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FROM THE LIBRARY

WHAT EVERY MEMBER OF THE MEDICAL PROFESSION SHOULD KNOW ABOUT PROTECTING WOMEN FROM DEATH FROM CANCER OF THE BREAST

It does not make any difference what your specialty is in medicine, or whether you are a general surgeon or general practitioner, or even whether you have retired from the medical profession, you still have an opportunity to help reduce the mortality of cancer no matter where it is first situated.

Those most familiar with cancer today agree that there is no treatment for malignant disease except some form of surgery the object of which is complete removal, or irradiation with x-rays or radium. There is also universal agreement that these therapeutic agencies accomplish the largest number of temporary and permanent cures the sooner they are applied after the first symptom.

Prolonged experience justifies me in making this statement to every lay and medical audience which is not yet fully appreciated by either. The prevention and cure of cancer today depend upon the view that in the beginning cancer is a local disease, and at first the cancer cells in that spot are not malignant, but simply abnormal cells changed by injury or chronic irritation of some character from the pre-existing normal cells in the spot. It is true that in some forms of malignant disease there are in the beginning multiple areas. These types are hopeless unless temporarily arrested and, in a very few instances, cured by irradiation with x-rays or radium. . . .

The essential features . . . in the attempt to reduce the deaths from cancer of the breast in women are: The recommendation, through education, of annual and semiannual examinations by a competent physician selected while well; the education of the general practitioner on the proper physical examination of the breast, so that he may select those patients who should be referred to a consultant with the least risk to those whom they diagnose themselves as belonging to a group of benign conditions for which operation is not necessary; the training of the surgeon to meet the new demands in the search for the lump that should be explored; and the training of that surgeon to maintain the technique of the complete operation for cancer successfully established more than thirty years ago; the training of the pathologist in the more certain differentiation between the benign and malignant breast tumors in the fresh frozen section made in the operating room. . . .

JOSEPH COLT BLOODGOOD, MD
(*NY State J Med* 1932; 32:259-264)

REVIEW ARTICLE

Vascular insufficiency: A significant factor in impotence

ADRIAN W. ZORGNIOTTI, MD

The ancient Greeks thought that air (pneuma) flowed into the penis to produce erection. The air explanation came about since the penis, like the arteries in the cadaver, was invariably found to be devoid of blood on dissection. Since air is less dense than blood, the rapidity with which erection could come about in man made this a logical explanation. In the 17th century, Regnier de Graaf demonstrated erection in the cadaver by injecting water into the hypogastric artery with a syringe of his invention.[1] In the 1920s, Leriche observed a relation between obstruction of the aortic bifurcation and impotence.[2] However, the cause of impotence, up until this time, was generally thought to be a sequela of gonorrheal infection, posterior urethritis, or masturbation earlier in life.[3] After World War I, an explanation for impotence based on the Freudian theory of Oedipal conflict became firmly embedded in the view of physicians and the public, so that most impotence came to be attributed to psychogenic causes.[3] In the mid-1970s Jean-François Ginestié, a French radiologist, and Vaclav Michal, a Czechoslovak vascular surgeon, independently performed selective pudendal arteriography demonstrating obstructive arterial disease in impotent patients.[4,5] This new information led to a further step when Michal performed the first inferior epigastric artery to corpus cavernosum microsurgical bypass.[6] We know that erection is a neurovascular/hormonal phenomenon which requires a rapid and large inflow of arterial blood and concomitant restriction of the venous outflow by the erectile body. Both are dependent on smooth muscle relaxation. When there is impotence, this vascular component is always involved, regardless of what etiology has been assigned by the clinician. In other words, even if impotence is diagnosed as psychogenic, the net effect is that blood flow to the penis is lacking. Understanding of this has given rise to new research into the physiology of erection and new ways to treat impotence which increase blood flow to the erectile bodies.

Patients who were reluctant to ask for help a decade ago are now telling their doctors about inability to have an adequately stiff erection. Patients relate that often the practitioner's response is to shrug off the matter with allusions to the inexorable advance in the aging process. This reaction is puzzling and may be explained by lack of knowledge or perhaps the practitioner's feeling threatened by what is perceived to be a quality of life rather than a medical issue. The patient is often administered an androgen in the mistaken belief that this restores virility. Androgen administration can produce an improved sense of well being and increased libido but very rarely any change in ability to penetrate a partner, unless there is severe hypogonadism and the patient is relatively young. Impotent older men have serum testosterone levels at or below the laboratory limits of normal. Hypogonadism is thought to be a factor in ischemic heart disease, and there may be a parallel in impotence.[7]

PREVENTION

The cavernous arteries, which aliment the corpora cavernosa, resemble the coronary arteries in that these are end arteries without collateral circulation. It is not surprising that epidemiologic studies have identified the same risk factors for impotence and ischemic heart disease: aging, hypertension, diabetes, smoking, and hyperlipidemia.[8] Efforts at preventing ischemic heart disease could produce a parallel decrease in impotence. In my experience, more than 70% of impotent patients either have or had been smoking heavily—20 to 60 cigarettes a day for decades.

ERECTILE FAILURE

Impotence is the inability to perform or complete the sex act, due to failure to achieve or maintain erection. Men who experience occasional performance difficulties or who can have coitus with one partner and not another do not have organic impotence and should be investigated for psychogenic impotence. The practitioner should consider that organic and psychogenic factors can and do coexist.

The list of diseases and medications implicated in impotence is long, so that evaluation to pinpoint a cause is important. Elimination of a disease or discontinuance of an implicated drug rarely results in restoration of potency, as, for example, when diabetes is brought under control, smoking is stopped, or an antihypertensive drug is either discontinued or substituted. Impotence can result from trauma, frequently to the pelvis and perineum. Occasionally, a congenital vascular anomaly can be uncovered. While the underlying disease may not be eliminated, patients can reach a point at which, on demand, vaginal penetration is a reality using techniques that alter existing penile hemodynamics. In general, oral medications, hormones, acupuncture, and psychotherapy give disappointing results.

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VASCULAR DIAGNOSTIC METHODS

A careful history and physical examination are essential. A striking observation is the change seen when there is loss of corpus cavernosum substance resulting in "floppiness" of the penis. This myopathy is associated with microvascular disease of the corporal smooth muscle.

Penile systolic blood pressure determination has limited diagnostic value. This reading is compared to the brachial blood pressure (penile brachial index), and is obtained by auscultation of superficial penile arteries and not the cavernous arteries, which are those mainly involved in erection. It is possible to have a normal penile brachial index (PBI) with severe cavernous artery disease. The practitioner should be aware that only low PBI has diagnostic significance for vascular insufficiency.

Penile air cuff plethysmography is an inexpensive and valuable method for diagnosis. While results are qualitative, decreased wave amplitude and absence of a dicrotic notch on the descending limb of the tracing suggest vascular impotence.

Duplex ultrasound scanning, in conjunction with the intracorporal injection of a vasoactive substance such as papaverine HC1, permits visualization of the penile arteries and determination of blood velocity in them. Unlike penile blood pressure studies, important quantitative information concerning the cavernous arteries is obtained. Invasive selective pudendal arteriography remains the "gold standard" for the delineation of disease of the internal pudendal and penile arteries. These two methods are central to establishing the diagnosis of vascular insufficiency.

TREATMENT: RESTORATION OF THE ABILITY TO PENETRATE

Penile Implants. For many years this was the only method for assuring reliable sexual penetration. Prostheses do not give erection, nor do they address the cause of impotence, but stiffen the penis for coitus. The advantage is a high degree of functional success, 95% or better. This is a permanent, surgical implant performed in the hospital; it is entirely within the body and not externally visible, although it may be detected by touch. These can be called on anytime and as often as the patient desires. There are two types: malleable rods implanted within the corpora cavernosa whose presence can be concealed by clothing, and mechanical prostheses which simulate erection but also give the penis a normally flaccid appearance when not in use. Being mechanical, these can be subject to device failure, which requires corrective surgery. The malleable prosthesis is preferable as it is more trouble-free than the mechanical type, although improved technologies have decreased the failures of these considerably. Guided by the surgeon, the patient participates in the selection of the prosthesis.

Implants have gained acceptance by urologists and the public. There can be problems, which occur very infrequently. Infection in spite of intensive antibiotic treatment could necessitate removal. Patients allergic to antibiotics are subject to additional risk. Unpredictable aching pain in the penis may result in a request for removal. Erosion of the device, usually through the distal urethra, will necessitate removal. Implantation is thought to destroy the erectile tissue in the corpus cavernosum so that other forms of treatment are no longer possible, yet some patients will report partial erection during coitus using an implant. Implanted penis size and length often fall short of expectation. Those whose non-erect penis is small should be aware of this potential problem, which is difficult to predict beforehand. The patient with a long non-erect penis can hope for a more satisfactory result. The glans is minimally stiffened by the implant and can be annoyingly "floppy," interfering with penetration.

In spite of potential but infrequent problems, implants deserve serious consideration when making a treatment decision. Men who are unable to stop smoking should consider an implant in preference to bypass microsurgery and pharmacologic erection, since the latter depend on maintenance of optimum blood supply, and continued smoking will cause further deterioration. Implants are an excellent backup if the alternatives prove unsuccessful or are unacceptable to the patient. There are regional differences in implantation practice; per capita implant sales are seen to increase as one moves from the East Coast to the West, with the largest sales in Texas and California.

INCREASING BLOOD FLOW TO THE PENIS

Bypass Microsurgery. In carefully selected patients, bypass microsurgery can yield satisfactory results in terms of spontaneous erectile function. Michal dissected the inferior epigastric artery with microsurgical anastomosis, via an inguinal tunnel, directly to the corpus cavernosum. This resulted in restoration of erection, but these bypasses soon became occluded and erection ceased. Another problem was the occurrence of priapism in the postoperative period. This led to modification with microsurgical arterial or venous graft interpositions to the dorsal and central penile arteries. Another approach that improves on the original Michal operation directs the feeder artery's blood flow via the deep dorsal vein of the penis and corporal perforating veins into the corpus cavernosum.

Prior to bypass surgery, an invasive procedure, selective pudendal arteriography, is necessary to identify the site of arterial obstruction and permit planning of the bypass. This examination is restricted to patients in whom there is a reasonable possibility that the operation will be performed, and who have agreed to stop smoking. The surgery requires ten hours to complete, but this can be reduced to about six hours by using two surgical teams. Because of the necessity of prolonged anesthesia and the possibility of poor results, bypass is not done in the aged or those with heart disease, diabetes, hypertension, prior extensive abdominal surgery, poor anesthesia risk, and inability to stop smoking. Young men who have never had adequate erections (primary impotence) or who become impotent from trauma are particularly suitable for bypass surgery and should not undergo implantation.

When successful, bypass leads to a return to spontaneous function. Doubts about the efficacy of bypass microsurgery have been expressed by Sharlip,[9] who reported on a personal series of 27 cases with five long term successes (19%). Goldstein et al[10] reported similar results in his early cases, but with improved technique, his long term satisfactory results were 74% in a second series. Similarly, Lizza and Zorgniotti[11] reported 78% short term ability to penetrate a partner. It would seem that with improved surgical technique, reasonable levels of patient satisfaction can be achieved.

CAVERNO-VENOUS LEAKS

While much impotence is due to vascular inflow impairment, failure of the corpus cavernosum outflow restriction mechanism, often referred to as venous leakage, does occur. The mechanism of this entity is not understood, but there is evidence that inability to retain blood by the corpus is due to failure of the sinuses in the corpus to compress venous outflow when smooth muscle relaxation takes place. Such leaks can be congenital or acquired. Dynamic cavernosometry (intracavernous pressure/flow studies with intracavernous papaverine) serves to identify abnormally high flow rates needed to attain artificial erection with intracavernous continuous saline pumping. Dynamic cavernosography demonstrates abnormal filling of the extracorporal penile veins by contrast medium in the presence of papaverine stimulation. When found, often in younger patients with soft erections, surgical correction by excision of the deep dorsal vein of the penis in conjunction with ligation of the crural veins produces good results. Plication of the crura, which is less difficult, is also being done. Long term results are not yet known. Caverno-venous leakage, if not too severe, can respond to intracavernous injections of vasoactive agents.

INTRACAVERNOUS PHARMACOLOGIC ERECTION PROGRAMS (PEP)

A nearly painless injection is made into the corpus cavernosum at the base of the penis which gives a temporary increase in blood flow lasting two to three hours. This can be sufficient to permit erection if there is also sexual arousal. Intercourse varying from excellent to satisfactory has been reported by 78% of patients who can then be trained to self-inject when they desire. The medications currently in use are papaverine HC1 with phentolamine mesylate or prostaglandin E_1.

Initial testing of this treatment requires that a patient have his sex partner available within 40 minutes. Once injected, the patient is required to attempt intercourse without delay. Intercourse trials are essential, since erection may not occur without sexual arousal; these give an indication of the ability of the arterial system to respond, provide proof that the corporal outflow restriction mechanism functions, give the patient a clear idea of what to expect from self-injection, and allow the practitioner to adjust dosage for optimum response. When the trials are successful, autoinjection can then be taught.

PEP has proven effective in vascular, diabetic, psychogenic, and neurogenic impotence. In younger men and those with neurogenic impotence, caution must be exercised and doses reduced in order to avoid prolonged erection. It also has proved effective for impotence after bowel surgery and radical prostatectomy. Intracavernous injection has been successful as a one-time treatment to overcome anxiety in men who are unable to consummate marriage. Because PEP is not without some risk, it should not be given to normal men who wish to improve their sexual performance.

Most men find self-injection minimally uncomfortable and not difficult. While complications are rare, those who have entered a self-injection program require strict supervision and follow-up, as these men are self-injecting without the practitioner present. The most common problem, aside from ecchymoses and transient penile pains, is the possibility of prolonged erection, which is most often seen on the initial trial injection and not thereafter. This should be treated as an emergency requiring prompt intervention within four to six hours of injection; treatment consists of intracorporal injection of a dilute vasoconstrictor followed by aspiration of retained blood. Another complication seen with long term injection consists of changes within the corpus cavernosum that do not appear as serious as originally believed. This takes the form of diffuse fibrosis or circumscribed nodular masses within the corpus cavernosum that do not necessarily correspond to the injection site. Tissue changes occur when incorrect technique extravasates the pharmacologic agent into the corporal smooth muscle. These fibrotic changes occur in about 5% of patients on long term self-injection and do not usually interfere with continued injection, but can lead to tolerance. These changes often disappear spontaneously when PEP is discontinued and even while treatment continues. Difficulty may be encountered in placing a penile prosthesis owing to these fibrous changes.

Regular follow-up is obligatory, as it provides for detection and correction of any problem that may arise, allows the physician to monitor progress, and permits dose modification to meet individual needs. With proper supervision, PEP is safe and effective. The physician must enforce the monthly follow-up visits by limiting the supply of medication dispensed to ten injections per month. PEP must be administered by a physician who is willing to provide 24-hour response to emergencies and who has the skill to treat potential urologic complications.

CONCLUSIONS

The etiology of impotence, long thought to be psychogenic, is now believed to be mainly vascular. The risk factors for ischemic heart disease and impotence are alike: aging, hypertension, diabetes, hyperlipidemia, and smoking. The criteria for successful intercourse are for the male to have stiffness for easy insertion and sufficient lasting power to satisfy both partners, and that intercourse must take place when the patient desires it. Three methods that assure penile penetration are penile microsurgical bypass, pharmacological erection (PEP), and prosthetic implant. While PEP is especially useful in vascular impotence, it finds application to neurogenic and psychogenic impotence as well.

A growing group of practitioners, many of whom are urologists and microsurgeons, now subspecialize in impotence, offering broadened diagnosis and treatment options. Informed consent requires that the patient be made aware of the risks, benefits, and alternative treatments. One can no longer restrict treatment options to prosthetic implant only, but must make all other modalities available to the patient.

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FROM THE LIBRARY

ON THE PHYSICAL CAUSES OF SEXUAL DEBILITY IN THE MALE, AS DISTINGUISHED FROM THE PSYCHIAL CAUSES

Until within comparatively recent years it was the custom among surgeons, when consulted by patients suffering from sexual debility, to refer the causes entirely to a disturbed condition of the mind, overlooking altogether the possibility that there might be some physical cause to account for the symptoms—symptoms which undoubtedly, in many cases, are conveyed through the nervous system, but which have not their seat alone in either nervous debility or in nervous depression. . . .

There are two points which enter into all the symptoms that I have heard made by patients who suffer from this cause. One is the imperfection of erections—sometimes their entire absence—and secondly, the prematureness of the emissions, followed immediately by a subsidence of the erection, supposing this function to have been perfectly normal at the start. Associated with this latter condition, there may also be an entire lack of emission, or else, if the emission takes place, the amount ejaculated is very small, and if examined under the microscope, is oftentimes found devoid or but imperfectly endowed with spermatozoa, constituting the condition of affairs which is known as azoospermism. The former may be considered as the earlier stage which tends toward the development of the second; the first point noticed by the patient being imperfect erection, associated with premature emissions and followed by an absence of erection. Since the introduction of the endoscope into urethral surgery, it has been possible to examine the urethra to see if there were any pathological conditions in this canal which were either associated with or perhaps caused this condition of affairs; but even before the introduction of this useful instrument, the older surgeons recognized the fact that these symptoms occur most frequently in patients who had been persistent masturbators, and in these, upon examination with sounds or bougies, a stricture, more or less definite, was found in certain portions of the urethral canal, and some went even beyond this, ascribing a hyperæsthetic condition of the canal as possibly one of the causes, or at any rate, one of the concomitants of this condition of affairs. . . .

Many eminent surgeons have considered that urethral stricture is induced by the habit of masturbation. If by stricture we understand any and every impediment which occurs to the passage of a sound, then, perhaps, their statement is correct, but if we consider stricture merely as those obstructions in the canal due to an infiltration beneath the mucous membrane, blocking up the lumen of the canal and preventing the passage of sounds, then I think some modification must be made of the statement that masturbation produces stricture; and particularly so if we consider for one moment the disproportion that exists between the number of males who at one time or other of their life have masturbated and the number of strictures which occur, I think we will recognize that it is too sweeping an assertion to make, that the one is caused by the other. If, however, we admit that there may be spasmodic contractions in the canal induced by some point of irritation in the urethra, then we shall modify this statement by saying that masturbation may produce a hyperæsthetic condition of the canal which leads to temporary and spasmodic contractions, but not to true stricture, and I have in a pretty fairly large experience with these cases come myself to that conclusion, for after the local treatment has been pursued, the puffiness and temporary obstruction in the canal disappear, the hyperæsthesia and inflammation subside and the canal returns to a more or less normal condition, and with it the symptoms for which the surgeon was consulted gradually improve and entirely disappear. I have noticed the same condition in men who have indulged to excess in the sexual act and who had not, so far as I could trust their word, masturbated for many years—a sufficiently long time to prevent me from considering that the masturbation had any thing to do with their sexual debility, and yet in these instances the physical conditions were precisely the same as in those who had masturbated. . . .

F. R. STURGIS, MD
(*Trans Med Soc State of NY* 1894; 328–336)

A New York initiative in medical education

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The medical schools of our country need to take a closer look at the format of medical education that was promulgated following the Flexner report over seven decades ago.

Recent reports have focused on the impact of the malpractice insurance crisis on family physicians in New York State.[1] Physicians nationwide, out of economic necessity, have reluctantly refused to accept new Medicaid patients.[1] Trust between patient and physician is diminishing, and physicians are limiting their practices, ordering more diagnostic tests, and consulting more frequently. The attrition of family physicians from death, retirement, disability, and dissatisfaction with unfavorable malpractice insurance costs and the current legal climate is extremely serious.

New York State has 15% of all medical residencies in the United States. The Report of the New York State Commission on Graduate Medical Education "accepts that residency training in New York is overbalanced in subspecialty fields."[2] In this same report, Senator (now Vice-President) Daniel Quayle is quoted as proposing that 70% of residents be in primary care specialties in order for hospitals to be eligible for Medicare reimbursement of residents' salaries. The report supported Senator Quayle's proposal and recommended that by 1990 the majority of the residents be in primary care specialties.

Kern et al[3] examined the lasting value of clinical skills of their former residents in internal medicine who had been in practice five or more years. They asked subjects what they felt their medical education lacked. Respondents indicated that there was an overemphasis on technical aspects of medicine and not enough emphasis on the fundamentals of history taking and physical examination, skills in interpersonal relations, and a more humanistic approach to caring for people. They indicated a need for more clinical counseling skills.

THE RURAL PHYSICIAN ASSOCIATE PROGRAM

The State University of New York Health Science Center at Syracuse is planning, through its Extended Rural Preceptorship (ERP), to do what the Minnesota Rural Physician Associate Program (RPAP) model has been doing for the past 17 years, which is to provide the third-year medical student physician with a nine-month experience in clinical community medicine and surgery with practicing physicians in underserved areas of upstate New York, similar to the experience reported in Minnesota.[4]

The Rural Physician Associate Program and the Extended Rural Preceptorship emphasize the points of Kern et al[3] throughout the nine-month experience. The Minnesota RPAP experience has 75% of its trainees going into primary care specialties, with 60% going into family medicine. The New York State Health Research Council has expressed keen interest in this effort. The 13 deans of the New York State medical schools have also asked for more detailed information on RPAP-ERP.

The RPAP-ERP effort may be adapted for urban as well as rural settings. Fifty family physician residents in Minnesota in the past decade have taken their 30-day community service and research effort in RPAP communities, and the majority of the others in urban settings. The stipend is paid by the practicing physicians, thereby reducing the graduate medical education costs. This effort could easily be expanded by RPAP and ERP. RPAP preceptors have said this is the best type of continuing medical education for them, as Heid[5] has reported in Minnesota.

Putting the right physicians in the right places, working harmoniously with other helping professionals in an interdisciplinary team, and linking the medical-surgical academic faculty at the State University of New York (SUNY) Health Science Center at Syracuse with practicing physicians in the community to develop a "Medical-Communiversity" is now becoming a reality. In the spring of 1989 two junior medical students from SUNY/Syracuse began a nine-month medical education journey. One is in the Canton-Potsdam area, and the other in the Hamilton-Sherburne area, with practicing physicians as their preceptors. Various SUNY/Syracuse medical and surgical specialists plan to visit the students, community physicians, and helping professionals on a more than monthly basis throughout the academic year, thereby helping to upgrade and support continuously the care of the people and the medical students' education, the practicing physicians, and helping professionals in their communities of upstate New York.

Such an ongoing effort will be beneficial to all concerned, including all New Yorkers. Students will receive full academic credit for their experiences and graduate with their classmates. They will be able to see themselves as the patients and patients' families see them, through numerous audiovisual tapings of real patients (not simula-

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tions). The SUNY faculty in the Department of Family Medicine will observe these events as they occur on a television screen in another consulting room (with permission of the patient, student, and the patient's physician). The tape will be rerun for the medical student and the faculty (and viewed by the patients if they so desire), showing what is being done and what can be done better in the history taking.[6] This educational exercise will be repeated at least five times throughout the nine months.

Efforts made by the community will help professionals to assist the student in readily identifying the person who is a substance, physical, and/or sexual abuser. Dysfunctional families within the healthy or sick community will also be identified. Ongoing close supervision of the student by the community physician (preceptor-tutor) and helping professionals, with continuous close support by the SUNY academic faculty, will help upgrade health and medical care for the citizens of this state. This program will reduce medical education costs—as preceptors are not paid for teaching—and will increase the accessibility of health care and improve interpersonal communications between doctors and the healthy, ill, and disabled citizens within the state. Physician retention and distribution should be improved, and reduction of medical malpractice litigation may also occur. The state of Minnesota has had some startling and favorable outcomes concerning matching the appropriate physician to the right location.[7,8]

ESSENTIALS OF A RURAL PHYSICIAN ASSOCIATE PROGRAM

Emphasis will be placed particularly on having preceptors and academic faculty models to enhance students' healthy life styles honoring personal and family commitments in equity and balance with professional and academic responsibilities. No student will be "on call with their preceptor" more than one night out of every four, and one weekend out of every month. This effort encourages all helping professionals to do the same (including practicing physicians), as has happened in Minnesota, where over 90% of all practicing physicians are in groups of four or more.

Medical students will be able to experience first hand the actual costs of caring for people in the doctor's office, where over 80% of a practicing physician's (preceptor's) work takes place. They will work with these preceptors in the hospital, under close supervision, and with other helping professionals, so they learn to work comfortably and harmoniously with other members of the health care community. The students will be invited to meetings of the hospital board of trustees and hospital administrators so they understand and learn that members of the nonmedical community, including lawyers, are also responsible for carrying out the hospital's responsibilities to the community. Students will be encouraged to accept invitations from rural citizens to give lectures and classes on breast and Papanicolaou examinations, prenatal care, good family and community health practices, such as preventive life style changes (smoking, alcohol) etc.

All married medical students are encouraged to live with their families in the community (separate from the preceptor's family) so that the entire family may take up new activities and meet new friends in the community. This will help students reaffirm their life career in medicine and decide more confidently on their choice of medical specialty. The Minnesota program found that RPAP students were far more confident of their ability to make career choice decisions than their classmates at the Health Science Center ($p<0.001$). ERP students will be allotted two hours of reading time each working day in the office or hospital (not at meal time) and will become computer literate if they are not already. They will have access to the Syracuse Health Science Center Biomedical Library and to the National Library of Medicine through an on-line computer network, which will enable them to perform computer literature searches from the rural hospital. These students will have one-to-one tutorialships and, in most cases, more than one tutor (preceptor) throughout the academic year with no competition from other students or classmates. They will learn the business aspects of running a private practice clinic, learn how and when to refer, see the results of their own procedures such as laceration or episiotomy repair, learn that caring for people includes caring for families and becoming involved with planned and unplanned family conferences, and learn that every disease and disability needs to be accepted and dealt with by the entire family.

Because these students will do complete histories and physical examinations on the people they serve, they will assume more responsibility and be motivated to research and read in order to help the patient and themselves in their immediate learning and caring process.

How will these students rate with their peers? In Minnesota, during the past 17 years, retention and application of this knowledge and experience helped over 500 students on RPAP pass all medical school evaluation tests at the medical school, including the National Board of Medical Examiners examination (Parts I and II), with no statistically significant differences from their classmates. The significant first-hand continuing care experiences they had while on RPAP are an added bonus when they eventually practice medicine and surgery. This is not possible in the tertiary and secondary environments provided by the medical schools of our country. Four, six, and 12-week preceptorships do not provide sufficient time to experience continuity of care, to understand personal-professional responsibility and liability issues, to develop trust and strong rapport with individuals and their families, and to develop competency in procedural, cognitive, and behavioral skills.

Students learn how to provide continuity of care in their office consulting room and learn that most devastating family and life problems are detected via outpatient encounters. Office care is the vehicle for continuity of care and gaining experience monitoring treatment response and individualizing therapy. Students learn that trust and rapport exerts a potent synergistic therapeutic and healing effect with any treatment plan, with very few (if any) side effects.

This experience is more than an apprenticeship, since the students learn to work closely as an interdisciplinary team member, and learn effective communication techniques for understanding a patient's needs, persuading patients to comply with treatment plans, and discussing sensitive medical and personal issues. Each student is given colleague status by the preceptor, which enhances personal confidence, so they do not feel as if they are "only the medical student." Married students with children felt they "wasted less time on RPAP" and had more time for themselves and their families because they did not round all day with "hordes of residents and students. We rounded to treat the patient and moved on."

It appears plausible that all state and private medical schools in New York could do the same thing. Medical schools in West Virginia, Arkansas, the Dakotas, and others are asking for more information in implementing the RPAP model. The massive transition in clinical undergraduate, graduate, and postgraduate medical education as proposed here would help to cut medical education costs and upgrade medical care and education throughout the state. It will produce a more humanistic physician with a broader knowledge base and steeped in primary care and family medicine experience, as recommended by the New York State Commission on Graduate Education and the Association of American Medical Colleges.[9,10] The hostile polarization of people towards hospitals and physicians will be reduced, eventually helping to alleviate the current malpractice crisis and producing more of the right physicians for the right place.

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THIRTY-EIGHT PHYSICIANS SEEK RURAL PRACTICE

Considerable concern has been manifested by welfare associations and those interested in rural community life, by the apparent growing tendency on the part of physicians to move their residence from the rural districts to the larger centers, where hospital and laboratory facilities will be more accessible. In some instances this withdrawal has occasioned considerable alarm on the part of the community thus apparently forsaken.

A more careful study of the problem recently thrust upon this office has afforded an opportunity for seeing the matter from another angle. In a majority of instances the community lost its old family physician because "Father Time" had a claim upon him, and no young man has offered to take up his practice because, with the advent of the automobile and good roads, it is possible for the residents of such communities to call a physician in attendance in less time than could the old family physician, when he was a young man, attend their fathers. The forlornness of these communities is, in many instances, considerably exaggerated through sentiment. The fact that the country store which at one time prospered at the cross-roads, has disappeared, has been accepted as a normal evidence of development. The farmers, with their automobiles, prefer to do their shopping in the larger centers, and are justified in doing so. Likewise, when they are in need of medical service, not emergency in character, they also prefer to seek such service in larger cities, and are capable of doing so with the aid of the automobile. Thus the physician living in the rural district is overlooked and the more lucrative practice is carried to the city, or the city physician is induced to come out, which he willingly does when the roads are passable; and the high regard which the community feels for his local physician never comes to the surface until he, responding to the pressure of conditions, decides to take up his residence in a larger center.

That physicians would be willing to live in rural districts, providing they could do so and effectively "keep the wolf from the door," is manifested by the fact that within the last month thirty-eight physicians have written to this office asking for a list of rural communities in need of physicians. Unfortunately we had at our disposal only four places where the County Medical Societies thought physicians might be able to develop a practice warranting their taking up the location.

J. S. L.

(*NY State J Med* 1925; 25:38)

Dietary changes and the decline of scurvy and tuberculosis in 19th century Europe

VINCENT J. KNAPP, PHD

The vast proportion of Europeans entered the 19th century as grain-eaters. In the unforgettable remark of the noted French social historian, Fernard Braudel, "Eating consist[ed] of a lifetime of consuming bread, more bread, and still more bread and gruels."[1] This precarious dependence by the masses on grain was further threatened time after time by ergotism or spoiled rye.[2] In 1800, luxury foods such as meat, fish, cheese, eggs, vegetables, and fruits were regularly a part of what the upper 10% of society ate, but the mass of Europeans saw them rarely.[3] All of this changed, and changed dramatically, in the decades after 1800. As the American historian E. Neige Todhunter has pointed out, "Not till the 19th century [in Europe] . . . , was there a marked improvement in the quantity and quality of food."[4]

LIMITED DIETS

This democratization of the food supply, which made items only previously available to the elite common to all levels of society, produced what the contemporary French historian Jean-Jacques Hémardinquer has justifiably called "a true nutritional revolution."[5] The widespread consumption by the majority of potatoes and other vegetables and of meat, fish, butter, and milk added unprecedented levels of nutrition to the general European diet.[6,7] Rising amounts of amino acids, lipids, vitamins, and minerals, along with carbohydrates, constantly improved levels of immunity.[3,8,9] The result of all this was a population better able to ward off disabling diseases such as scurvy, and deadly diseases such as tuberculosis, along with other disorders.

Writing recently about the connection between diet and disease, the American social historian Ann G. Carmichael has commented with some incisiveness that "the links between undernutrition and infection among the poor of today's Third World are socially and demographically significant. From birth to an early grave the victims of hidden hunger endure episodes of illness which wealthier nations and individuals do not suffer."[10] What is true of the Third World now was equally the case in Europe during the late 18th and early 19th centuries. This fact, however, was not easily perceived, even by Europeans living at the time. In 1837, the English sociologist Andrew C. Combe realized what few other Europeans had seriously considered up to that point. Speaking frankly, he declared, "To those who have never reflected on the subject, it may seem like exaggeration to say that . . . at least 9/10 of the lower order suffer physically and intellectually from being overworked and underfed."[11] Few, he felt, died from want of food, but many had their lives shortened "by [an] excess of labour and deficiency of nourishment."[11] Actually, the situation was far worse than Combe perceived in 1837. For Europe was in that year, and indeed beforehand, a disease-ridden society—a condition that was directly linked to poor nutrition in general, and to Europe's long time overdependence on breadstuffs in particular. Bread really was the culprit here, largely because alone and by itself it supplied so little of the nutrition required by Europeans in order to live a reasonably healthy life.[3]

This intimate connection between human misery on the one hand and pathological disorders on the other was pointed out in more recent times by E. Parmalee Prentice, when he noted that "a diet too largely made up of bread, may . . . bring whole armies of disease."[12] And it did. This distressful state was the direct result of a society within which the average individual in Europe before 1800 was forced by fate to eat more than two pounds of bread each and every day.[4] As it turned out, the misery of this day and age was defined in terms of a series of disabling and deadly diseases that, in the words of the famous 19th century German philosopher Rudolf Virchow, "destroy[ed] the enjoyment of life for the many."[13]

Everyday life was too often disrupted in pre-industrial Europe by disabling diseases such as scurvy, psoriasis, malaria, diphtheria, influenza, and measles, along with their much more deadly counterparts, typhoid, syphilis, typhus, cholera, and tuberculosis. There truly was a vicious cycle at work in European society before it experienced its authentic nutritional revolution. It began with prolonged undernourishment and hidden hunger which, again in the words of Ann G. Carmichael, created a fatal situation in which "malnutrition enhanc[ed] infection . . . and . . . infection caus[ed] further malnutrition."[10] The unqualified link between poor nutrition and the existence of pathologic conditions can be seen clearly and historically in the cases of two diseases: scurvy and tuberculosis. Analytically, the tie between scurvy and insufficient diet turned out to be obvious, while the causal connection between tuberculosis and improper nutrition is somewhat more subtle and difficult to see, but was likewise there.

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THE UNIVERSALITY OF SCURVY

Scurvy was one of the numerous maladies that harassed European society down through the centuries. Its constant presence in one European country after another attests to the fact that the bulk of the population ate little in the way of vegetables and fruits, the very foods that easily could have banished this disabling disease before the 19th century. The age of seafaring, which began with Columbus, da Gama, and Cabot in the 1490s, highlighted this somber disorder as never before, primarily because scurvy felled crewmen in large numbers. Sailors plying the great Atlantic and Pacific sea lanes were all too familiar with its early symptoms, which included a debilitating lassitude, sore gums, and loosening teeth. But they had far more to fear from its later symptoms, which included hemorrhaging from the lungs and swollen and blackened limbs.[14] However, long before the 1490s, in the cities of Europe, where most vegetables and fruits were virtually unobtainable through the fall and winter months, these symptoms were both well known and pronounced in victims. Indeed, as Anthony J. Lorenz has so accurately stated: "[Land] scurvy was an annual spring epidemic for the large cities." "No wonder," Lorenz goes on to say, "the earliest writers on the subject were continental. They hailed from Amsterdam, Leipsic [sic], Marburg. It was not until after 1492 that we had read of sea scurvy."[15] The presence of this disease was also widely reported among children, who were often said to be subject to infantile scurvy upon weaning.[16]

We now know that this excruciating condition was caused by a very specific avitaminosis.[17] The first man to suspect this, even vaguely, was the Scottish doctor, James Lind (Fig 1). Writing in 1753 about experiments he had judiciously conducted with scorbutic sailors, he explains how he quickly discovered that those given citric foods recovered rather instantly from this disease. His carefully conceived experiments proved without a doubt that when it came to this disease, oranges, lemons, and cider had therapeutic qualities that had not been previously discerned.[15] A century later, in 1842, the noted British physician George Budd argued to a public becoming more and more appreciative of vegetables and fruits that the only cause of scurvy "is [the] complete and prolonged abstinence from succulent vegetables or fruits, or their preserved juices as articles of food . . . ."[18] Scurvy was hard to vanquish prior to 1800 because vegetables and fruits had such a low priority among doctors and the general public.[19,20] This ingrained prejudice against vegetables and fruits, along with the sheer scarcity of these sources of ascorbic acid, the key nutrient here, tended to doom the general population to this disease.[21,22]

As it turned out, the defeat of this disorder occurred subtly and inevitably, first with the introduction of potatoes and onions to the diet and then later with the coming of oranges and lemons. In the interest of precise analysis here, it should be pointed out that the potato only helped partially, since it contains but one fifth of the vitamin C (ascorbic acid) that can be found in such citric products as oranges and lemons.[23] This whole process was also aided and abetted as the 19th century wore on by the progressive addition to the diet of other legumes, besides the potato and onion, and other fruits like apples and cherries.[7,24]

FIGURE 1. James Lind (1716–1794). Pioneer in developing a cure for scurvy (from the Bettmann Archive, New York City).

TUBERCULOSIS

Scurvy was more often than not a disabling rather than a deadly disease. Unfortunately, tuberculosis was both. This disease was often active within the body for years before it finally claimed its victims, and only after the individual had had an extended bout with some truly agonizing symptoms. These often disquieting symptoms were described in 1799 by the British physician Thomas Beddoes. He reported that those terminally ill from pulmonary tuberculosis had to endure symptoms that included a

> hard rending cough, attended sometimes by retching and vomiting, [by] expectoration sometimes nauseous, [and] always offensive to the eye and harassing when it is not free, [by] langour . . . [and] the extremes of cold and heat . . . [by] sweats . . . ,[by] breathlessness . . . , and [by] a sense of drowning . . . terminating in actual drowning when there is no longer strength to bring up fluids secreted by the chest."[25]

The tyranny that tuberculosis could exercise at this time was best described by the great English novelist Robert Louis Stevenson. Talking about his own experience with this disease, he stated plaintively, "I lie awake troubled by a hacking, exhausting cough and praying for sleep or morning, from the bottom of my little shaken body. I have written in bed, and written out of it, written in haemorrhages, written in sickness, written torn by coughing . . . ."[26]

Before it was finally overcome, tuberculosis was the most widespread disease to occur in European history. The father of modern bacteriology, the Germany medical researcher Robert Koch, called it Europe's greatest health problem ever[27] (Fig 2). According to statistics compiled by John Bateman, the death toll from tuberculosis in Lon-

FIGURE 2. Robert Koch (1843–1910). Discoverer of the cause of tuberculosis (from the Bettmann Archive, New York City).

don rose steadily from one in every 6.2 deaths in 1669 to one in every 5.5 mortalities in 1749, to one in every 3.6 people dying in 1808.[28] These figures were definitely on the low side, since Bateman's statistics included only deaths from pulmonary tuberculosis. What he did not realize in the early 19th century was that the other forms of tuberculosis, not yet discovered by the medical profession, were killing off an additional 17% of the population.[29] After apparently peaking in Britain early in the century, the disease took hold on the Continent. Here the population was indisputably saturated with the disease. Koch's discovery of tuberculin in 1894 gave the medical profession a diagnostic tool for determining just how extensive the disease really was. The results were shocking, for tuberculin proved unquestionably that a full 85% of the population was in some way wrestling with the consequences of this disease.[30]

The initial decline of tuberculosis within European society was evidently not the result of any medical miracle, although there was growth in medical knowledge about this disease as well as significant public health campaigns. Even with these changes, however, the first workable vaccine against this disease was not marketed until 1929, and the final defeat of tuberculosis was not a given reality until the introduction of streptomycin in 1946.[31,32] Yet prior to these dates, deaths from tuberculosis were noticeably on the wane. In Britain, the decline was almost precipitous by the middle of the 19th century. In major English urban centers such as London, Liverpool, and Ipswich, the death toll from tuberculosis had already dropped to just 12%.[33] On the Continent itself, where the death-dealing effects of this disease had been felt later in the century, death rates also fell off beginning in the 1880s and 1890s, especially in major industrial states like France, Belgium, and Germany.[34] By the eve of World War I, tuberculosis was no longer seen as a disease that society passively had to put up with. The unmistakable loosening of tuberculosis' grip on this society may have been due to a decline in the virulence of the bacteria that causes the various forms of this disease. But just as probably, the disease was in eclipse because of rising levels of immunity within the general population aided by dietary changes.

The belief that tuberculosis, like scurvy, was actually a deficiency disease somehow tied to nutrition is by no means a novel one. The first individual in western civilization to insist on the still contentious point that there was an intimate connection between tuberculosis and diet was none other than the Greek medical thinker Hippocrates.[35] If we assume, as Hippocrates did, that this connection was there, then the reason tuberculosis declined in Europe was the direct result of the steady addition of certain co-enzymes and amino acids to the food of the masses. When this occurred, and it did rapidly after the 1830s and 1840s, it meant that Europeans were able, most likely better than ever before, to produce antibodies against invading microorganisms. The increasing biological strength of most Europeans had to be the consequence of the ingestion of these key nutrients. That process, in turn, evidently produced a rising level of immunity within the population, thanks in part to a greater intake of vegetables and fruits, which provided higher levels of vitamins A, C, and E—the first two critical co-enzymes and the third needed to sustain the other two; and the dramatic addition of meat and fish, which produced the vital amino acids the body needed to create antibodies.[36,37] Without really knowing their redeeming biochemical worth, famous medical men like the Frenchman Marcel D'Arcet pushed for the greater consumption of vegetables and meats among mass elements.[38] These novelties became basics over the course of a very few decades. This fact was quickly recorded by Charles Booth, that justifiably famous student of English poverty. In the 1880s, even among the less well-off of London he noted that "meals are more regular. For dinner meat and vegetables are demanded every day."[39]

What Charles Booth proved about the poor of London later in the 19th century has been substantiated elsewhere, and with respect to other social groups by more recent historical research. Those studies, stemming from any number of European countries, demonstrate unequivocably that the general population was absorbing vitamins A, C, and E at this time as never before. It is true that for a generation or so too many people were totally dependent on the potato for most of these nutrients.[40] But gradually, other vegetables and fruits, also carrying these co-enzymes and biochemical supporters, spread into the popular diet. Included here were such previous "strangers" as peas, parsnips, carrots, cabbages, peaches, apples, and oranges.[41] These foods had been consumed regionally before this, but never universally. The question might be legitimately asked at this juncture, what happens to a population deprived of just these vitamins? The answer has come from two scientists, both Rumanians, who in recent years have been in the forefront of current nutritional research. These two men, Iancu Gontzea and Florin Jantea, have shown through their studies that "insufficient vitamin intake" has a very negative effect on the body's "susceptibility to infections and the organism defence mechanisms."[35] From an ever-growing amount of evidence, it could be concluded that the European population, because of undernutrition, had been at risk for many centuries, with tuberculosis in the 19th century being just the most recent of these risks.

DIET AND DISEASE

Prior to the great dietary revolution of the industrial period, Europeans were biologically imperiled in another way, and that was caused by the acute shortage of certain amino acids in their food supply. As early as the 1910s, medical science knew that malnutrition, the susceptibility to disease, and death could be the direct result, as two of the pioneers in this area, the Americans Thomas Osborne and Lafayette Mendel, said, "[of a] lack of some fundamental unit like tyrosine, trytophane, lysine or cystine."[42] With meat, fish, and milk in such short supply prior to the 19th century, it was little wonder that the population at large was so starved for protein. Deprived of key amino acids, these people suffered from low antibody production and extremely low levels of immunity. Once meat and milk became integral parts of the everyday diet, Europe's long history of massive protein deprivation came to an end.[7,22,43-45] Concomitantly, Europe's generations-long bout with the death-dealing effects of tuberculosis also started to come to an end, and as the evidence suggests, more than likely not by coincidence.

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CASE REPORTS

Meningitis following bacteremia with *Streptococcus sanguis*

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Certain species of bacteria are known to be meningiotropic, with a high potential to invade the meninges when carried in the blood.[1] These organisms include *Neisseria meningitidis*, *Streptococcus pneumoniae*, and *Haemophilus influenzae*. Other organisms such as *Staphylococcus aureus*, viridans streptococci, and the enteric gram-negative rods rarely invade the meninges, despite documented episodes of continuous bacteremia.[2]

We report a case of *Streptococcus sanguis* bacteremia following an esophageal dilation procedure, with subsequent meningitis by the same organism. A detailed account of the development of the signs and symptoms of bacterial meningitis is presented.

CASE REPORT

A 48-year-old man was admitted to the hospital for dilation of an esophageal stricture secondary to esophageal reflux. During his two-month hospital stay, he underwent four sessions of bougienage using sequentially larger Maloney dilators with no sequelae. The dilators were decontaminated with 2% glutaraldehyde prior to each use. Three hours after the fifth session, in which no complications were observed during the procedure, the patient complained of chills. He was not receiving antibiotics prior to or during the procedure. One half hour after the chills began, he started to complain of severe headache involving his entire head.

His medical history included chronic alcoholism, chronic pancreatitis for five years, multiple episodes of diabetic ketoacidosis, and undescended right testicle. He had had insulin-dependent diabetes for 13 years. He had smoked one pack of cigarettes a day for 21 years. He had no risk factors for acquired immunodeficiency syndrome.

On physical examination, the oral temperature was 37.2°C and 40.0°C, three hours and six hours, respectively, after the procedure. The heart rate was 120/min; respiratory rate, 26/min; and blood pressure, 140/95 mm Hg. The patient appeared to be well nourished. Fundi and mucous membranes were normal. There were no petechiae or purpura on the skin. The three remaining teeth were in good hygienic condition. No heart murmurs were audible, and the lungs were clear. The abdominal examination was normal. The patient had Brudzinski and Kernig signs. The mental status remained normal until six hours after the procedure, when he became very lethargic. No focal neurologic signs were detectable at any time.

The chest roentgenogram was normal, as was an emergency computed tomogram of the head. The peripheral leukocyte count was 3,000/mm^3 prior to the procedure and 12,000/mm^3 at three hours after the procedure during his chills. It showed 83% polymorphonuclear forms, 10% band forms, and 7% lymphocytes. Hemoglobin was 10.8 g/dL. A lumbar puncture was performed with an opening pressure of greater than 40 cm water. The fluid was cloudy with a cell count of 1,170/mm^3 (97% polymorphonuclear forms and 3% lymphocytes), and 18 erythrocytes/mm^3. Cerebrospinal fluid (CSF) protein and glucose levels were 275 and 127 mg/dL, respectively, with a simultaneous serum glucose of 425 mg/dL. Gram stain of the CSF showed many neutrophils and occasional intracellular gram-positive cocci (Fig 1). Three sets of blood cultures and the CSF grew *Streptococcus sanguis* with the same identification number on the API 20S Strep system (Analytab Products, Plainview, NY).

The patient was initially treated intravenously with 2 g every four hours of nafcillin, ampicillin, and cefotaxime at eight hours after the procedure. Gentamicin was given simultaneously in a dose of 80 mg every eight hours. The minimum inhibitory concentrations of antibiotics for the organism were penicillin, 0.03; ampicillin, 0.03; cefotaxime, 0.1; and gentamicin, 0.5 μg/mL. After 48 hours of empiric broad spectrum antibiotics, therapy was changed to ampicillin and continued for the next five days. After 72 hours, the headache began to resolve. The chills did not recur after the therapy, and the rectal temperature returned to normal within 24 hours. The patient continued with the esophageal dilation procedure with prophylactic ampicillin, and he was well at six-month follow-up.

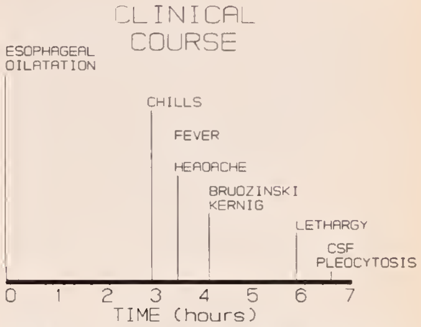

FIGURE 1. Clinical course of the patient immediately after the esophageal dilation. Note the sequence of signs and symptoms.

The course of the development of symptoms of meningitis following meningeal infection is summarized in Figure 1.

DISCUSSION

Meningitis caused by the viridans group of streptococci is rare, despite occasional reports of such cases.[3,4] Studies of bacteremia during endocarditis by these organisms show that the bacterial densities in the blood are approximately 50 to 500 organisms per milliliter.[5] Yet, despite large numbers of patients with viridans streptococcal endocarditis, few develop meningitis. This group of oropharyngeal organisms can easily enter the bloodstream with even only slight trauma, such as that which occurs during brushing of the teeth.[6] The degree of bacteremia is small.[6]

A review of infectious complications resulting from bougienage is presented in Table I. The incidence of bacteremia ranged from 8% to 94%.[7-15] This incidence of bacteremia varied depending

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on how long after the procedure the blood samples were obtained for culture. Data from Botoman and Surawicz[15] support the transient nature of the bacteremia. One author believes that contaminated instruments greatly contributed to bacteremia,[8] while another investigator correlated bacteremia with the degree of trauma to the esophageal mucosa.[10] Species of alpha hemolytic streptococci and staphylococci are the predominant organisms isolated from the blood. The streptococci were most likely host flora, and staphylococci may be from contaminated instruments. Therefore, both factors may be important in contributing to the complications of such procedures. The reported complications from esophageal dilation (Table I) included brain abscess, endocarditis, and bacteremia. No previous cases of severe bacteremia and ensuing bacterial meningitis have been reported following this procedure; and, in fact, prophylactic antibiotics for the procedure are commonly accepted as unnecessary.

In the patient described here, who was not acutely ill prior to the procedure, a clear episode of shaking chills occurred three hours following the procedure. This acute episode would indicate that the bacteria entered into the bloodstream after the procedure, probably in an area traumatized by the dilator. This is especially likely as the intra-abdominal and intragastric pressures fluctuate quickly and greatly. There exists the potential for large numbers of organisms to be forced into a low pressure, open, and traumatized vein in the esophagus. *S sanguis* is an oral flora organism and can be found in the intestinal tract, not on instruments. Therefore, the sterility or the lack of sterility of the dilator did not seem to contribute to the course of events. From the clinical course of the patient, a large number of organisms must have invaded the bloodstream. Blood cultures taken all contained the same organism.

This case offers a rare opportunity to observe the time course of meningitis following seeding of the meninges by the bacteria in the bloodstream. In contrast to patients often seen with pneumococcal or *Haemophilus influenzae* meningitis, in which the symptoms have usually been present for 24 to 48 hours,[16] the symptoms developed within minutes of the chills and fever in this patient. Signs of meningeal irritation were present within one hour of the bacteremia. Less than four hours after the onset of chills, many neutrophils were already present in the CSF. There is a possibility that the patient had been incubating meningitis prior to the procedure, but the complete lack of physical findings or complaints makes this possibility difficult to support.

No one will disagree that *S sanguis* is not a meningiotropic organism like meningococus. Yet this case demonstrates in humans a conclusion based on experimental *H influenzae* meningitis in infant rats:[17] the propensity for development of meningitis during bacteremia is related to the following equation: propensity = (density of bacteria in the blood) × (duration of bacteremia)/ability of the host to mount a defense against the organism.

It is unfortunate that quantitative blood cultures were not performed during this patient's illness. Nevertheless, the shaking chills following the procedure are highly suggestive of a high degree of bacteremia. Clinicians should conclude that, as in the experimental animal model of meningitis, any organism that is carried in the blood may cause meningitis if found in high densities in the blood for an adequate period of time. High-level or persistent bacteremia by any organism should be treated as soon as possible.

TABLE I. Summary of Reported Bacteremic Episodes and Complications from Esophageal Dilation

No. of Patients Studied	Bacteremia Rate	Organisms Recovered from Blood	Conclusions or Complications	Reference
1	1/1	*Staphylococcus*	Cerebral abscess	Rontal,[7] 1973
18	17/18 (94%)	*S epidermidis; Staphylococcus aureus*		Raines,[8] 1975
14	2/14 (14%)		Bacteria from nonsterile instruments	Raines,[8] 1975
3	0/3		Cerebral abscess	Leahy,[9] 1977
11	5/11 (45%)	Viridans streptococcus; *Staphylococcus* coagulase-negative	Mucosal trauma caused bacteremia	Stephenson,[10] 1977
17	2/17 (12%)	*Staphylococcus epidemidis*	Contaminants	Welch,[11] 1983
1	1/1	Viridans streptococcus		Yin,[12] 1983
13	1/13 (8%)	*Klebsiella*		Yin,[13] 1983
1	1/1	Viridans streptococcus	Endocarditis	Niv,[14] 1985
59	27/59 (46%)	*S aureus*; Viridans streptococcus		Botoman,[15] 1986
1	1/1	*Streptococcus sanguis*	Meningitis	Present case

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Determination of cerebral blood flow in an arteriovenous malformation using SPECT scanning

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Arteriovenous malformations (AVMs) of the brain are one cause of massive intracerebral hemorrhage and sudden death syndrome.[1] Recently, earlier recognition of these congenital blood vessel abnormalities, using such modalities as magnetic resonance imaging (MRI), has helped to identify patients in whom the only manifestations are headaches, seizures, and transient neurologic deficits.[2]

The disturbances of neurologic function have been attributed to underlying cerebral ischemia caused by the AVM. Deprivation of blood flow and nutrients has been postulated to be secondary to either "steal phenomenon," in which blood is shunted away from cerebral tissue into non-nutrient AVM vessels, or to venous hypertension and the compressive effects of the engorged veins within the malformation.[1]

Previous studies of cerebral blood flow in patients with AVMs have done little to quantify the perfusion of the area of brain intimately associated with the malformation. Radiographic or fluorescein angiography demonstrates alterations in large vessel flow adequately but does not reflect microvascular patterns of perfusion. Rosenblum et al[2] used laser Doppler velocimetry to measure local cerebral blood flow but could only use this technique in the zone surrounding an AVM. Conclusions regarding adequacy of blood flow and ischemia were therefore inferred. Positron emission tomography (PET) using radiolabeled oxygen, although an excellent indicator of local cerebral blood flow, is expensive and not readily available.

Single photon emission computed tomography (SPECT) scanning is an attractive method for measuring local cerebral blood flow, since it can easily be performed with standard nuclear medicine equipment.[3] This is the first report to our knowledge of blood flow imaging with SPECT scanning in a patient with an AVM before and after surgical obliteration.

CASE REPORT

A left-handed, 31-year-old man, previously healthy, had a single grand mal seizure. Neurologic examination after recovery demonstrated no deficits.

Computerized axial tomographic (CAT) scanning with contrast enhancement demonstrated several vermiform structures in the right parietal region, which were consistent with tortuous, enlarged blood vessels (Fig 1). Cerebral angiography revealed the presence of an arteriovenous malformation supplied exclusively by branches of the right middle cerebral artery and draining exclusively via a distended cortical vein into the superior sagittal sinus (Fig 2). SPECT scanning with n-isopropyl p-iodoamphetamine iodine 123 (IMP) showed diminished perfusion of the brain beneath the AVM, although surrounding areas appeared to receive normal blood flow (Fig 3). Cerebral dominance testing revealed lateralization of the speech center to the left hemisphere.

The patient underwent right parietal craniotomy and microsurgical excision of the AVM. After surgery, the patient showed no evidence of neurologic deficit. Repeat SPECT-IMP scan revealed a wedge-shaped defect representing absent perfusion in the region of the surgical resection. Surrounding brain demonstrated no increased blood flow when compared to the preoperative study (Fig 4).

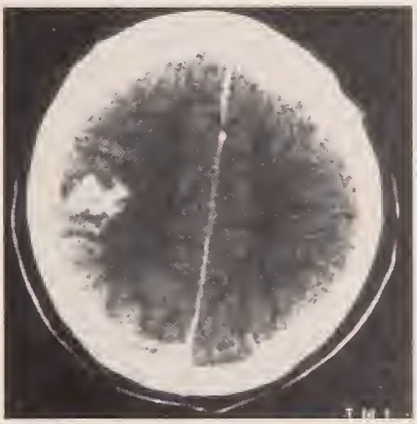

FIGURE 1. Contrast enhanced computerized axial tomogram reveals prominent vascular structures in the right parietal region consistent with an AVM.

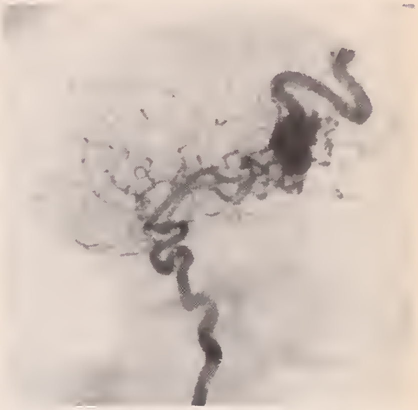

FIGURE 2. Cerebral angiography via a right internal carotid artery injection demonstrates an AVM supplied by the middle cerebral artery and draining into the superior sagittal sinus.

DISCUSSION

IMP uptake is a good measure of cerebral blood flow because of its high first-pass extraction by the brain and relatively slow washout.[4]

SPECT scanning with IMP has been used in the imaging of cerebral tumors and in patients with epilepsy and dementia, but has not added as much to the diagnostic information in these areas[5,6] as in the field of ischemic states and the evaluation of intracranial occlusive disease, stroke, and transient ischemic attacks.[7] This technique can demonstrate zones of diminished perfusion with greater accuracy and sensitivity earlier in their evolution than routine computerized axial tomography. Delayed scans have also been useful in outlining the ischemic penumbra and refilling phenomenon around vascular cerebral insults. This has great predic-

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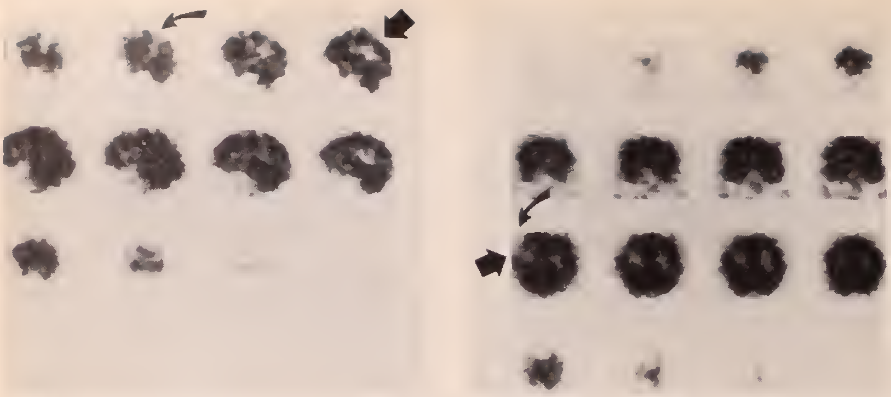

FIGURE 3. Preoperative SPECT-IMP study shows diminished perfusion in the brain beneath the coils of the dilated blood vessels within the AVM. Surrounding cerebrum has normal blood flow. Curved arrows demonstrate a linear decrease in activity which corresponds well with the distended cortical vein draining the arteriovenous malformation into the superior sagittal sinus. This supports the role of the compressive effect of engorged veins and venous hypertension in the diminution of cerebral blood flow. Left, sagittal images. Right, coronal images.

tive value for evaluating clinical recovery.[7]

In this instance, SPECT scanning with radiolabeled iodoamphetamine exquisitely demonstrated a zone of ischemic brain in the region of the AVM. Routine neuroradiologic examination would not have the capacity to measure physiologic function, as these tests only demonstrate anatomic detail. Normal perfusion was demonstrated in the region surrounding the malformation both before and after operative obliteration. This picture does not support the concept of steal phenomenon. If "steal" did occur, one would expect far-reaching areas of decreased local cerebral blood flow surrounding the malformation. Delayed scanning may be useful to check for the presence of blood flow redistribution, a hallmark of the "steal" phenomenon. The SPECT images, however, do validate the AVM as a localized congeries of shunt vessels which are not involved in cerebral supply, and which can therefore be safely removed. Decreased IMP activity within the AVM supports the concept of brain within the lesion as tissue that has been damaged by loss of local blood flow to the malformation. This results in loss of normal function by the cerebral tissue.

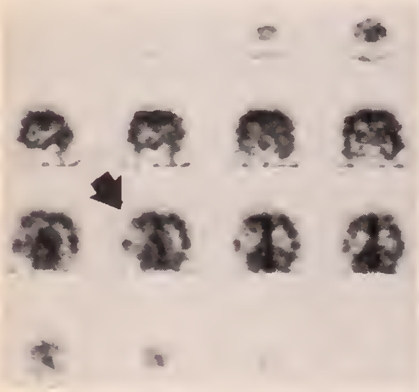

FIGURE 4. Postoperative SPECT-IMP scan showing lack of perfusion in the area of surgical resection. The linear defect from the superficial draining vein is no longer present. The remainder of the brain shows no change in blood flow.

SPECT scanning with IMP is a safe, easy to perform, and reliable test which can be performed with a standard rotating gamma camera. Its expense and relative nonspecificity may limit its utility as a screening tool for AVMs in patients with few or no neurologic findings but with a history of headaches, seizures, syncope, or transient ischemic attacks. In those with AVMs, the presence of underlying zones of cerebral ischemia suggests a causative correlation with neurologic symptomology and may help to guide future evaluation and the need for surgical therapy. In conjunction with angiography, SPECT scanning may be helpful as a tool to guide the surgeon.

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Treatment of dolichoectasia of the middle cerebral artery with bypass and exclusion

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Dolichoectasia of the intracranial arteries is an uncommon but serious disorder.[1-5] Although the most familiar clinical presentation is in the vertebrobasilar circulation,[6-9] it is also known to occur in the carotid distribution. When the anterior circulation is affected, the most common site of disease is the supraclinoid internal carotid artery (ICA), and the middle cerebral artery (MCA), particularly the M-1 segment.[1,10,11] These lesions present a therapeutic challenge, with several major issues that must be addressed when surgical management is proposed. We report a case of dolichoectasia of the proximal MCA. Treatment included intracranial clip exclusion of the aneurysm from the circulation and superficial temporal artery (STA) to deep middle cerebral artery (STA-MCA) bypass. The literature is reviewed, and treatment options are discussed.

CASE REPORT

An 18-year-old woman presented to the emergency department of Montefiore Medical Center after suffering her first grand mal seizure. The patient had previously been in excellent health, but reported experiencing a headache of precipitous onset for the three days prior to the ictus; the headache began on the right side prior to generalizing. The patient was awake and alert within 30 minutes following the ictus. Examination revealed nuchal rigidity, photophobia, and unsustained ankle clonus on the left side in association with an ipsilateral extensor plantar reflex. Routine hematologic and biochemical studies were unremarkable; serum human chorionic gonadotrophin levels were elevated, and pelvic ultrasonography was consistent with an early gravid state.

A non-contrast computed tomographic (CT) scan demonstrated diffuse subarachnoid blood. Additionally, there was the suggestion of an aneurysmal dilation of the right MCA (Fig 1). The remainder of the study showed minimal hydrocephalus and an old infarct in the right globus pallidus. Four-vessel angiography (Fig 2) revealed an aneurysm of the right MCA extending from the middle portion of the M-1 segment to the trifurcation. The aneurysm appeared to be fusiform in configuration. The lenticulostriate arteries were thought to originate from the proximal (preaneurysmal) portion of the M-1 segment. A moderate amount of vasospasm was present, but there were no other aneurysms seen. A common carotid injection showed the superficial temporal artery to be adequate for utilization in case an extracranial to intracranial (EC-IC) bypass would be required. On the 11th hospital day (post-bleed day 14), the patient underwent a surgical procedure.

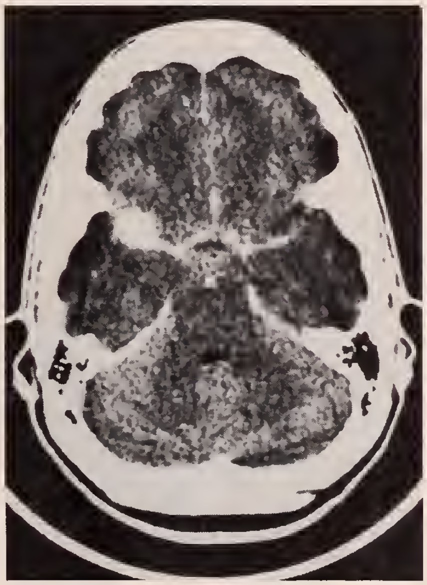
FIGURE 1. Non-contrast CT scan of head demonstrating subarachnoid hemorrhage and suggesting large right middle cerebral artery aneurysm.

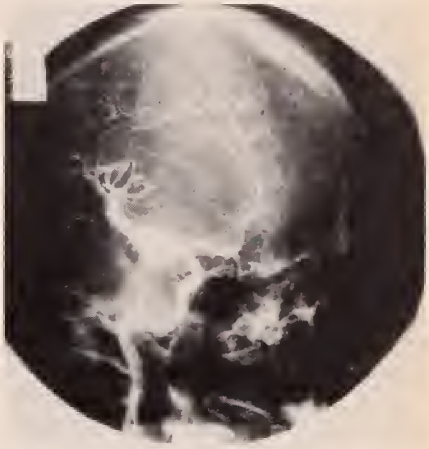
FIGURE 2. Selective right internal carotid arteriogram, anteroposterior oblique projection, demonstrating fusiform middle cerebral artery aneurysm. Mild vasospasm is also present.

During the surgery, complete dissection of the aneurysm confirmed the angiographic impression of the fusiform nature of the lesion (Fig 3). The lenticulostriate arteries were found to be arising in the preaneurysmal portion of the MCA. Due to an overhanging, saccular portion at the proximal end, direct visualization of the lenticulostriates and the M-1 segment just proximal to the aneurysm was impossible until this

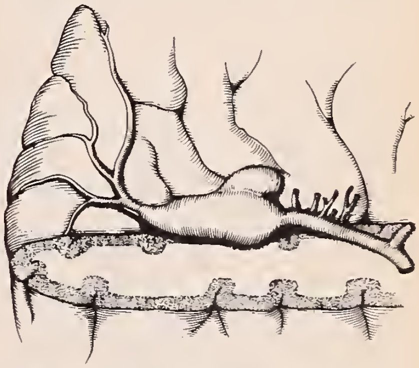
FIGURE 3. Artist's conception of lesion as it was situated in the right Sylvian fissure. Note lenticulostriate vessels arising from the middle cerebral artery prior to the origin of the aneurysm and the overhanging pouch of the aneurysm.

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portion of the aneurysm dome was clipped and resected. It was then evident that the entire portion of the M-1 distal to the lenticulostriate branches was aneurysmal. It was felt that an acceptable lumen could not be reconstructed, and in light of the fact that this patient had presented with subarachnoid hemorrhage, it was felt that wrapping was not an acceptable treatment. The decision was made ultimately to exclude the lesion. The STA was anastomosed to one of the main (M2) branches of the trifurcation of the MCA in the Sylvian fissure. The aneurysm was then excluded from the circulation by the use of clips placed proximal to the beginning of the dilation (beyond the most lateral lenticulostriate), and proximal to the trifurcation (Fig 4). During the procedure, the patient was maintained in a normotensive state, and mannitol was used. The patient promptly awoke from surgery, and no postoperative deficits were noted. Prior to discharge, an elective termination of pregnancy was performed, but the patient otherwise enjoyed an unremarkable recovery. A postoperative angiogram (Figs 5, 6) confirmed the exclusion of the aneurysm from the circulation and the patency of the bypass graft, with filling of the entire distal middle cerebral circulation through the bypass. The lenticulostriate vessels were visualized. The patient was discharged to home on anticonvulsant therapy and aspirin.

Histopathologic analysis of the resected aneurysm was consistent with that reported elsewhere. There was a diffuse loss of elastic tissues, as well as a disordered pattern to the muscular fibers. There was no focal area that was completely devoid of muscular fibers. There were no other findings suggestive of connective tissue disorders.

Two months after the surgery, the patient presented with acute onset of a left upper extremity monoparesis. A neurologic examination was otherwise within normal limits. The CT scan was without change, and cerebral angiography confirmed the patency of the bypass, with good filling of the distal MCA as well as the anterior circulation. There was occlusion of the M-1 segment proximal to the aneurysm, and some of the lateral lenticulostriate vessels were no longer seen. The patient was placed on heparin and regained normal neurologic function over the next 24–48 hours. A repeat CT scan confirmed the presence of a new, small infarct in the internal capsule. The patient was discharged on warfarin, without neurologic deficit, and has remained well as of her last follow-up visit, one year after the surgical procedure.

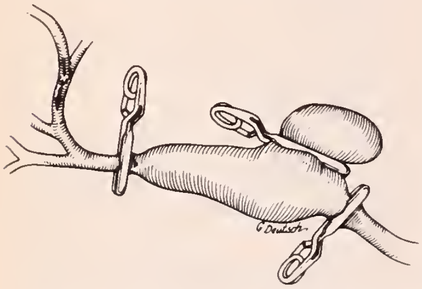

FIGURE 4. The final appearance of the treated lesion. Clips have been placed proximal and distal to the lesion, as well as on the pouch-like dilation (which was resected), prior to final placement of the proximal MCA clip. The superficial temporal artery has been anastomosed to a main branch of the middle cerebral artery in the Sylvian fissure.

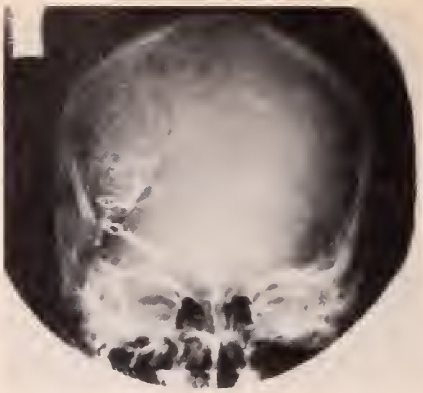

FIGURE 5. Postoperative angiogram with selective anteroposterior right external carotid injection. The bypass is patent and readily fills the middle cerebral artery circulation, and there is no reflux into the aneurysm.

Discussion

Although Dandy[12] is generally credited with the first complete description of this entity, several reports did precede this. The first angiogram actually demonstrating a fusiform aneurysm can be seen in Moniz's 1934 work.[13] The first actual case reports come perhaps from Saphir,[14] in 1935, and Adson,[15] in 1942, although there may be even earlier descriptions.[16-20]

Dandy's contribution, then, was not that he was necessarily the first to report this entity, but rather that he was the first to describe it in detail, including a nomenclature. He utilized the nomenclature of an earlier author, calling the entity "serpentine" aneurysm; hence the "S" aneurysms of Dandy. Others refer to this, when in the posterior circulation, variably as the "megadolichobasilar artery," "basilar ectasia," or "arteriectasis."[21-23]

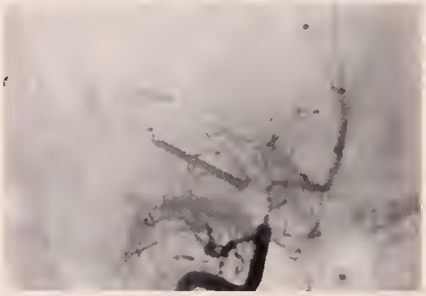

FIGURE 6. Postoperative angiogram, internal carotid artery injection. There is no filling of the aneurysm, and the lenticulostriate vessels are well visualized.

According to Drake,[24] Moffie,[25] and others, fusiform aneurysms affecting the anterior and posterior circulations represent two distinct epidemiologic entities. These authors suggest that posterior circulation lesions are generally seen in older patients, in which case arteriosclerosis may be a greater contributing factor; whereas the anterior lesions can be more frequently seen in a younger population in which congenital factors may be related. As previously stated, most authors report a greater incidence in the vertebrobasilar system, although Yu et al[26] found the reverse in their substantial series. The same was true of the report from Hulten-Gyllensten et al.[23] These lesions may be seen in association with Marfan's syndrome,[27] Ehlers-Danlos syndrome,[28-30] tuberous sclerosis,[31,32] arteritis,[33] nonsyphilitic aortitis,[34] Moyamoya disease,[35] and other connective tissue disorders,[3] further supporting a congenital etiology. In the celebrated study of the pathology of fusiform aneurysms by Sacks and Lindenberg,[36] it was found that the media and smooth muscle layer were frequently deficient of fibers and incomplete in their formation. However, in contrast to the pathology in saccular aneurysms, the medial element deficiency is more diffuse and haphazard, but without the discrete focal deficiency of these elements seen in saccular aneurysms. These impressions were furthered by the report from Azzarelli et al,[37] describing a case of fusiform aneurysms that appeared five years after radiation therapy for a pineal germinoma. Although this appears to have been the first case of such lesions occurring intracranially after irradiation, it is well known that these lesions can occur elsewhere in this setting.[38,39] It is also generally accepted that radiation therapy injures blood vessels, particularly the medial elements.[40,41]

The clinical presentation is most often secondary to mass effect.[1,3-6,10,12,15,21-23,25,26,36,42-46] These symptoms may include headaches, cranial neuropathies, seizures, visual impairment, and even focal deficits such as hemiparesis. Subarachnoid hemorrhage is reported to occur in a smaller percentage of cases. Embolization is also thought to occur from these lesions, both in the anterior[1,47] and posterior[48] circulations. The patient described in this report presented with subarachnoid hemorrhage, but the initial CT scan also showed evidence of an old infarct in the distribution of the MCA. As this was proximal to the aneurysm, this was not consistent with previous embolization but rather with occlusion of lenticulostriate arteries consequent to proximal expansion of the aneurysm.

Altogether, there have been approximately 120 cases of dolichoectasia of the anterior cerebral circulation reported in the literature (Table I).

The therapeutic rationale for these lesions must provide protection from both ischemic complications as well as subarachnoid hemorrhage, in the setting of a lesion that cannot be secured in the standard fashion (clipping). Traditionally, "unclippable" aneurysms were treated with hunterian ligation[49,50]; this has been the standard against which other therapeutic approaches have been measured. The risks and complications of this procedure, as well as its efficacy in preventing recurrent subarachnoid hemorrhage, have been documented by a number of reviews.[51,52] A review by Morley and Barr[53] supported the use of this procedure in selected settings; nevertheless, subsequent long term follow-up reviews on such patients, including reports by Winn et al,[54] German and Black,[55] Mount,[56] and others,[57–59] have all demonstrated that not only do ischemic complications ensue, but that this does not confer complete protection against recurrent hemorrhage.

TABLE I. Reported Cases of Anterior Cerebral Circulation Dolichoectasia

Author, Year, Reference No.	Number of Cases
Saphir, 1935[14]	3
Adson, 1942[15]	2
Tassman, 1944[44]	1
Dandy, 1947[12]	6
Taptas, 1948[43]	7
Ley, 1950[42]	5
Yaskin & Schlezinger, 1952[89]	1
Housepian & Pool, 1958[90]	5
Courville, 1962[8]	47*
Bladin & Donnan, 1963[91]	1
Mitts & McQueen, 1965[45]	4
Hilton & Hoyt, 1966[92]	1
Moffie, 1968[25]	1
Sacks & Lindenburg, 1969[36]	34
Todorow & Niessen, 1974[93]	1
Ammerman & Smith, 1977[78]	1
Pia, 1979[94]	2
Samson et al, 1980[83]	1
Bushe & Bockhorn, 1980[85]	1
Goldstein & Tibbs, 1981[5]	1
Little et al, 1981[1]	11
Brambilla et al, 1982[70]	1
Yu et al, 1982[26]	23
Azzarelli et al, 1984[37]	1
Nakasu, Saito, Handa, 1984[4]	1
Tognetti et al, 1984[74]	1
Yasargil, 1984[95]	1
Yamada et al, 1985[35]	1

* In this paper, Courville reports 47 patients with both anterior and posterior ectasia but does not delineate between them.

Of course, these studies included saccular as well as fusiform lesions, making extrapolation of this information to the incidence of recurrent hemorrhage in the setting of the patient presented here somewhat conjectural. The data regarding ischemic complications are probably more applicable. A number of procedures have been proposed to help better define the suitability of operative candidates for ligation, including the Matas test,[60] intraoperative electroencephalographic (EEG) monitoring, preoperative cerebral blood flow (CBF) studies with various isotopes, and preoperative angiographic evaluation for cross-filling. Most notably, Miller and associates[61] have reported criteria utilizing a combination of cerebral blood flow with internal carotid artery pressure, which attempt to predict which patients will tolerate carotid ligation. Using these criteria, approximately 20% of patients will not be suitable candidates for ligation. Weir[62] points out, however, that although these criteria are frequently cited, their accuracy has yet to be validated in a prospective fashion. Nishioka,[63] in reporting the results of the Cooperative Aneurysm Study with respect to this issue, stated that there was a 27% rate of immediate or early (first four days) ischemic complications with this procedure. In further analyzing these data, he showed that the incidence was less with common carotid occlusion than with cervical internal carotid occlusion. To raise yet another concern, Nishioka stated that the incidence of ischemic complications was less with gradual occlusion than it was with an abrupt procedure. The issues of long term ischemic complications were addressed by this study, as well as several other authors, including Odom and Tindall,[64] Roski et al,[65] and Oldershaw and Voris.[66] The risk of long term ischemic complications of ligation appears to be between 10% and 20%.

Thus, it is conceptually attractive to consider cerebral circulatory augmentation in any setting in which ligation or trapping may become necessary. These issues are amplified when MCA occlusion is considered. In contrast to ICA occlusion, there is far less collateral circulation and a greater likelihood of ischemic complications. Whether extracranial-intracranial (EC-IC) bypass surgery, and in particular a deep STA-MCA bypass, reduces ischemic complications in this setting remains speculative.

The use of bypass procedures as part of the surgical therapy for giant or unclippable aneurysms was introduced by Yasargil in 1969.[67] Several other authors have reported on small series of patients using this procedure, usually in conjunction with carotid ligation.[68–72] In reviewing 65 cases in which EC-IC bypass was performed in association with occlusion, Peerless and Hampf[73] reported successful outcomes in 57 cases. In this study, a variety of techniques were used to occlude parent vessels, including carotid ligation, balloon occlusion after the establishment of EC-IC bypass, and a "Drake" tourniquet applied to the MCA. The tourniquet technique again offers the advantage of permitting controlled closure of the parent vessel while observing the patient's neurologic status. Other authors have also reported this approach.[74] This may appear to be the natural evolution of surgery for these lesions, but other issues are raised by this approach. In particular, one concern that may be more theoretical than real questions the possibility that establishing a bypass without immediate exclusion of the aneurysm from the circulation may result in altered hemodynamics ultimately culminating in aneurysmal hemorrhage. Aneurysmal rupture after the establishment of an EC-IC bypass was reported by Hopkins and Grand[75] in one of 11 patients treated with the combination of bypass and ligation. This prompted these authors to support a strategy of bypass and immediate ligation. Scott et al[76] also reported on a patient with a previously unruptured giant aneurysm who suffered a fatal hemorrhage 13 days after an EC-IC bypass was performed. Since it is an oft-quoted notion that these lesions do not frequently bleed, some surgeons may find this to be less of a concern. As previously stated, however, these lesions in fact can bleed more than is commonly thought; more importantly, the subject of this case report presented after subarachnoid hemorrhage. This made us concerned about performing a bypass without reducing the arterial flow to the lesion in some way.

Diaz et al[77] have reported a series of 13 patients in whom EC-IC revascularization was performed to a main limb of the MCA, rather than the standard fashion of utilizing one of the cortical branches. These authors postulated that this provides a large-caliber anastomosis which immediately restores flow into the MCA tree. The disadvantages cited by the authors, including temporary occlusion of a main branch of the MCA and retraction on the Sylvian fissure, were already required for treatment of the aneurysm in the case reported here. Therefore, we felt that this "main branch" anastomosis would reduce the likelihood of a postoperative ischemic complication, considering the increased blood flow such an anastomosis would potentially provide. Thus, we felt more comfortable with immediate exclusion of the aneurysmal portion of the MCA, rather than performing exclusion in a delayed fashion with a balloon or a Drake tourniquet.

The first report of intracranial trapping and exclusion of an aneurysmal M-1 segment with simultaneous EC-IC bypass appears to have been that of Ammerman and Smith in 1977.[78] The patient suffered a postoperative hemiparesis which cleared gradually over the two subsequent months. Several others report the use of intracranial trapping with EC-IC bypass, with generally acceptable results.[79,80] There are, of course, reports of failure of this procedure due to thrombosis of the proximal vessel,[81,82] such as occurred in this case, but this appears to be uncommon. For this reason, as well as the limited clinical experience in general with this situation, it is unclear how aggressive one should be in terms of long term anticoagulation. The patient was initially discharged on aspirin; whether the episode of thrombosis which occurred two months after surgery would have been avoided had warfarin been used initially is at best conjectural and awaits more clinical data. Also, Samson and Neuwalt[83] reported a case in which EC-IC bypass failed to protect against ischemic postoperative deficit.

Other authors have reported on a variety of other approaches. Segal and McLaurin[84] reported the excision of two such lesions. This was also reported by Bushe and Bockhorn,[85] who also performed EC-IC bypasses in two patients. Sugita and associates[86,87] reported treating these lesions with the use of multiple angled fenestrated clips, thus reconstructing a lumen and excluding the "aneurysmal" portion of the wall. This procedure appears to be attractive, but it may present a problem if the "aneurysmal" portions of the wall cannot be clearly identified. Other authors report the use of thrombogenic substances, such as wire,[78] or catheter-guided balloon placement.[88] Given the configuration of this patient's lesion, these were not viable alternatives.

Ultimately, more experience must be gained with these uncommon but difficult lesions before a consensus can be reached concerning their management. With current understanding of cerebral blood flow and the nature of these lesions, current technology favors the use of exclusion in combination with EC-IC bypass in selected cases.

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LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Omental infarction in pregnancy

TO THE EDITOR: Since 1851, some 200 cases of omental infarction have been reported in the English literature.[1] Three have been reported during pregnancy: one at term,[2] one at 34 weeks,[3] and one at three months.[4] The diagnosis of omental torsion is rarely made preoperatively. Right upper quadrant pain secondary to peritoneal irritation mimicking cholecystitis or appendicitis is the most common presentation.

Case Report. The patient was a 26-year-old woman, para 1-0-0-1, who had a bilateral tubouterine implantation in 1985 after confirmation of bilateral cornual closure. Her last menstrual period had been on July 18, 1987; there was confirmation of a dizygotic twin gestation; the estimated date of confinement was April 24, 1988. The patient had an otherwise uneventful antepartum course until February 21, 1988, when she experienced right upper quadrant pain, loose stools, and nausea. Pertinent findings upon examination included tenderness in the right upper quadrant with no rebound, a twin intrauterine gestation compatible with 32 weeks, a normal fetal heart rate (FHR) x 2, a closed cervix, and a trace of protein in the urine. On admission, the only positive finding was that of extreme periumbilical tenderness, along with a 3 x 4-cm tender mobile mass that appeared to be contiguous with the dome of the bladder. The patient was observed with dual monitors and bled slightly with uterine cramping. A sonogram and biophysical profile were obtained, showing no sign of abruptio placentae or placenta previa and +10 ratings on both 32-week infants. The bleeding increased along with increasing uterine tenderness and pain in the periumbilical area. Cesarean section was performed with delivery of twin girls weighing 3 lb 1 oz and 2 lb 14 oz, respectively. There was a 30% abruption of the placenta. A 5 x 4-cm purple infarcted and twisted piece of omentum was found on the anterior aspect of the uterus, which was resected. The postoperative course was uneventful, and the patient was discharged on the third postoperative day. Both children and mother are doing well today.

Discussion. The pathophysiology of omental torsion with resulting infarction is still not fully understood. Venous congestion, Valsalva maneuver, trauma, sudden body position change, overeating, and infective or inflammatory foci have all been suggested as inciting factors along with omental malformations.[5]

Perhaps the end result of torsion with secondary infarction of smaller portions of omentum may be the pebble-like concretions occasionally found incidentally at the time of exploratory laparotomy. This entity must be thought of anytime one investigates the acute abdomen with periumbilical pain.

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Thallium stress tests and the White House security system

TO THE EDITOR: A report was recently published[1] of a patient who received thallium-201 and for the next eight days continually triggered a bank radiation detector. We describe a patient who set off a radiation monitoring device more than two weeks after an injection of thallium.

A 59-year-old woman received 2.7 mCi of thallium-201 chloride as part of a stress test for the evaluation of atypical chest pain. Fifteen days later, she visited the White House in Washington, DC. Alarms went off after she passed through what she thought was a metal detector at the visitors' entrance. This five-foot tall, 149-pound grandmother was immediately surrounded by several security personnel with gun holsters opened. She was informed that she had set off a sensitive radiation detection device, and one of the security agents asked her when she had her stress test, suggesting that this was not an isolated event.

This incident reveals that sensitive radiation detectors can be triggered more than two weeks after a thallium injection. We are concerned that the shock of setting off alarms and being accosted by security personnel could be detrimental to patients with significant coronary artery disease,[2] especially if they are unaware of the potential for these situations.

The radionuclide vendor has informed us that the assay of the thallium given to this patient contained 98.5% thallium-201 and 1.2% thallium-202. After fifteen days of decay, there is still approximately five times more thallium-201 (T ½=73 hours) than thallium-202 (T ½=12.23 days). We suspect that the thallium-201 was probably responsible for setting off the detector.

We strongly agree with the recommendation of Levin and Fischer[1] that patients be advised of the protracted potential for setting off radiation detectors after nuclear studies using relatively long lived radionuclides.

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Primary meningococcal conjunctivitis

TO THE EDITOR: Primary acute purulent conjunctivitis caused by *Neisseria meningitidis* is apparently unusual, but may be associated with serious medical and social consequences if the diagnosis is not established early and accurately. This report describes an eight-year-old girl with primary meningococcal conjunctivitis.

Case Report. An eight-year-old Vietnamese girl was seen with bilateral purulent conjunctivitis of two days' duration. The inflammation was first noted as redness of the right conjunctivum, with involvement of the left eye after 24 hours. Thick, purulent discharge increased and lid swelling progressed to near closure of the palpebral fissures. Photophobia was present from the outset. There was no fever, headache, or other complaints.

Physical examination revealed a well-developed, alert, afebrile child in moderate distress from ocular discomfort. The palpebral fissures were all but closed by lid swelling, with yellowish, thick, purulent discharge, moderate in amount, and marginal crusting. The bulbar conjunctivae were injected, the corneae were clear. Ocular movements and pupillary reflexes were normal.

The palpebral conjunctivae were bright red and chemotic. No preauricular nodes were palpable. The neck was supple, and the ears, nose, throat, and lungs were normal.

Laboratory findings included a white blood cell count of 10,700/mm^3, with a differential of 67% segments, 5% band forms, 20% lymphocytes, and 6% monocytes. The blood glucose and serum electrolyte levels were normal. Blood culture was negative. Gram-stained smear of the exudate showed many polymorphonuclear cells with gram-negative diplococci seen intracellularly and extracellularly. The presumptive diagnosis was gonococcal conjunctivitis, but the final culture report was *N meningitidis*, type B, beta-lactamase negative, sensitive to penicillin-G and erythromycin.

The child was treated with hourly normal saline ocular irrigation, erythromycin ophthalmic ointment, and penicillin-G, 250,000 U intravenously every six hours. She remained afebrile and alert. On day two, the swelling and discharge were significantly diminished and she was able to open her eyes to about 1 cm.

The patient's condition continued to improve and she was discharged in good condition to pediatric and ophthalmologic follow-up. Oral rifampin was prescribed for family members and the patient following discharge.

Discussion. Some 40 cases of primary meningococcal meningitis have been reported in the medical literature, the majority occurring in children.[1] The onset is usually unilateral, but may become bilateral. Clinically, the infection is not distinguishable from gonoccoccal conjunctivitis. In 10% of the cases, systemic infection (sepsis, meningitis) follows the conjunctivitis.[2,3] Dillman[4] reported the case of a child in whom fulminant meningitis developed after three days of treatment with topical sulfacetamide ointment. Nussbaum et al[5] described a seven-month-old infant with meningococcal meningitis which developed three days after the onset of conjunctivitis. A case of ophthalmia neonatorum caused by *N meningitidis* has been reported in a five-day-old infant.[6] Other complications include corneal ulceration, residual corneal opacity, endophthalmitis, and meibomitis.[7,8] In view of the present and past experience, *N meningitidis* must be included in the differential diagnosis in infants and children with acute purulent conjunctivitis and Gram's stain of the exudate should be examined as soon as possible. If gram-negative diplococci are seen on stain, systemic and topical antibacterial treatment that is effective against both the meningococcus and gonococcus should be started and appropriate precautions instituted to protect contacts. The systemic drug should be administered intravenously. Finally, it should not be presumed that the presence of gram-negative diplococci indicates gonococcal infection. No action of a sociolegal nature should be initiated until the definitive culture report is available.

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Caring and serving

TO THE EDITOR: In the modern era, a medical society's membership committee has to bring to the task a different perspective and thrust. Membership is no longer a given. Physicians no longer clamor to join. They must see the organization as a caring and serving society: caring in the sense that we must be aware of their needs; serving in that we exist to meet those needs.

For those senior physicians who are planning to retire, the Medical Society of the State of New York's membership committee has designed programs geared to easing the transition. For young physicians, we have developed a plan for a Lake Placid weekend to answer some of their pressing concerns (when they are not enjoying the ski slopes). Other programs are tailored to medical students and to welcoming newly arrived residents to New York. Those physicians who are contemplating changing their type of practice have had a program to inform them of their options.

We have been concerned about the physician who is going through a medical liability lawsuit. With MSSNY staff members we have initiated and supported a program of peer support. This enables such a physician to have a peer to listen to and to encourage him or her. We are also sensitive to the Auxiliary's effort to maintain the viability of the family structure during those difficult times.

We are planning to survey our members in a comprehensive fashion so that we will have a better grasp of their interests. We do not know, for example, what their hobbies are. We need to discover fun things that we can do together.

We have to find various means to support our county societies in their membership endeavors. Theirs is a difficult and unsung task. Our resources must continue to assist them and we are exploring initiatives to expand our commitment to them.

If in the process our efforts meet with your approval, the members of the membership committee, the staff, and yours truly will have achieved the ultimate accolades, the positive responses of satisfied members.

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HIV Partner/Contact Notification Assistance Programs

The New York State Department of Health and New York City Department of Health endorse voluntary partner notification and have established programs to assist HIV-positive individuals in notifying their recent sexual and/or needle-sharing partners. For areas outside New York City the program is known as the New York State Partner Notification Assistance Program (NYS P-NAP). In New York City the program is designated as the Contact Notification Assistance Program (CNAP). These programs seek to locate, counsel and offer HIV testing to partners of confirmed HIV-positive individuals, a group at high risk of HIV infection.

Partner Notification Assistance Program

Local health departments outside New York City provide notification services within the context of pre-existing HIV counseling and testing services offered by sexually transmitted disease (STD) clinics located throughout the state. P-NAP provides the following services:

- information to HIV-positive individuals regarding partner notification services;
- counseling to encourage and enable HIV-positive individuals to notify and refer sexual and/or needle-sharing partners;
- assistance to HIV-positive individuals to complete the partner notification process;
- services to locate and notify partners of HIV-positive individuals;
- HIV risk reduction and prevention information to all STD patients.

Every reasonable effort is made to locate, notify and offer counseling and testing opportunities to those individuals who may have been exposed to the HIV virus. This process begins during the counseling session with an HIV-positive patient. Patients are urged to notify their partners directly and are provided with information, guidance and the skills needed to notify partners sensitively and effectively. If the patient prefers to have the counselor make the notification, it then becomes the counselor's responsibility to obtain a complete and accurate description of the partner, to locate information to expedite the partner notification process and to ensure that the correct partner is notified. The partner's name, nicknames, age, race, sex and marital status are obtained, as well as a physical description including approximate height, weight, hair appearance, facial hair, glasses, complexion and other visually identifying characteristics.

The counselor obtains at least one additional item of locating information: work address, work telephone number, places where the partner spends time and the name of an individual familiar with the partner's whereabouts. Finally, the patient is asked if the partner lives with another individual and the best time and place to make the notification. All notifications are made with strict confidentiality. Under no circumstances is the identity of the index person given to the partner notified.

Standard STD investigative techniques are used to locate and notify named partners with the following important exception: notifications must be made in person. Only one first person notification with the partner is attempted in order to conduct a counseling session. If notification or counseling is not completed, the counselor leaves a telephone number with the partner. The partner is then advised to call the counselor to obtain further information. The telephone and mail may only be used to set up an appointment to speak with the partner in person. No more than two first person telephone contacts take place.

The partner notification system accepts referrals from three different provider categories: STD clinics, anonymous test sites and a third category of all other providers. This third category includes family planning programs, prenatal care assistance programs (PCAP), private physicians, tuberculosis clinics, drug treatment programs, blood banks, hospitals and community health centers. In each of these settings, there are three universal goals for P-NAP:

- All patients with a positive HIV test result must be made aware of the existence of the partner notification assistance program and given the P-NAP brochure.
- All health care providers providing HIV counseling and testing services must be made aware of the procedure for referring patients in order to expedite the partner notification process and minimize trauma to the patient.
- All referrals must be made in a manner that allows the P-NAP counselor to confirm the patient's positive test result. No partner notification efforts can be undertaken until a positive HIV test is confirmed.

The key to the implementation and success of partner notification in all settings is the emphasis given in pre- and post-test counseling sessions. Patients requesting P-NAP services should be referred to the local health department for a partner notification counseling session.

The medical provider should immediately (or as soon as possible) call the local health department P-NAP program to set up a counseling session. Some identifying information regarding the patient (usually the name) will be

Partner Notification—Referral Telephone Numbers

County	Telephone Number
Albany County Health Department	(518) 447-4640
Allegany	(716) 268-9250
Broome	(607) 772-2839
Cattaraugus	(716) 375-4121
Cayuga	(315) 253-1301
Chautauqua (Jamestown)	(716) 488-0755
Chautauqua (Dunkirk)	(716) 366-8805
Chemung	(607) 737-2528
Chenango	(607) 335-4606
Clinton County Health Department	(518) 565-3270
Columbia County Health Department	(518) 828-3358
Cortland	(607) 753-5036
Delaware County Nursing Service	(607) 746-3166
Dutchess	(914) 431-2071
Erie	(716) 846-7853
Essex County Nursing Service	(518) 873-6301
Franklin County Nursing Service	(518) 483-5100
Fulton County Nursing Service	(518) 762-0720
Genesee	(716) 344-2580
Greene County Nursing Service	(518) 943-5201
Hamilton County Nursing Service	(518) 548-4281
Herkimer	(315) 867-1176
Jefferson	(315) 782-9289
Lewis	(315) 376-5453
Livingston	(716) 658-2866
If Busy	(716) 658-2757
Madison	(315) 366-2361
Monroe	(716) 274-6166
Montgomery County Nursing Service	(518) 853-3531
Nassau	(516) 535-3280
Niagara	(716) 284-3114
Oneida	(315) 798-5747
Onondaga	(315) 425-3252
Ontario	(716) 396-4343
Orange	(914) 562-5832
Orleans	(716) 589-7004
Oswego	(315) 349-3568
Otsego County STD Clinic	(607) 547-3391
Putnam	(914) 225-2115
Rensselaer County Health Department	(518) 270-2640
Rockland	(914) 354-0200 X2547
St Lawrence Planned Parenthood	(315) 386-8821
Saratoga County Nursing Service	(518) 584-7460
Schenectady City Health Department	(518) 382-5038
Schoharie County Health Department	(518) 295-8174
Schuyler	(607) 535-2704
Seneca	(315) 539-9294
Steuben	(607) 776-9631
Suffolk	(516) 348-2748
Sullivan	(914) 292-5910
Tioga	(607) 687-0623
Tompkins	(607) 335-4606
Ulster	(914) 338-8443 X230 & 278
Warren County Nursing Service	(518) 761-6415
Washington County Nursing Service	(518) 747-0621
Wayne	(315) 946-5749
Wyoming	(716) 786-8890
Westchester	(914) 241-3900 X6251
Yates	(315) 536-4401

required in order to identify the patient upon arrival. A signed release specific for HIV must be obtained and filed with the patient's record. This system allows the P-NAP counselor to call the provider back in order to confirm any questionable information or to obtain new information.

The patient should be told to be prepared to provide the kind of locating information that will be necessary to successfully complete partner notification. This information includes full name, address, telephone number, hangouts, living situation, best time of day to notify partners and distinguishing physical characteristics of partners.

The P-NAP program was initiated in March of 1989. During the months of March and April, a total of 15 individuals received counseling through the P-NAP system. P-NAP counselors obtained the names of 20 partners from 14 of those patients who received counseling; 14 partners were notified by P-NAP counselors.

Contact Notification Assistance Program

Within New York City, a similar contact notification activity is underway. CNAP provides consultation with health care providers on contact notification counseling protocols, skills and strategies. Patients can also access CNAP directly for information about contact notification counseling. Health care providers and patients may call to request that a public health officer notify contacts. To make referrals or to obtain more information about the New York City Department of Health's CNAP activity call (212) 340-4439.

Hemolytic uremic syndrome and verotoxin producing *E coli*

Hemolytic uremic syndrome (HUS) and thrombotic thrombocytopenic purpura (TTP), a related illness with overlapping clinical signs, have been associated with a variety of infectious and noninfectious agents.[1,2] Some of the agents include shigella, campylobacter, pneumococcus, verotoxin-producing strains of *E coli* (VTEC) as well as oral contraceptives, mitomycin and cyclosporine.[1–3]

HUS is an acute illness characterized by microangiopathic hemolytic anemia, thrombocytopenia and renal failure. TTP shares these clinical features and is also accompanied by fever and neurologic symptoms. While HUS may be associated with multiple agents, a significant portion of cases are related to bacterial enteric pathogens, which may necessitate public health intervention.

Clinical Aspects of HUS

The triad that composes HUS consists of hemolytic anemia, thrombocytopenia and acute renal failure. There is microangiopathic hemolysis characterized by falling hemoglobin, the presence of microspherocytes and schistocytes on peripheral smear and by a low platelet count. Renal failure is manifested by oliguria or anuria. Urinalysis shows hematuria and proteinuria and red blood cell casts. Blood chemistries show elevated serum creatinine, blood urea nitrogen, potassium as well as metabolic acidosis.[4–6]

Clinically, patients with HUS appear seriously ill; they are pale, or sometimes jaundiced and often hypertensive. Other organ systems may be involved including the gastrointestinal tract (mimicking ulcerative colitis) and the cardiovascular and central nervous systems. This involvement can lead to myocardial decompensation (heart failure), seizures, coma, hypertensive encephalopathy and can exacerbate renal failure.

The pathogenesis of HUS related to enteric pathogens appears to be associated with a toxin (such as the verotoxin produced by *E coli* O157:H7) which damages endothelial cells[3] triggering the clotting mechanism. The resulting microthrombi partially or completely occlude capillaries of the kidneys and other organs.[1,2] Generally, children with HUS respond well to standard supportive therapy for acute renal failure.[1,4 6] In specific instances, plasmapheresis or plasma infusions may be beneficial. Morbidity and mortality are considerably higher in adults, particularly in the aged.

Role of E coli

Although a number of inciting agents have been associated with HUS, VTEC have attracted recent attention due to a number of HUS outbreaks where this particular agent has been implicated. Outbreaks have been described in children in day care centers, households, nursing home patients, patrons of a fast food hamburger chain and patients in institutions for the mentally retarded.[7-11] Ground beef and raw milk have been implicated as food vehicles in several outbreak investigations and dairy cattle have been suggested as the animal reservoir for this organism.[12] Person-to-person transmission via the fecal-oral route has also been described.[13]

Exposure to one particular strain of VTEC referred to as *E coli* O157:H7 has recently been described in the literature, in association with HUS, TTP and hemorrhagic colitis. Exposure to *E coli* 0157:H7 may result in illness ranging from mild, loose stools to bloody diarrhea, HUS or TTP. In one study, 75% of patients with idiopathic HUS had evidence of VTEC.

Laboratory Aspects

While laboratory identification of *E coli* is relatively simple, identification of verotoxin producing strains is more difficult.[3] Methods involve a tissue culture technique plus use of specific antisera for confirmation and is not routinely performed by most clinical laboratories. Stool cultures for VTEC and assays for presence of free verotoxin can be performed by the New York State Department of Health Wadsworth Center for Laboratories and Research (WCL&R). Since *E coli* O157:H7 tends not to ferment sorbitol, local laboratories may screen *E coli* isolates using MacConkey's sorbitol media and submitting suspicious colonies to WCL&R for confirmation. Stools obtained within the first six days of illness will have the highest yield.

Control and Future Plans

The Bureau of Communicable Disease Control of the New York State Department of Health is cooperating with the US Centers for Disease Control in a multicenter study to obtain additional epidemiologic data regarding the association between HUS and *E coli* 0157:H7. Several teaching hospitals in New York State are participating. The purposes of the study are:

- to determine the proportion of HUS cases that are associated with VTEC;
- to improve laboratory diagnosis of VTEC;
- to identify geographic clusters that could represent common source exposures (outbreaks).

While individual cases of HUS or hemorrhagic colitis are not reportable, any cluster or outbreak should be reported to the Bureau of Communicable Disease Control at (518) 474-3186. In addition, people with bloody diarrhea whose stools are negative for common enteric pathogens (salmonella, shigella, campylobacter) may be screened in local laboratories to identify suspect VTEC (*E coli* colonies which do not ferment sorbitol). Isolates may be submitted to WCL&R for confirmation.

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Childhood central nervous system tumors

Central nervous system (CNS) tumors are an important component of childhood malignancies, ranking second only to leukemia among cancers afflicting children. The most frequent CNS tumors are various types of brain cancer. Although these tumors are a prominent cause of morbidity and mortality, their etiology is not well understood. Previous clinical studies have suggested an association between the occurrence of CNS tumors in children and neurofibromatosis. Type 1 neurofibromatosis (von Recklinghausen's disease) is a genetic condition characterized by small discrete pigmented lesions (cafe au lait spots) which often appear in infancy or early childhood. These can develop into multiple subcutaneous neurofibromas (tumors) that may slowly increase in size and number. It may develop into a seriously disfiguring and debilitating disease, especially if the neurofibromas compress important organs. Neurofibromatosis is popularly identified as the disease dramatized in *The Elephant Man.*

The Bureau of Cancer Epidemiology conducted a population based case-control study with 338 patients, less than 15 years of age, who have been diagnosed with a primary tumor of the central nervous system from January 1968 through December 1977 in 53 New York State counties. The study included 676 controls selected from birth certificate files of the New York State Department of Health. The study explored several factors which may be related to CNS tumor risk. Neurofibromatosis was among the possible factors examined, as well as other conditions in the children and their families. Information was obtained on neurofibromatosis, congenital anomalies and seizures in study subjects, their siblings and parents by telephone interview with the mother of each case and control. Supplemental information was collected on neurofibromatosis in the patients and their families from hospital medical records.

This study confirmed the strong association of neurofibromatosis with CNS tumor risk. Thirteen cases were found to have neurofibromatosis while none of the controls had the disease. Seven of the thirteen cases with neurofibromatosis also had optic gliomas. This is consistent with previous clinical observations of the tendency for neurofibromatosis to occur in conjunction with optic gliomas in young children. None of the patients in this study exhibited bilateral acoustic neuroma, characteristic of the rarer type 2 neurofibromatosis. Two fathers and three mothers of cases had neurofibromatosis; five cases had siblings with neurofibromatosis. None of the parents or siblings of controls had neurofibromatosis. These findings yielded relative risk estimates of infinity, which were statistically significant for cases compared to controls and for siblings. The relative risk estimate was not found to be significant for parents.

A relative risk of 4.5 for seizures was observed in the patients. This was expected, as seizures are often a presenting symptom of CNS tumors. Seizures were also more common among the parents of CNS tumor cases than controls, however, the excesses for parents were not statistically significant.

There was no association of congenital anomalies with the risk of CNS tumors when cases were compared to controls. There was a nonsignificant increased relative risk (1.4) for those having a sibling with a congenital anomaly, but this was reduced to 1.1 when controlling for the number of siblings.

The results of previous research and this study suggest a strong role for genetics in the causation of some childhood CNS tumors. More research with large case groups incorporating chromosome studies and detailed family histories will help to clarify the causal mechanisms. The data strongly support the recommendation that the presence of CNS tumors, especially optic gliomas, be explored in individuals with neurofibromatosis and that patients with optic gliomas be evaluated for manifestations of neurofibromatosis.

The information for this article was drawn from a detailed report of the study which was recently published (Baptiste MS, Nasca PC, Metzger BM, et al: Neurofibromatosis and other disorders among children with CNS tumors and their families, *Neurology* 1989; 39:487–492).

UNLOCKING THE FAMILY DOOR: A SYSTEMIC APPROACH TO THE UNDERSTANDING AND TREATMENT OF ANOREXIA NERVOSA

By Helm Stierlin, MD, and Gunthard Weber, MD. 256 pp, illustrated. New York, Brunner/Mazel Publishers, 1989. $27.50 (hardcover)

In this book, Drs Stierlin and Weber present a challenging picture of their technique for the treatment of anorexia nervosa. Both authors are physicians and well qualified, with many years of hospital and private practice experience as family therapists.

Since their assumption is that the underlying pathology which exists within the family is responsible for the anorexia, therapy is directed at the family rather than specifically at the patient. Treatment is limited, and may consist of one to a maximum of ten conjoint family sessions, with emphasis on interpersonal rather than intrapsychic processes. In the systemic approach, therapy is based on the findings of cybernetics, systems control theory, information and communication theory, and game theory. It should also be mentioned that the responsibility for change or nonchange rests squarely with the family. The goal is a reorganization of the family structure that will provide greater individuation, and result in the patient's recovery.

The authors recognize that the other types of therapy—such as analytic, cognitive and behavioral, nonanalytic psychotherapy, and hospital or residential care—are also utilized in the treatment of anorexia. They make no claim that systemic family therapy is a panacea, but are interested in presenting their experiences which they consider successful. It is a therapy designed to be relatively short-term, and the guiding principle is to provide stimuli rather than to work things through jointly. The medical model of illness, however, is rejected, and replaced by the hypotheses founded on a cybernetically based epistemology.

The chapters are all carefully organized and the history of each patient and family is carefully detailed. This material is presented in a direct and straightforward style which may almost create for the reader a feeling of being present at an interesting lecture. There is also a valuable bibliography as well as a name and subject index. In summary, even if one does not agree with the authors, one will find their book challenging and thought provoking.

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MOODSWING

Revised and Expanded Edition. By Ronald R. Fieve, MD. 276 pp. New York, William Morrow and Co, 1989. $22.95 (clothcover)

This book is a revised edition of a book originally published in 1975. The book is aimed at the layman and should be considered in that light. The book's central thesis is that lithium is a valuable and necessary chemical agent for the treatment and prevention of recurrent mood disorders. Dr Fieve cites many moving case examples from among the more than 10,000 patients he has treated in his own practice. He also discusses evidence for mood swing disorders in creative people, successful businessmen, and politicians. The majority of the book deals with the treatment of bipolar disorders but he also describes the value of lithium in the management of recurrent unipolar depressions. Dr Fieve notes that for many years American psychiatrists failed to differentiate mood disorders from schizophrenia, in contrast to British psychiatrists, who more frequently made the correct diagnosis. This situation persisted as long as there was no difference in the treatment of the two disorders. He notes that since the general introduction of lithium in the 1970s, this situation has changed.

Lithium is now regarded as an essential component in the treatment of bipolar mood disorders. Dr Fieve envisions that lithium will play a significant role in the treatment of various other conditions including drug abuse, alcoholism, gambling, and the control of aggressive outbursts and behavioral disorders in children and adolescents. He discusses the possibility that lithium may be of value in the prevention of heart disease and the treatment of premenstrual syndrome (PMS) and some kinds of cancer. He considers studies suggesting that lithium should be added to the water supply but states that he has taken a position against it.

Dr Fieve writes about a very important subject, the treatment of mood disorders, a subject about which it is important for the general public to be better informed. Unfortunately the book is written with a combined contentious and evangelical tone. In spite of a specific statement that lithium is not a panacea, it is easy to see how one could receive the opposite impression from this book. The jacket of the book announces that this "eminent psychiatrist . . . reveals a revolutionary new way to prevent depression." The revolution he is revealing is lithium. Dr Fieve describes this as the third revolution in the treatment of mental ills. The first was the work of Philippe Pinel in 1791, changing people's attitudes towards the mentally ill, resulting in more humane treatment, and the second was the work of Sigmund Freud. The book frequently extols Fieve's own work, giving the book the flavor of a marketing tool for him and his Clinic-Foundation.

The book contains a series of loosely related chapters. In the third chapter, "Moods and Creativity," Dr Fieve discusses madness and creativity. He believes there is some unclear relationship between the two but he is surer there is a relationship between creativity and manic-depressive illness. In the fourth chapter, "Wall Street Wizards and the Midas Touch," he discusses manic behavior and business. Throughout, he ventures into popular social commentary, explaining corporate conglomerates as the "epitome of the manic art form." In chapter 8, "Moods and Great Men," he discusses the lives and careers of Abraham Lincoln, Winston Churchill, and Theodore Roosevelt. The lives of these three men make very interesting reading and Dr Fieve is able to deal with them in one chapter because his focus is on the signs and symptoms of manic-depressive illness in each of them. I could only wonder where Lincoln and the United States would be if Dr Fieve had been in charge of Lincoln's case. His recommendation: "I would insist on hospitalization . . . " The book does contain important information about depression but it is scattered all about. The book would be more valuable if it contained a better organized discussion about depression, its diagnosis, its signs and symptoms, its disguised expression, and the biochemistry and genetics. Dr Fieve reports that he estimates that a parent with manic-depressive disorder has about a 10–15% likelihood of transmitting it to a child. He says he recommends that a manic depressive avoid marrying another manic-depressive. I am sure the interested reader would appreciate a fuller discussion of such a vital area rather than the one sentence statement given.

Throughout the book there are warnings of the dangers of the wrong treatment which can lead to a "real disaster." Psychodynamic psychotherapy seems to be a red cape waved in front of Dr Fieve. He uses a special phrase for its negative effects, "Psychotherapy toxicity." The side-effects of lithium which he is recommending are clearly underplayed. The index contains only three references to side-effects, two of which state that there are none when used in the correct dose.

Lithium is clearly a very important therapeutic agent. Reifman and Wyatt have estimated that the use of lithium in stabilizing manic-depressive disorder has led to a cost savings of over $400 million yearly in the United States alone. The world psychiatric literature in general supports the value of prophylactic lithium use in recurrent unipolar depressive illness. Dr Fieve could have better contributed to the major effort needed to reach individuals suffering from the spectrum of depressive disorders who remain unrecognized or misdiagnosed with a better balanced book.

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THE TREATMENT OF PSYCHIATRIC DISORDERS

By William H. Reid, MD. 414 pp. New York, Brunner/Mazel Publishers, 1989. $35.00 (hardcover)

This is an excellent treatment handbook which supplements the current American Psychiatric Association's Diagnostic and Statistical Manual-III-Revised. The author aptly describes this volume as a "guide, not a reference text."

In this revised edition of a book published in 1983 as a therapy guide to the DSM-III, the author has not only updated it to complement DSM-III-R, but has expanded on the subjects previously discussed. In the preparation, several thousand references were reviewed, especially those dated since 1983, and nearly 1,000 have been listed as references for readers who seek further details on any subject discussed in this edition.

In its Diagnostic Manual, III and III-R, the American Psychiatric Association defined and described psychiatric disorders much more accurately than had ever been done before. This was accomplished by providing extensive discussion and lists of criteria for each diagnostic category, with the requirement that a patient can be given that diagnosis only when a specific number of criteria have been met. This created in DSM-III-R a detailed volume of more than 500 pages. As elaborate as the diagnostic manual may be, however, it suggests nothing regarding treatment. This book by Dr Reid and his colleagues follows the structure of the DSM-III-R and discusses the accepted treatments for each disorder listed. It does so in a very clear, concise fashion. If there is more than one accepted treatment procedure, there is advice as to which ones are considered to be more effective, or it discusses the order in which alternative treatments might be applied.

Dr Reid is very well qualified as the editor and principal author of this book, having already gone through the extensive literature review necessary for the first edition and thousands of more recent articles for this revision. His other books are *Basic Intensive Psychotherapy* and *Psychiatry for the House Officer*. He also has been editor or co-editor of five books on antisocial disorders and such related subjects as assaults and terrorism. He is currently Clinical Professor of Psychiatry at the University of Texas Health Science Center in San Antonio; serves on the editorial boards of five professional journals; has written numerous papers, journal articles, and book chapters; and has edited or co-edited several monographs.

Each of the other three contributors to this book is an expert in his particular subjects. George Bayliss, Professor of Psychiatry at the University of Maryland School of Medicine, did the sections on organic mental disorders and psychoactive substance use disorders. Dr Jerry J. Tomasovic, Clinical Associate Professor of Child Neurology at the University of Texas, San Antonio, contributed the section on sleep disorders; and Dr James S. Wicoff, a child and adolescent psychiatrist, also on the faculty at San Antonio, contributed the section on disorders usually first evident in infancy, childhood, or adolescence.

The importance of this book is its usefulness to the busy clinician. It does not attempt to offer new information unavailable elsewhere. Instead, the authors have compiled with care, and with an eye to practicality, a therapy guide for any and every diagnostic category listed in DSM-III-R. Its value to the psychiatrist or other mental health professional is obvious, but it will also be valuable to the primary care physician, who may have had limited training in the field of psychiatry, but who nevertheless must often deal with a number of psychiatric problems which may occur in his practice.

In addition, the appendices to this edition not only list available psychotropic drugs by generic and trade names, but also provide an extensive list of drug-drug interactions, a subject that should be of particular concern to any physician prescribing psychotropic medications. Many psychotropic drugs have a number of troubling side reactions and yet must often be prescribed for patients also receiving other medications.

This is a book to be used as needed rather than read cover to cover. Nevertheless, it has a very readable style and there are some convenient tables on such subjects as drugs capable of causing delirium, the equivalent doses of neuroleptics, sleep hygiene, the various causes of organic mental disorders, and medications frequently used in the treatment of insomnia.

All in all, it is a very valuable addition to one's professional library.

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DR EDEN'S HEALTHY KIDS: THE ESSENTIAL DIET, EXERCISE, AND NUTRITION PROGRAM

By Alvin N. Eden, MD. 285 pp. New York, Nal Penguin, Inc, 1987. $8.95 (paperback)

Dr Eden's Healthy Kids presents parents with a comprehensive nutrition and feeding guide to promote optimum health and performance in children from birth through adolescence. He dramatically states his case for the Eden Diet Plan by detailing the rather disturbing state of health of our youngest generation.

The average child of today is presented as one exhibiting increased weight and decreased fitness who craves television and junk food and is already setting up multiple health risks for later life. The author offers his Eden Diet Plan as hope for altering this seemingly inevitable course to disaster.

Dr Eden, as a distinguished pediatrician and chairman of pediatrics at Wyckoff Heights Hospital in New York, has many years' experience in dealing with parents and children, and firsthand knowledge of their current life styles and concerns. He has wisely enlisted the assistance of Dr Andrea Boyar, a registered dietitian, to help produce the Eden Diet Plan.

The book is written for the layman but is carefully substantiated and documented with government surveys and medical research. It certainly fulfills a need. In a nutrition/diet book market that is fraught with quackery and promises of quick cures, it is often difficult for the layman to distinguish truth from fiction. In the area of nutrition for children there is little available at all for parents who want to make the effort to start their children off right.

After presenting his case and establishing the need for a conscious, healthful plan of eating and exercise designed for today's and tomorrow's children, Dr Eden goes on to provide a map for "the best way to put your child on the road to lifetime fitness." His six essential guidelines are as follows:

1. Emphasize low-fat, low-cholesterol foods.
2. Cut down on salt.
3. Hold the line on sugar.
4. Include enough iron.
5. Add more fiber.
6. Fight the SOB (sitting on behind) syndrome.

These guidelines are basically in line with those of the American Academy of Pediatrics, the American Heart Association, the American Cancer Society, and the United States Dietary Guidelines. Dr Eden cites numerous references and provides a very convincing argument for his plan.

If he errs at all it is in overstatement. His overconcern for prevalence and effects of subclinical iron deficiency goes far beyond that established by the bulk of scientific evidence. Likewise his concern that children over the age of two be given only low fat milk fails to take into consideration the thin child who is a poor eater and may need the additional calories from fat. In general, however, Dr Eden's guidelines are very reasonable and aimed at assuring present-day as well as long-term health.

After providing his general guidelines Dr Eden addresses each individual stage of childhood, including infant, toddler, preschooler, school-ager, and adolescent. He explains nutritional needs as well as relevant developmental and psychosocial needs. His discussions are clear and very readable. He lays out both basic principles and specific ways to translate principles into practice. His advice includes what foods are best to eat, how to accomplish healthier cooking, and how to establish good eating habits. Weekly menus and snack lists are included for each age group, clearly showing how sound nutritional principles can be incorporated into daily living. Not only the child but the entire family can benefit.

As with nutrition, the discussions on exercise are sound and practical. Again the author has done his homework. Statements are documented and referenced throughout. Individual age groups are addressed and advice includes not only what to do but how to do it. Guidelines are aimed at establishing lifetime habits for optimum health now and later on.

In *Dr Eden's Healthy Kids*, the information is all there. It is practical and stated with clarity and simplicity. The principles are nutritionally and medically sound. The ultimate test lies with the readers. Are they willing and able to take the necessary steps to achieve a healthier diet for themselves and their children? Can they ensure that their children adopt these healthy eating habits and the benefits they provide?

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THE HETEROSEXUAL TRANSMISSION OF AIDS IN AFRICA

Edited by Dieter Koch-Weser, MD, and Hannelore Vanderschmidt, PhD. 203 pp, illustrated. Cambridge, Mass, Abt Books, Inc, 1988. $29.00 (paperback)

In these days of the extraordinarily prolific growth of information about acquired immunodeficiency syndrome (AIDS), one might ask why the medical literature needs a publication about AIDS that presents previously published materials? Dr Koch-Weser (retired Chairman of the Department of Preventive and Social Medicine and Associate Dean for International Programs, Harvard University) and Dr Vanderschmidt (co-director of the Center for Educational Development in Health at Boston University and a faculty member of the Harvard School of Public Health and the Boston University School of Public Health) have compiled 62 studies, reviews, and editorials that chronicle the generally available medical literature in English describing a broad range of seemingly unique issues concerning acquired immunodeficiency syndrome on the African continent. This resource provides the researcher, clinician, social scientist, and interested reader an opportunity to access rapidly information and opinion that "provide[s] a general understanding of the peculiar characteristic of the [impact from the historical perspective of the medical literature of the] AIDS virus in Sub-Saharan Africa . . . The abundant references in each of the papers . . . serve as a guide for further studies," as the authors have intended.

The 280-page paper-bound volume is organized into six general areas categorizing the development of information about AIDS in Africa, under the headings of "General Dimension of the AIDS Epidemic;" "Historical Perspective;" "Heterosexual Transmission and Prostitution;" "Epidemiology, Serology, and Virology;" "Relationship to Other Diseases and Conditions;" and "Social and Cultural Factors." One of the more important aspects of this work is the focus on the critical aspects of control of the worldwide epidemic.

The editors clearly make the point that understanding the spread of HIV (and how helping those infected with HIV may be accomplished) is seemingly easy when compared to the challenge of effecting worldwide cultural changes that may be needed to curtail the continued dissemination of HIV as well as the suffering, death, and economic disaster that come in the wake of this spread. This compendium serves to fill an important void in the medical literature that challenges the English reading readership not to overlook the many cross-cultural issues faced both worldwide and within each of our own regions when dealing with the AIDS epidemic.

The cost of $29.00 is small for those who have an interest in further understanding some of the cross-cultural issues the control of HIV demands. As Schoepf et al say (p 275), "unless behavior changes before HIV infection becomes widely established in heterosexual populations in other countries, Central Africa's health and development of the 1980's may turn out to be a harbinger, a grim model of the West at the dawn of the twenty-first century."

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PREVENTING EMERGENCY MALPRACTICE

By Raymond M. Fish, MD, and Melvin E. Ehrhardt, MD, JD. 179 pp. Oradell, NJ, Medical Economics Co, Inc, 1989. $31.95 (hardcover)

The emergency department (ED) in a hospital is second only to the department of obstetrics and gynecology in the number of medical malpractice suits it generates. This is true because the doctor/patient relationship is not given time to develop, patients may not follow or even remember the doctor's directions given their tense response to being in the emergency room setting, and key elements of the history may be unknown to the physician. Although chapters in other books, newsletters, and seminars have touched on the subject of emergency medicine malpractice, there has never before been a book fully devoted to avoiding it.

Raymond Fish and Melvin Ehrhardt, both practicing ED physicians, and the latter an attorney as well, explore the traps inherent in managing complex cases in the busy emergency department. The book is readable and short enough (179 pages) to absorb in full. Chapters are clearly structured and key points highlighted. The authors repeatedly underscore the importance of executing and fully documenting the thorough history and physical examination as the best malpractice defense. Dozens of cases are presented to illustrate the wrong approach and the missteps that have led to malpractice suits.

Topics discussed in the book include the proper role for the ED physician; issues concerning consent and refusal of treatment; "red flag" cases, which alert the physician to follow a very conservative course; the conditions that most often precipitate major lawsuits, and why: myocardial infarction, meningitis, fractures, wounds, ectopic pregnancy, and appendicitis; do-not-resuscitate (DNR) orders; the ED physician's legal responsibility in cases of child abuse, rape, and other crimes; patient confidentiality; and finally, psychiatric cases.

The authors note in passing that the practice of defensive medicine is as unfortunate a response in the ED as in other medical arenas. In the book's case analyses, it appears that an idealized standard of practice is being advocated. The authors acknowledge that while in hindsight many a lawsuit could have been avoided if only the physician had held to this ideal standard, the physician has real world constraints, too. Time and financial considerations may hinder the completeness of the history and physical exam and laboratory workups. The physician will not x-ray every area of minor pain and will not admit all patients with minimally suspicious chest pain or head injury. We are left with a book that serves as a useful general guide without helping the physician find the best balance between "real" and "ideal."

Moreover, there are two omissions in the book. First, malpractice in the ED is committed not only by the physician, but also by the rest of the staff including nurses, clerks, and aides. A teamwork approach should be instituted in the ED, with the physician as coordinator. Nurses should act as additional eyes and ears for the physician. Individuals suited to emergency medicine should be picked to join the team; the wrong mix of people also contributes to the risk of malpractice. Second, the personal characteristics of physicians are ignored. My own experience is that the physician with a confident, logical approach will rarely get sued. It is the physician who is unsure of himself, ordering excessive workups but missing the key symptoms, who will be involved in a malpractice case.

It should not be expected that this or any book can teach the subtleties that only an experienced emergency physician possesses. As a starting point in coaching the emergency practitioner, however, the book fills a void as a concise definition of the medicolegal danger zones in the ED setting.

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HISTORY OF MEDICINE

Edited by Rebecca Greene, PhD. 133 pp. Binghamton, NY, The Haworth Press, Inc, 1988. $24.95 (hardcover)

I was completely misled by the title of this book! As a medical history enthusiast, I accepted a request to review it, expecting to receive a very thick volume. Instead I received a very small package containing an attractive 133-page book bearing the impressive title *History of Medicine*. The introduction, as written by editor Rebecca Greene, outlines the purpose of the work and includes a series of questions which are answered by the authors in five short essays (18 to 26 pages in length). Those of us who prefer practical and/or clinical papers will enjoy "The History of Birth Control in the United States" by Esther Katz and "The History of Medical Care in Infants in the United States" by Gerald Oppenheimer. The other three papers, John Scarborough's "Classical Antiquity," Robert Goler's "Health Sciences in Early America," and Faye Getz's "Western Medieval Medicine" will appeal to medical historians.

The book is unique and valuable as a source of information for those interested in researching the past history of our profession. It can be best described as a complete list of references knitted together by interesting comments and reviews written by knowledgeable experts.

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Promising new diagnostic tool for early detection of breast cancer

Needle localization is the most effective and reliable method available for marking potentially cancerous lumps in the breast before they are even palpable. Performed under local anesthetic during a mammographic examination, marking of suspicious masses simplifies subsequent biopsy or surgical removal of the lesion.

The "double helix" marker, developed at Tulane University Medical Center (New Orleans, La), offers advantages over current tools used in needle localization: it maintains its position in the tissue, without slipping, and it produces sharp, clear images with x-ray or ultrasonography. The unique, yet simple design of the marker incorporates a steel wire with helical coils at both ends. As the marker is introduced through a cannula, the first helix enters the tissue and becomes firmly anchored in the lesion. The other helix extends beyond the cannula and serves as a depth gauge as well as a stabilizer to prevent deflection of the needle as it passes through the breast tissue. The increased surface area of the helical structure enhances ultrasonic imaging.

The developers of the double helix marker are now seeking patent protection and licensing agreements for its manufacture.

High tech yields new treatment for bacterial infections

As many as 200,000 people each year in the United States contract bacterial infections that can lead to septic shock. Most victims contract the infections while in the hospital, and approximately 40% of such infections are fatal (*NY Times*, September 19, 1989, p C5). A new treatment regimen, based on monoclonal antibody technology, has shown promise in halting these infections and preventing the development of septic shock.

The results of a large study of this treatment, which included nearly 500 patients at 33 medical centers, were recently announced by Xoma Corp (Berkeley, Calif), producers of the monoclonal antibodies. The treatment was effective when instituted shortly after the onset of infection and prior to the development of septic shock. Xoma reported a decrease in deaths by 46% with the treatment regimen. There was no reduction in mortality among patients with infection who were already in shock.

Gram-negative bacteria that normally inhabit the digestive tract can cause these infections when they become hematogenous. Surgery, abdominal wounds, and insertion of contaminated tubes into blood vessels provide opportunities for the bacteria to become bloodborne. Antibiotic therapy can usually destroy bacteria, but antibiotics do not counteract the toxins released by the bacteria that produce the symptoms of septic shock.

Monoclonal antibodies specific for these toxins can bind to the compounds and make them ineffective, but cannot reverse the progression of septic shock once organ damage has begun. Effective treatment depends on a combination of antibiotics and targeted monoclonal antibodies administered at the first signs of infection and before the patient goes into shock.

Food and Drug Administration approval of the product is anticipated toward the middle of 1990.

Pressure from AIDS patients and a growing market lead to AZT price reduction

The antiviral drug azidothymidine (AZT) can slow the progression of AIDS and prevent the development of its associated opportunistic infections in some patients by inhibiting the replication of human immunodeficiency virus (HIV), reducing serum HIV antigen levels, and blocking immunologic deterioration. However, treatment with AZT is very expensive, thus limiting its accessibility.

AIDS patients and their advocates have been demanding a price reduction by Burroughs-Wellcome Co (Research Triangle Park, NC), the manufacturer of AZT. Faced with growing pressure from AIDS activists as well as an anticipated increase in market size—recent studies suggest that AZT may delay the development of AIDS in people infected with HIV but not yet exhibiting symptoms of the disease—Burroughs-Wellcome reduced the price of AZT by 20% (*Wall Street J*, September 19, 1989, p A3). This produced a drop in the average retail price of AZT from $8,000 to $6,400 a year for each patient. "But recent studies suggest that the drug may be effective at 40% or 50% of the original dose, which could further reduce the cost to patients to about $3,000."

The company cited the initial uncertainty surrounding AZT's effectiveness against AIDS and the mounting costs of research as the primary reasons for AZT's high price tag. These concerns become more difficult to support as the numbers of AIDS patients increases, the demand for AZT continues to rise, and the potential market for AZT among the hundreds of thousands of people infected with HIV but not yet exhibiting symptoms of AIDS could boost sales of the drug to $1 billion by 1992.

FDA investigations sting pharmaceutical companies

Under increasing scrutiny by the US Food and Drug Administration (FDA), several drug companies face charges of unsafe products and submission of false or incomplete data from drug tests.

Halcion (UpJohn Co; Kalamazoo, Mich), a commonly prescribed sleep medication, is under investigation for reports of "a disproportionately high number of negative side effects, including hallucinations and heightened aggressiveness" (*Wall Street J*, September 20, 1989, p A16). Defending its product, the company avers that the side effects associated with Halcion are no more severe than those of similar prescription drugs.

The FDA has charged Eli Lilly & Co (Indianapolis, Ind) employees with providing false or incomplete data on drug studies, such as neglecting to report failed tests for drug stability (*Wall Street J*, September 21, 1989, p A4). Investigators also cited incidents in which Lilly employees signed records verifying procedures in drug production that they did not actually witness. The investigations sub-

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NEWS BRIEFS are compiled and written by Vicki Glaser.

committee of Congress' House Energy and Commerce Committee will pursue the FDA charges.

Misrepresentation of data is the charge against Quantum Pharmics, Ltd (Amityville, NY), a generic drug unit of American Home Products Corp (*Wall Street J*, September 21, 1989, p A4). An FDA investigation of information supplied by the company during the approval process of three drugs prompted Quantum to interrupt its shipping of products and to stop operating. During a congressional investigation, Quantum was shown to have had a "special" relationship with a former FDA reviewer who apparently accepted gifts and favors in exchange for favorable treatment of a company's application for approval to market a drug. According to the *Wall Street Journal*, three FDA employees resigned from the agency and pleaded guilty to criminal charges related to helping certain generic drug companies obtain approvals to market generic versions of brand-name drugs.

FDA probes into the operations of two other generic drug makers—Par Pharmaceutical, Inc (Spring Valley, NY) and Vitarine Pharmaceutical Co (Springfield Gardens, NY)—recently forced temporary production stoppages at those companies.

JAMA's education issue highlights trends

A recent issue of *JAMA* (1989; 262:1001–1002) examined the status of medical education in the United States in 1989. Following are excerpts from that issue:

- The number of applicants to US schools accredited by the Liaison Committee on Medical Education (LCME) for the 1988–1989 academic year decreased by 1402 individuals.
- Of the 26,721 applicants, 17,108 students (64%) were accepted by at least one school for enrollment in the fall of 1988.
- The percentage of "A" students entering has decreased and the average Medical College Admission Test score of entering students is down.
- The care of patients as a duty of medical school faculty has expanded over the past several years as medical school budgets have become more dependent on patient care.
- The average indebtedness of 1988 medical school graduates with debt was $38,489.
- The number of women enrolling in and graduating from medical schools continues to increase, although the number applying to medical schools shows a decrease.
- Of the residents on duty in 1988–1989, about 15% were foreign medical school graduates (FMGs).
- There are currently nine state medical societies that require continuing medical education (CME) for membership. There are 22 state licensing boards, plus one in Puerto Rico, that require the physician to have CME credits for reregistration of the medical license.

When physicians lose their malpractice insurance: what are the options?

Physicians may be refused malpractice insurance for a variety of reasons: primarily, poor claim and payout histories, as well as loss of hospital privileges, drug and alcohol abuse, charges of sexual harassment of patients, and Medicare fraud. William B. Schwartz, MD, and Daniel N. Mendelson examined the options available to physicians who are refused standard malpractice insurance, and they collected and analyzed demographic data on 920 physicians who applied to a so-called surplus-lines insurance company (*JAMA* 1989; 262:1335–1341).

Physicians who cannot obtain standard malpractice insurance often turn to surplus-lines companies, which can afford to insure high-risk applicants by setting premiums and deductibles at whatever amount the insurer believes to be appropriate and establishing limitations on the practices of the insured. "Laws in most states prohibit physicians from applying to a surplus-lines carrier unless they have been denied coverage by at least three standard-lines companies."

The authors found that practice specialties including neurosurgery, plastic surgery, obstetrics/gynecology, and orthopedics are overrepresented among physicians who applied to a surplus-lines company. Physicians who practice psychiatry, pediatrics, and internal medicine are least likely to apply to the company represented in the study. "Surplus-lines physicians aged 45 to 54 years are overrepresented . . . ($P<.01$). By contrast, physicians aged 35 to 44 years are underrepresented . . . ($P<.01$)." The percentage of applicants who were foreign medical graduates (FMGs) did not differ significantly from the proportion of FMGs in the US population.

For the specific insurance company depicted in the study, premium rates were two to four times higher than those charged by standard-lines insurers. Deductibles ranged from $5,000 to $10,000 per claim, and the policies were commonly written for $1 million per claim. "Up to the limit of the deductible, the physician is responsible for investigative and legal costs as well as settlements or awards." Restrictions imposed by the insurance company included the right to perform various types of high-risk surgery as well as the right to practice obstetrics.

The estimated 2,000 physicians each year in the United States who lose their malpractice insurance due to negligent behavior do have other options. They can stop seeing patients and apply their medical training to an administrative or research-oriented position in a medical center, government agency, health-related organization, or pharmaceutical company, for example. They can continue to practice with no insurance coverage and limit their practice to lower risk patients and procedures. Alternatively, as described, they can seek insurance from a surplus-lines company and accept the financial penalties and practice restrictions that accompany their malpractice coverage. Another option is a joint underwriting association, which, by law, must insure any physician who applies, and typically does not carry the burden of higher premiums, has no deductibles, and imposes no practice limitations. "The easiest and least disruptive path for the physician who wishes to stay in practice is to turn to a joint underwriting association."

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Joseph Carl Addonizio, MD, Valhalla. Died August 20, 1989; age 59. Dr Addonizio was a 1956 graduate of Georgetown University School of Medicine, Washington, DC. He was a Diplomate of the American Board of Urology and a Fellow of the American College of Surgeons. His memberships included the New York Academy of Medicine, the New York State Urological Society, the American Urological Association, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

E. Mark Beckman, MD, New York. Died August 17, 1989; age 54. Dr Beckman was a 1961 graduate of the Chicago Medical School. He was a Diplomate of the American Board of Obstetrics and Gynecology and a Fellow of the American College of Surgeons and the American College of Obstetricians and Gynecologists. He was a member of the New York Obstetrical Society, the American Society of Clinical Oncology, the New York County Medical Society, and the Medical Society of the State of New York.

H. Hermann Bonheim, MD, Coram. Died August 25, 1989; age 82. Dr Bonheim was a 1931 graduate of the Medizinische Fakultaet der Universitaet Hamburg, Hamburg, Germany. He was a Fellow of the American Academy of Family Physicians. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Hubert Francis Carroll, MD, Indian Lake. Died July 19, 1989; age 95. Dr Carroll was a 1918 graduate of the Albany Medical College of Union University. He was a Fellow of the American Academy of Family Physicians. He was a member of the Medical Society of the County of Warren and the Medical Society of the State of New York.

Irving Cramer, MD, New Hartford. Died July 30, 1989; age 74. Dr Cramer was a 1941 graduate of the Albany Medical College of Union University. He was a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons. His memberships included the New York Academy of Medicine, the New York State Surgical Society, the Society of Surgical Oncology, the Medical Society of the County of Oneida, and the Medical Society of the State of New York.

Rodolph M. Cutino, MD, Lake Hopatcong, New Jersey. Died July 1, 1989; age 92. Dr Cutino was a 1921 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Michael Angelo Diana, MD, New York. Died August 20, 1989; age 80. Dr Diana was a 1931 graduate of the New York Medical College. He was a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons. He was a member of the New York Academy of Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Louis Jay Gilbert, MD, New York. Died July 16, 1989; age 75. Dr Gilbert was a 1939 graduate of the Indiana University School of Medicine. He was a Diplomate of the American Board of Psychiatry and Neurology. He was a member of the American Psychiatric Association, the American Academy of Child Psychiatry, the Academy of Psychoanalysis, the New York County Medical Society, and the Medical Society of the State of New York.

Meyer Leo Goldman, MD, Lawrence. Died August 18, 1989; age 82. Dr Goldman was a 1933 graduate of the University of Maryland School of Medicine. He was a Diplomate of the American Board of Surgery and a Qualified Fellow of the International College of Surgeons. His memberships included the New York Academy of Medicine, the Pan American Medical Association, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Flem Goolsby, Jr, MD, Knoxville, Tennessee. Died August 13, 1989; age 50. Dr Goolsby was a 1975 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the Dutchess County Medical Society and the Medical Society of the State of New York.

Raymond Harris, MD, Albany. Died August 25, 1989; age 70. Dr Harris was a 1943 graduate of the Albany Medical College of Union University. He was a Diplomate of the American Board of Internal Medicine and a Fellow of the American College of Cardiology. He was a member of the New York State Society of Internal Medicine, the American Rheumatism Association, the American Geriatrics Society, the Medical Society of the County of Albany, and the Medical Society of the State of New York.

Frederic W. Holcomb, Jr, MD, Kingston. Died August 1, 1989; age 70. Dr Holcomb was a 1944 graduate of the Albany Medical College of Union University. He was a member of the Medical Society of the County of Ulster and the Medical Society of the State of New York.

Robert Sherman Hotchkiss, MD, New Fairfield, Connecticut. Died August 1, 1989; age 86. Dr Hotchkiss was a 1928 graduate of the University of Michigan Medical School. He was a Diplomate of the American Board of Urology. His memberships included the American Association of Genito-Urinary Surgeons, the American Urological Association, the American Fertility Society, the New York County Medical Society, and the Medical Society of the State of New York.

Lee Jackson, MD, Rochester. Died July 9, 1989; age 67. Dr Jackson was a 1950 graduate of the University of Rochester School of Medicine-Dentistry. She was a Diplomate of the American Board of Pediatrics. She was a member of the American Psychiatric Association, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Jose E. Jaen, MD, Binghamton. Died August 11, 1989; age 59. Dr Jaen was a 1956 graduate of the Faculty of Medicine and Surgery University of Santo Tomas, Manila, Philippines. He was a member of the Broome County Medical Society and the Medical Society of the State of New York.

Kurt Lange, MD, New York. Died July 29, 1989; age 83. Dr Lange was a 1932 graduate of the Friedrich-Wilhelms Universitaet Medizinische Fakultaet, Berlin, Germany. He was a Fellow of the American College of Physicians and a Fellow of the American College of Cardiology. His memberships included the Society for Experimental Biology and Medicine, the American Society of Nephrology, the American Association of Pathologists and Bacteriologists, the New York County Medical Society, and the Medical Society of the State of New York.

John Peter Liu, MD, New York. Died June 9, 1989; age 37. Dr Liu was a 1977 graduate of the Columbia University College of Physicians and Surgeons. He was a Diplomate of the American Board of Psychiatry and Neurology. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Harvey John Merk, MD, New York. Died August 13, 1989; age 76. Dr Merk was a 1940 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a Diplomate of the American Board of Obstetrics and Gynecology and a Fellow of the American College of Surgeons and the American College of Obstetricians and Gynecologists. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

William Frances Nitty, Jr, MD, New York. Died August 9, 1989; age 67. Dr Nitty was a 1946 graduate of the New York University School of Medicine. He was a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons and the American College of Gastroenterology. His memberships included the New York Academy of Medicine, the New York State Surgical Society, the New York Gastroenterological Society, the American Geriatrics Society, the American Trauma Society, the American Gastro-Enterological Association, the New York County Medical Society, and the Medical Society of the State of New York.

Alice E. Bennett Murray, MD, Phoenix, Arizona. Died July 31, 1989; age 92. Dr Murray was a 1937 graduate of the State University of New York at Buffalo School of Medicine. She was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Bernard S. Puglisi, MD, Champaign, Illinois. Died August 1, 1989; age 82. Dr Puglisi was a 1935 graduate of the University of Rochester School of Medicine-Dentistry. He was a Fellow of the American Academy of Family Physicians and the American College of Allergists. He was a member of the New York Academy of Medicine, the Pan American Medical Association, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

John Morris Rodgers, MD, East Moriches. Died August 11, 1989; age 61. Dr Rodgers was a 1955 graduate of the State University of New York Health Science Center at Brooklyn. His memberships included the New York State Society of Internal Medicine, the American Thoracic Society, the American Society of Internal Medicine, the Suffolk County Medical Society, and the Medical Society of the State of New York.

Isidore Segal, MD, Scotch Plains, New Jersey. Died May 25, 1989; age 39. Dr Segal was a 1984 graduate of the Albert Einstein College of Medicine-Yeshiva University, Bronx. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Solomon Slepian, MD, Union, New Jersey. Died July 30, 1989; age 91. Dr Slepian was a 1929 graduate of the State University of New York Health Science Center at Brooklyn. He was a Fellow of the American College of Allergists. His memberships included the New York State Society of Internal Medicine, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Samuel Stone, MD, New York. Died July 26, 1989; age 76. Dr Stone was a 1937 graduate of New York University School of Medicine. He was a Diplomate of the American Board of Pediatrics and a Fellow of the American Academy of Pediatrics. His memberships included the New York Academy of Medicine, the American Thoracic Society, the New York County Medical Society, and the Medical Society of the State of New York.

Charles Bernard Storch, MD, Brooklyn. Died August 7, 1989; age 77. Dr Storch held a Registrable Qualification Granted by the Scottish Conjoint Board in 1939. He was a Diplomate of the American Board of Radiology. His memberships included the Radiological Society of New York State, the New York Roentgen Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Arthur M. Tunick, MD, Hackensack, New Jersey. Died August 7, 1989; age 81. Dr Tunick was a 1934 graduate of the State University of New York Health Science Center at Brooklyn. He was a Diplomate of the American Board of Internal Medicine and a Fellow of the American College of Physicians. He was a member of the New York State Society of Internal Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Manfred Weichsel, MD, Floral Park. Died August 17, 1989; age 86. Dr Weichsel was a 1927 graduate of the Medizinische Fakultaet der Johann Wolfgang Goethe Universitaet, Frankfurt-am-Main, Hessen, Germany. He was a Diplomate of the American Board of Pediatrics and a Fellow of the American College of Allergists and the American Academy of Pediatrics. His memberships included the American Public Health Association, the American Association of Immunologists, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Attention Delegates
Your Next Meeting

The 184th Annual Meeting of the Medical Society of the State of New York will be held at the New York Hilton, New York, on March 29–April 1, 1990

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Nominating Committee, 1990

(The committee nominates Officers, Councilors, and Trustees of MSSNY, and Delegates to the American Medical Association.)

Member-at-Large—Charles D. Sherman, Jr., M.D., Monroe, *Chairman*
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Sixth District—Antonio F. LaSorte, M.D., Broome
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Ninth District—John A. Ramsdell, M.D., Westchester
Member-at-Large—Seymour R. Stall, M.D., Dutchess

FOR MY PATIENTS

NUMBER 10, 1989

YOU CAN MAKE WEAK BONES STRONGER

Dear Patient:

If you are a female, you have a 50% chance of having osteoporosis before you reach age 65. If you are a male, your chance is less than 7%. Osteoporosis or thinning of bones results from a gradual loss of bone mass over many years. Bone loss increases with aging and can begin in the teenage years if the daily diet has too little calcium. It is more common in older women because the estrogen hormone which maintains bone mass in females is produced in smaller quantities after their menopause.

Dangers of Osteoporosis

Little by little, many bones become weakened and fragile. When it affects the spine, it can lead to kyphosis ("round back") or scoliosis ("tilted back"). It can also result in serious fractures of the hip, spine, wrist, or ribs following slight injuries or falls which ordinarily would not affect healthy persons.

Related Risk Factors

Besides poor diet and lack of exercise, the following factors are related to osteoporosis:

- ☐ petite, thin body build
- ☐ smoking
- ☐ alcoholism
- ☐ family history
- ☐ fair complexion
- ☐ Caucasian or oriental background

Prevention

Taking estrogen hormone regularly within 3-6 years after menopause will slow down or stop further loss of bone mass. Daily weight-bearing exercises such as walking also help prevent the condition. Eating a proper daily diet which includes enough calcium (1000-1500 milligrams) and vitamin D (400 international units) is necessary from adolescent years to old age. Before you take these important steps, you must consult me so that I can tailor the prevention to your exact needs.

Boosting Calcium in Your Diet

Focus your intake on these items:

- ☐ Milk and milk products such as yogurt and cheese
- ☐ Sardines (with bones)
- ☐ Steamed vegetables such as broccoli and collards
- ☐ Calcium supplements (see me for recommendations)

Ways to Detect Osteoporosis

- ☐ Physical examination of spine for deformity
- ☐ X-rays can show large amount of bone loss (late stage)
- ☐ CT scans can show small amount of bone loss (early stage)
- ☐ Absorptiometry (imaging) can show small amount of bone loss at the wrist (early stage)

Treatment

Treatment of osteoporosis is individualized according to the age of the patient and the amount of bone loss. Usually the goal is to prevent further bone loss or replace lost bone. Estrogen hormone can slow or prevent further bone loss even if it is first prescribed later than six years post-menopause. Some studies have indicated that sodium fluoride added to calcium and vitamin D each day may stimulate more bone formation in patients who have had spinal fractures.

Begin now to strengthen the framework of your body — your bones. Let me figure out your risk of this disease and advise a course of action, if you need one.

MEDICAL SOCIETY OF THE STATE OF NEW YORK
ORGANIZED 1807

Office Handouts for Your Patients

This issue of the *New York State Journal of Medicine* contains another "For My Patients" letter, which appears on the preceding page.

Use this letter series to keep your patients informed about timely issues concerning diseases, treatments and health maintenance. Simply make photocopies of the page and distribute them to your patients with your newsletter or other correspondence, or place them in your reception area for your patients to read and take home.

MSSNY and the *New York State Journal of Medicine* have waived the copyright for these handouts, so you may duplicate them without permission.

As an added feature, MSSNY will personalize this form and make copies for you, at a nominal charge. See the example below for how these forms would look with your name and address professionally printed on them. Use the order form below, and we'll ship the personalized forms to you as soon as possible.

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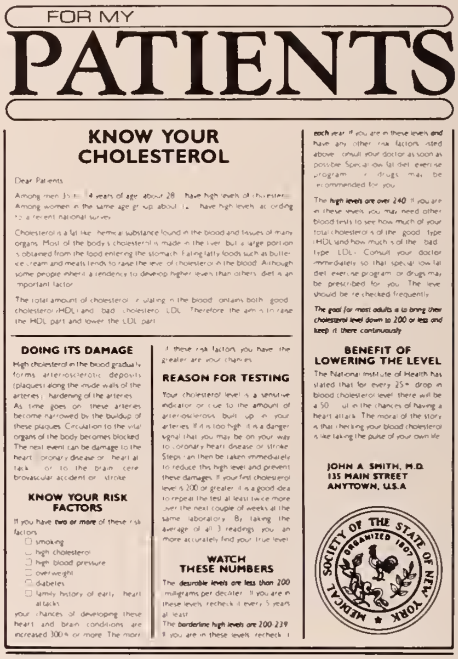

FOR MY

PATIENTS

KNOW YOUR CHOLESTEROL

Dear Patients

Among men 35 to 74 years of age, about 28 [illegible] have high levels of cholesterol. Among women in the same age group, about [illegible] have high levels, according to a recent national survey.

Cholesterol is a fat-like chemical substance found in the blood and tissues of many organs. Most of the body's cholesterol is made in the liver, but a large portion is obtained from the food entering the stomach. Eating fatty foods such as butter, ice cream and meats tends to raise the level of cholesterol in the blood. Although some people inherit a tendency to develop higher levels than others, diet is an important factor.

The total amount of cholesterol circulating in the blood contains both "good" cholesterol (HDL) and "bad" cholesterol (LDL). Therefore the aim is to raise the HDL part and lower the LDL part.

DOING ITS DAMAGE

High cholesterol in the blood gradually forms arteriosclerotic deposits (plaques) along the inside walls of the arteries (hardening of the arteries). As time goes on, these arteries become narrowed by the buildup of these plaques. Circulation to the vital organs of the body becomes blocked. The next event can be damage to the heart (coronary disease or heart attack) or to the brain (cerebrovascular accident or stroke).

KNOW YOUR RISK FACTORS

If you have **two or more** of these risk factors

☐ smoking
☐ high cholesterol
☐ high blood pressure
☐ overweight
☐ diabetes
☐ family history of early heart attacks

your chances of developing these heart and brain conditions are increased 300% or more. The more of these risk factors you have, the greater are your chances.

REASON FOR TESTING

Your cholesterol level is a sensitive indicator or clue to the amount of arteriosclerosis built up in your arteries. If it is too high, it is a danger signal that you may be on your way to coronary heart disease or stroke. Steps can then be taken immediately to reduce this high level and prevent these damages. If your first cholesterol level is 200 or greater, it is a good idea to repeat the test at least twice more over the next couple of weeks at the same laboratory. By taking the average of all 3 readings, you can more accurately find your true level.

WATCH THESE NUMBERS

The **desirable levels are less than 200** milligrams per deciliter. If you are in these levels, recheck it every 5 years at least.

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The **high levels are over 240**. If you are in these levels, you may need other blood tests to see how much of your total cholesterol is of the "good" type (HDL) and how much is of the "bad" type (LDL). Consult your doctor immediately so that special low fat diet, exercise program or drugs may be prescribed for you. The level should be rechecked frequently.

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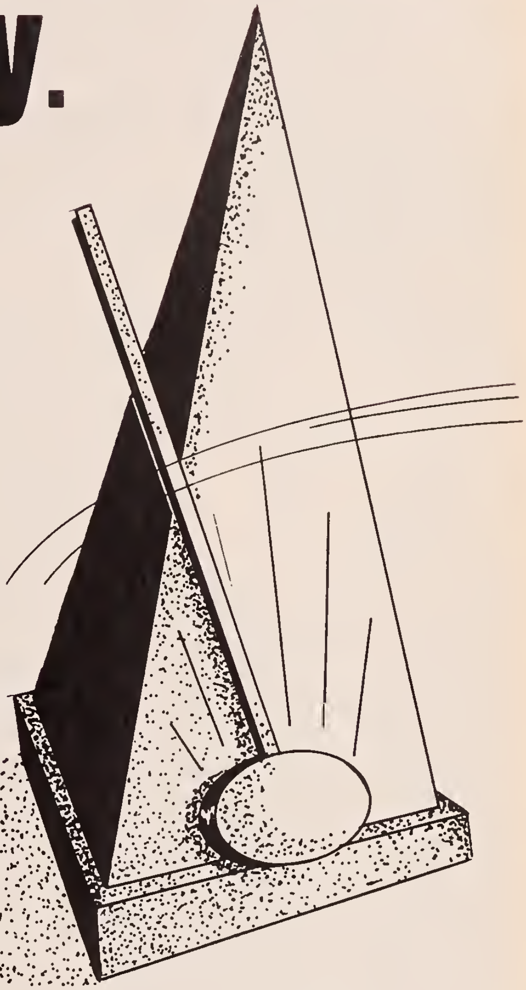

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References:

1. A. Morales et al., New England Journal of Medicine: 1221, November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188, McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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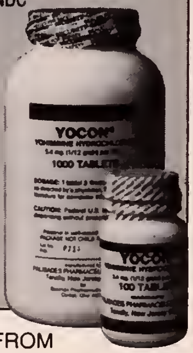

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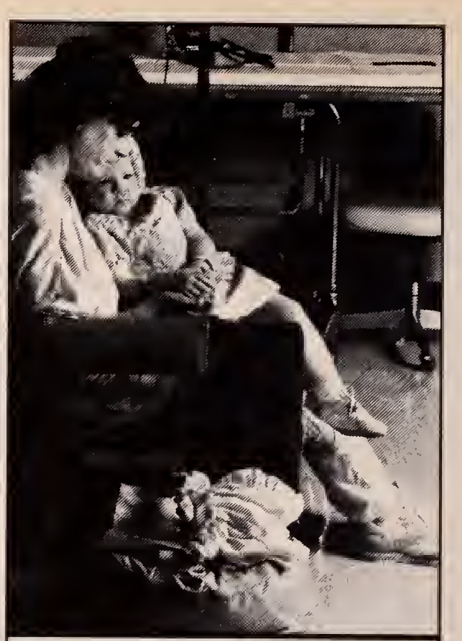

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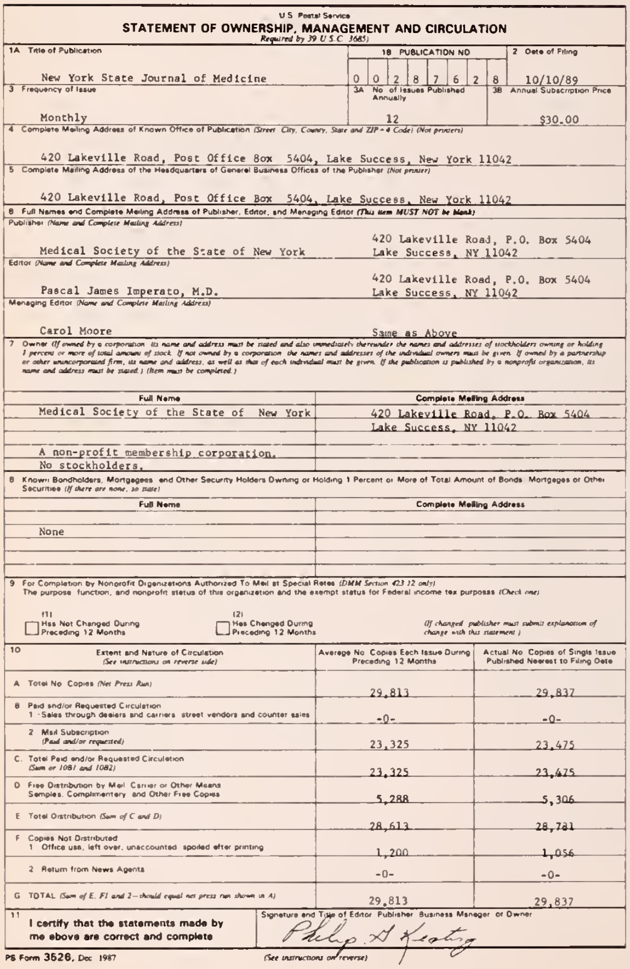

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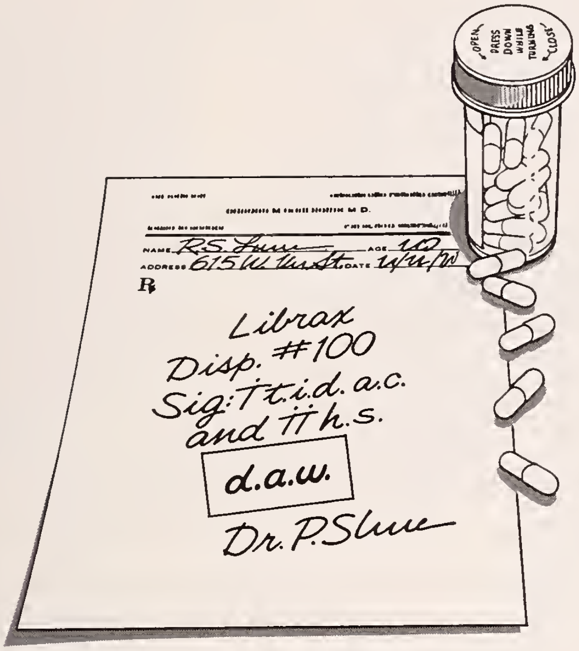

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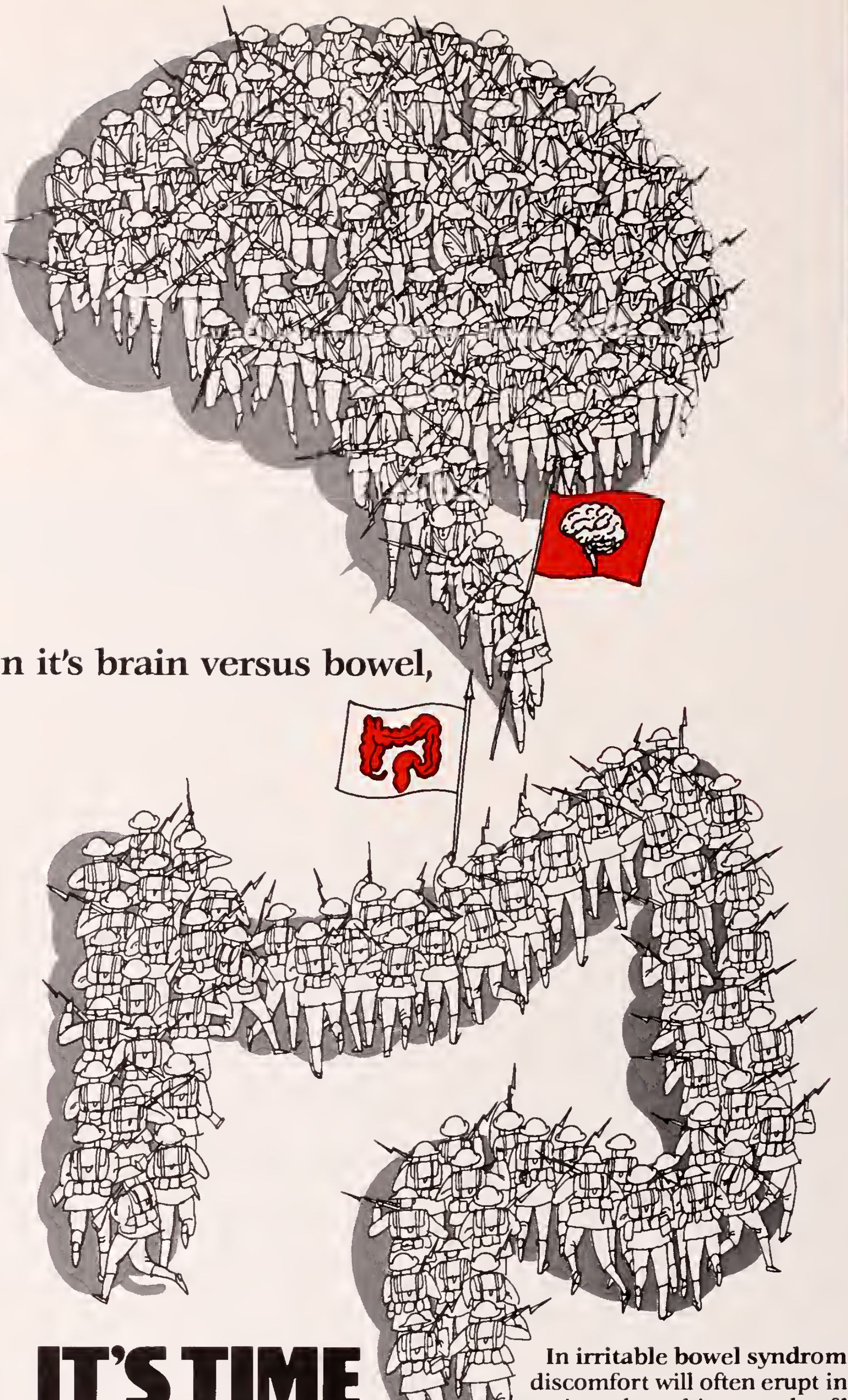

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Contents

COMMENTARIES

Health problems associated with urban poverty: A narrowing gap between the Third and First Worlds 649
KATHERINE KAYE, MD, MPH

Demolishing the lead danger 651
SUSANA R. ALVAREZ, MD, MPH

RESEARCH PAPERS

Resuscitation of cardiac arrest victims by advanced life support units in Monroe County, New York 652
THOMAS A. SWEENEY, BS, EMT, MARTHA R. DORN, MD

Paraesophageal hernia 654
CARLO E. VITELLI, MD; BERNARD M. JAFFE, MD; KIM U. KAHNG, MD

REVIEW ARTICLE

The crisis in New York City's perinatal services 658
WENDY CHAVKIN, MD, MPH; CYNTHIA R. DRIVER, RNC, MPH; PAT FORMAN, MPH

ESSAYS IN PEDIATRICS

The management of respiratory failure in infants and children 664
BRUCE M. SCHNAPF, DO

COMMITTEE REPORT

Animal experimentation for medical research 671
FRED ROSNER, MD; ALLEN J. BENNETT, MD; ERIC J. CASSELL, MD; PETER B. FARNSWORTH, MD; ABRAHAM L. HALPERN, MD; ALLISON B. LANDOLT, MD; LAURENCE LOEB, MD; PATRICIA J. NUMANN, MD; FERNANDO V. ONA, MD; HERMAN M. RISEMBERG, MD; PHILIP H. SECHZER, MD; PETER P. SORDILLO, MD; JONATHAN R. WOLPAW, MD

SPECIAL REPORT

A strategic plan for the elimination of tuberculosis in the United States 677

CASE REPORTS

Lead poisoning during home renovation 679
JOHN P. CURRAN, MD, JORGE R. NUNEZ, MD

Development of multiple carcinomas in a long term survivor of Wegener's granulomatosis treated with immunosuppressive drugs 680
DANIEL S. J. CHOY, MD; ROBERT P. GEARHART, MD; WILBUR J. GOULD, MD; JOHN SAUER, MD; LEONARD JACOBSON, MD; BARRY ROSENTHAL, MD

Transient cortical blindness following an acute hypotensive event in the postpartum period 682
LEONARD B. STEIN, MD; RICHARD I. ROBERTS, MD; JEFFREY MARX, MD; LEONARD ROSSOFF, MD

LETTERS TO THE EDITOR

House staff stress and the impact of the new regulations on postgraduate medical education 684
PHILIP P. BONANNI, MD

The future of medicine 684
HANS POLLAK, MD

Tombstone erected on grave of Arthur Donaldson Smith, MD 685
MARGARET A. POTTER

The academic boycott of South Africa 685
FRED ROSNER, MD

LEADS FROM EPIDEMIOLOGY NOTES 686

BOOK REVIEWS 690

NEWS BRIEFS 695

OBITUARIES 697

1989 INDEX 699

MEDICAL MEETINGS AND LECTURES 4A

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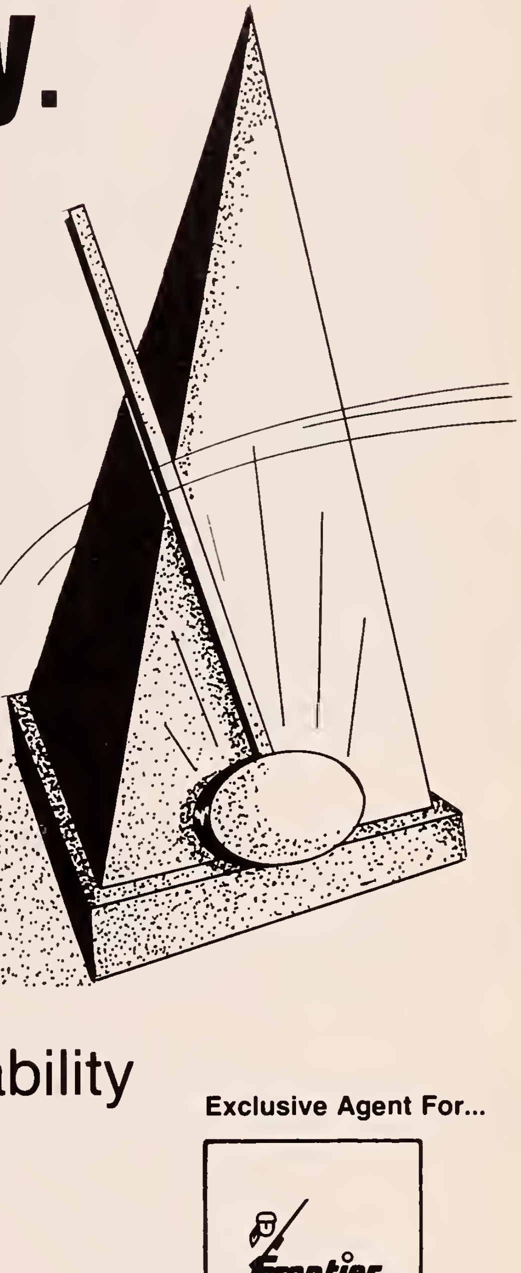

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1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
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COMMENTARIES

Health problems associated with urban poverty: A narrowing gap between the Third and First Worlds*

Health leaders who met at the World Health Organization's conference at Alma Ata in 1978 defined primary care as the key to obtaining "Health for All by the Year 2000."[1] Now that the fanfare marking the tenth anniversary of that historic conference has subsided, we are left with two important questions: First, how far have we in the United States advanced towards achieving the goals in primary care which were then set, and second, do the differences in health problems between developed and developing worlds remain as prominent as they were ten years ago? It is with regard to the health status of the urban poor that these questions deserve the most attention.

The growing similarity between certain health problems in developed and developing worlds arises from the rapid urbanization which is seen in many areas of the Third World, as well as from a failure in this country during the 1980s to improve the health status of the poorest urban populations. Certainly the fact that 50% of the world's population will be living in cities by the year 2000[2] lends some urgency to the need for a dialogue between developed and developing worlds about approaches to a common problem. The benefits to be derived from such a dialogue will in no way accrue only to the developing world: The opportunity to draw upon innovative ideas is one of the hidden benefits of US participation in international programs.[3]

Health indices in even the poorest sections of American cities tend to be much better than those of Third World slums. It is instead the disparity which exists between health indices in the poorest and most affluent sections of American cities that resembles the situation found in developing countries. Data compiled by the New York City Department of Health illustrate the bimodal distribution of some health indicators: In 1986, the infant mortality rate for Central Harlem rose to 27.6, while that for the more affluent section of Kips Bay was 7.5; almost 23% of births in Mott Haven were to teenaged mothers, compared to 5.9% in Richmond.[4] This type of disparity is even more pronounced in Third World cities, where the wealthiest inhabitants have access to the most advanced technologies while people living in slums experience living and working conditions that may actually be worse than those of the rural poor.[5] The tendency on the part of governments in developing countries to aggregate all data from urban areas or fail to even collect information about people living in slums masks the true health status of the urban poor.[6] It should be remembered, however, that the absence of data about urban subpopulations is also a problem in New York City, where information about the health status of homeless persons and undocumented immigrants—whose numbers are not negligible—is sketchy at best.

In the last ten years, we have realized that the provision of primary care in urban slums worldwide has generally not kept pace with programs designed for rural areas.[6] The extent to which preventive primary care remains an illusion for many poor children in New York City is illustrated by the occurrence of 1,413 measles cases during the 1986–1987 measles epidemic (New York City Department of Health Immunization Bureau, unpublished data, 1988). This is especially noteworthy because it is in the field of child health that health care workers, in very diverse settings, have tried hardest to create comprehensive programs that include preventive care. The young age distribution of New York City's poorest people indicates that, as in the Third World, maternal and child health issues are of vital importance. Within Manhattan, the proportion of the population younger than 24 years ranges between 34% and 44% in the three community districts with the lowest median household incomes (the Lower East Side, Central Harlem, and East Harlem), as compared to 16% and 20% in the two districts with the highest household incomes (the Upper East Side and the Murray Hill area).[7] Children in New York City are more likely to be of nonwhite race or Hispanic descent than are older persons; almost half of all births in the city are to women classified as minority group members.[4]

Until the goal of providing ongoing preventive care to all city children can be realized, it seems sensible to emphasize, here in New York as well as in slums of the developing world, some selective primary care interventions that adhere to the growth monitoring/oral rehydration/breast feeding/immunizations—food supplementation/family planning/female literacy (GOBI-FFF) model developed by UNICEF.

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*This commentary is based in part on a paper prepared while the author was a public health resident with the New York City Department of Health, and presented at the 1988 conference of the National Council on International Health in Washington, DC.

Just as in the field of international health, the problem of inadequate immunization status lends itself most easily to direct intervention. In order to address the problem of poor preschool immunization levels, particularly among homeless and uninsured children and those without regular medical care providers, Department of Health personnel have started vaccinating homeless preschoolers in welfare hotels, and the Bureau of Child Health and Immunization have opened walk-in clinics where preschoolers may be vaccinated without appointment; there have also been intensified public health education efforts.

Parent-held child health records (which include growth charts, immunization records, and records of hospitalizations and illnesses) are currently being piloted at a few major city hospitals (personal communication, D. Goldberg and N. Cunningham, 1988). Successful use by both parents and medical care providers of such parent-held records will facilitate more accurate growth monitoring and provision of good health care to patients who see a variety of providers. There is certainly a greater role for nutrition education in New York City's pediatric facilities, but for families with very limited incomes, education alone will not ensure the provision of nutritious diets. Also indicated is an expansion of such domestic versions of "food supplementation" as the Women, Infants, and Children (WIC) and State Nutritional Assistance Programs which, because of inadequate federal funds, currently serve only 46% of New York City's eligible children, infants, and pregnant women (S. Pirani, unpublished data, 1988).

The ability of GOBI-FFF type interventions to improve the health of children in New York City is borne out by the success of breastfeeding promotion campaigns within municipal hospitals and of citywide breastfeeding education efforts. Between 1979 and 1986, breastfeeding rates upon discharge from the city's municipal hospitals increased from 14% to 40%[8]; the latter rate is comparable to that observed among more affluent mothers who deliver at the city's voluntary hospitals.[8]

The "female literacy" and "family planning" components of the GOBI-FFF model are most aptly applied to the phenomenon of teenage pregnancy where they are, given a broad interpretation of female literacy, closely interrelated. Of all live births in New York City in 1986, 11% were to teenagers[4]; in this same year, most teenage pregnancies were terminated by abortion.[4] Data on use of family planning methods among all women in New York City are scarce, but the high proportion of abortions among teenagers as well as women in their early 20s suggests that the prevalence of contraceptive use is far lower than it should be. Of all teenage births in New York City, 83% were to Hispanic and nonwhite women.[4] These births are concentrated in the poorer districts: In Manhattan, the proportion of births to teenagers ranged from 12% to 20% in the poorest districts, as compared to 2% to 6% in the most affluent.[7] Educational setbacks are one of the most serious consequences of teenage parenthood. The New York City Commission on the Year 2000 reported that teenage mothers were only half as likely to graduate from high school as older mothers, and teen fathers, 40% as likely.[9] The Department of Health and many other health care providers have endeavored in recent years to increase access to family planning, prenatal care, and adolescent parenting programs for women living in poorer districts. There is the strong possibility, though, that lack of interest in finishing high school precedes and contributes to early childbearing. The very limited vocational and educational options that confront many inner city teenagers make early parenthood a more meaningful alternative. Thus, the intertwined problems of teenage pregnancy and educational setbacks among adolescent parents illustrate how limited health care interventions can be when they are implemented alone. Without a truly comprehensive program that incorporates medical, social, and economic interventions in an attempt to create for disadvantaged youths the same set of career options enjoyed by their more affluent counterparts, health care providers will continue to treat only the most superficial manifestations of a more deeply rooted environmental problem.

The emphasis here on GOBI-FFF interventions should not obscure the fact that the urban poor in the United States and, increasingly, in cities of the developing world also bear a heavier burden of chronic disease.[2,4] Improving maternal and child health and erasing the disparities in health indices between poor and more affluent urban residents may require choices between ". . . disease-specific, vertically organized health services. . . " and a ". . . multi-purpose, horizontal primary health care approach . . . ."[2] Use of "appropriate technology" in delivering health care services to mothers and children and to the general population is as important in New York City as it is in the Third World, particularly with regard to health education. More innovative means of presenting educational messages should be employed, using channels accessible to the urban poor and languages familiar to them. For any lasting impact to be achieved, however, integration of primary health care programs with broader agendas for socioeconomic development must be encouraged in both Third and First Worlds. Such integration will not occur unless there is a degree of community organization among the urban poor sufficient to allow for exertion of a cohesive political will in a responsive sociopolitical environment.[2,5]

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Demolishing the lead danger

North American physicians trained in the last decade have probably never faced the dramatic cases of severe lead poisoning reported more than 40 years ago, describing papilledema, seizures, encephalopathy, and blunted cognitive skills.[1] Lead-related deaths had virtually been eliminated by the decade of the 1970s. Data from the second National Health and Nutrition Examination Survey indicated that between 1976 and 1980 the mean blood lead level in the United States population fell significantly, paralleling a decrease in the use of lead-containing gasoline.[2] More recent population studies by Needleman et al,[3] Winneke et al,[4] and Yule et al[5] clearly demonstrated that children with low to moderate blood lead levels may suffer adverse effects on intellectual functioning and behavior.

Congressional hearings in 1970 led to the passage in 1971 of the Lead Based Paint Poisoning Prevention Act (PL 91-695) which prohibited the future use of lead based paint in residential structures. Lead based paint in both interior and exterior surfaces of housing is by far the most important source of lead poisoning for infants and preschool children. A 1978 US census survey found that 22 million occupied dwellings in the United States built prior to 1940 contained lead based paint. An additional 22 million dwellings were built between 1940 and 1960, and 75% of these units were estimated to contain lead based paint.[6] The use and manufacture of lead based paint decreased considerably after 1950, but exterior lead based paint continued to be available into the 1970s.

Removal of lead based paint is a costly, labor intensive process. A recently recognized problem is that of improper removal of lead. Heat gun torches and sanding machines can create dangerous fumes or tiny dust particles that if not properly disposed of or removed can multiply the hazard. However, nondilapidated homes have not previously been considered hazardous.

The Centers for Disease Control considers that "a lead-based paint hazard exists when . . . the surfaces being tested are chewable or damaged (cracked, chipped, loosened, chewed) paint. Lead based paint on intact walls, ceilings, or other surfaces that are not chewable does not constitute an immediate hazard."[7]

The report by Curran and Nunez in this issue of the *Journal* introduces a new twist to the problem of lead poisoning.[8] The case presented occurred during elective structural modernization which included demolition and replacement of most of the walls and plumbing in a pre-World War II single family dwelling located in a middle class neighborhood. If renovations with structural modifications of leaded walls are happening in many homes across the United States, there is a potential danger for young children to be intoxicated and permanently brain damaged unless detection and treatment promptly occur.

The American Academy of Pediatrics recommends lead screening of children nine months to six years of age living in older dilapidated housing and children of any age living in older housing when renovation is occurring.[9] Pediatricians should make a conscientious effort to inquire about the conditions of homes where young children live. Pediatricians who practice in more affluent communities should now be more astute in their approach to the screening of children and in their advice to parents who are planning renovations and remodeling. Finally, the present approach to screening for lead and to inspection of the homes and environment of lead poisoned children needs to be reviewed.

Children should no longer be used as "trouble shooters" for environmental lead. Old, dilapidated homes should be de-leaded in a safe fashion and children and pregnant women should remain out of the homes while de-leading is in progress. Renovations and remodeling of old but nondilapidated homes should also be done in a safe, nonhazardous fashion.

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Resuscitation of cardiac arrest victims by advanced life support units in Monroe County, New York

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ABSTRACT. Four hundred sixty-three cases of cardiac arrest treated in the pre-hospital setting by advanced life support (ALS) or paramedic units in Monroe County, New York, were evaluated using Eisenberg's criteria, which define factors known to be critical for successful resuscitation. Forty-eight patients met the criteria of witnessed collapse and cardiopulmonary resuscitation (CPR) within four minutes and ALS within ten minutes, with the initial rhythm of ventricular fibrillation or pulseless ventricular tachycardia. Of these, 16 (33%) patients were discharged from the hospital. This compares to 12 of 415 (3%) patients discharged who did not meet the criteria. Of the 171 patients who suffered witnessed arrests of cardiac origin, 20 survived to be discharged. This represents a successful resuscitation rate of 12%. These percentages are within the range noted for other ALS services in the United States.

(NY State J Med 1989; 89:652–654)

In 1984, cardiovascular disease accounted for almost 50% of all deaths in the United States. A total of 540,000 deaths were due to coronary artery disease, the majority of which were sudden deaths. As 60% to 70% of sudden cardiac deaths occur before the patient arrives at the hospital, and the greatest risk of death from myocardial infarction is in the first two hours after the onset of symptoms, emphasis is being placed on reducing the time elapsed between the onset of cardiac symptoms and definitive care.[1] A major tool in this effort is the paramedic-manned ambulance or advanced life support (ALS) unit. In a well-served area, an ALS unit is able to respond to any location within minutes of a call and has the capability to defibrillate, secure an airway, start an intravenous line, administer medications, monitor a patient's heart rhythm, send a rhythm strip by telemetry to a physician at a base station hospital, and discuss the case with the physician by radio.

The criteria used for this study were those proposed by Eisenberg et al[2] as a way of measuring the effectiveness of emergency medical programs in reducing mortality from cardiac arrest. The purpose of this study was to collect data on ALS-treated cardiac arrests in Monroe County and apply Eisenberg's criteria to determine the impact of the pre-hospital care system on mortality.

METHODS

Monroe County, NY, has a population of 712,000 and is served by 29 911-dispatched ambulance services, 12 of which have advanced life support capability. Fire departments act as emergency medical service (EMS) first responders in some districts. Advanced life support response times vary among urban Rochester and the suburban and rural localities. ALS technicians are New York State certified at the Category III Emergency Medical Technician level, and some have been trained in endotracheal intubation. Protocols for pre-hospital care are the responsibility of the Physicians Advisory Group and adhere to the American Heart Association Standards.

All ALS response records from Monroe County between April 1, 1986, and March 31, 1987, were gathered and screened for cases of cardiac arrest. All cases in which vital signs were recorded as absent or in which CPR was started were entered into the study. Patients pronounced dead at the scene were omitted.

The response records were screened to identify those cases that met the Eisenberg criteria. If the record indicated that the arrest was of cardiac origin and was witnessed (either seen or heard); if CPR was begun within four minutes of the arrest and ALS within ten minutes; and if the initial rhythm was ventricular fibrillation or pulseless ventricular tachycardia, the case met the Eisenberg criteria and as such represented the potential for survival. Cases involving trauma, suicide, terminal cancer, or other significant noncardiac problems were excluded. All unexplained cases of sudden collapse were assumed to be of cardiac etiology.

Hospital records for 420 (91%) cases were examined for patient outcome. Forty-three hospital charts were not reviewed as they could not be located. None of these patients fit the Eisenberg criteria and all were classified as nonsurvivors. Patient outcomes were categorized as death in the emergency department, death after admission, or survival to discharge.

Data expressed as a mean is given with the standard deviation (SD). Significance was determined using the chi-square test. Findings with $p<0.05$ were considered significant.

RESULTS

Four hundred sixty-three cases of cardiac arrest treated by ALS units were evaluated utilizing the Eisenberg criteria (Fig 1). The average age of the patients with arrests of cardiac etiology was 67 years (SD=12.9; range, 32–105 years). Sixty-two percent of the patients were male and 38% were female. Nineteen patients who fit the Eisenberg criteria were admitted. The average age of the 16 survivors was 63 years (SD=10.8; range, 48–83 years). Sixty-nine percent were male and 31% were female.

The hospital charts of 372 of the 415 cases that did not fit the

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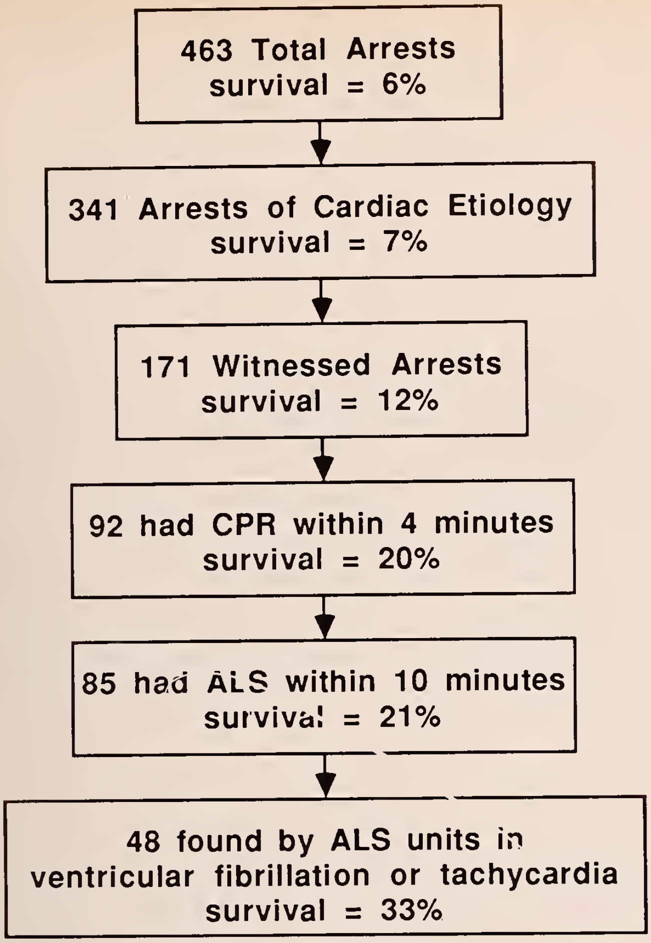
463 Total Arrests
survival = 6%
341 Arrests of Cardiac Etiology
survival = 7%
171 Witnessed Arrests
survival = 12%
92 had CPR within 4 minutes
survival = 20%
85 had ALS within 10 minutes
survival = 21%
48 found by ALS units in ventricular fibrillation or tachycardia
survival = 33%

FIGURE 1. Patient stratification by Eisenberg criteria.

Eisenberg criteria were examined. Sixty-six (18%) patients were admitted, and 12 (3%) survived. The arrests of four of these survivors were not confirmed by the ALS unit arriving at the scene, but CPR was reportedly started by civilians or first responders who then observed that the patients had been revived. Five of these survivors suffered cardiac arrest due to a noncardiac cause. The other seven survivors suffered arrests due to a probable cardiac cause but did not fit the Eisenberg criteria.

One hundred seventy-one (50%) of the arrests of cardiac origin were witnessed. Those patients who suffered a witnessed arrest had a greater chance for survival (12%) than those who suffered an unwitnessed arrest (2%) ($p<0.001$). If the cardiac arrest incident was witnessed by an ALS unit instead of a bystander, survival was 29% ($p<0.05$).

Witnessed arrests that were not witnessed by an ALS unit ("bystander-witnessed") posed a significantly greater challenge to the EMS system. Generally a civilian with no medical training witnessed such an event. Only nine (7%) of the 136 patients suffering such an arrest were released from the hospital. All nine patients were treated by two ALS services.

Civilians began CPR in only 18% of the 136 cases of bystander-witnessed arrest of presumed cardiac etiology. The initiation of CPR within four minutes is considered by Eisenberg et al[2] to be critical to survival. More patients who fit the Eisenberg criteria received their initial CPR from civilians than from dispatched EMS units (Table I). For those patients who did not fit the Eisenberg criteria, the great majority received their initial CPR from dispatched EMS units.

More time was spent at the scene for medical than for trauma cases (Table II). The most time was spent at the scene for those patients who fit the Eisenberg criteria and who were released from the hospital. Thirteen cases involving extrication difficulties were excluded.

TABLE I. Number and Percentage of Persons Initiating Cardiopulmonary Resuscitation in Bystander-witnessed Arrests of Presumed Cardiac Origin

Persons Initiating CPR	Eisenberg Patients	Non-Eisenberg Patients
Civilians	14 (41%)	10 (10%)
EMS first responders	9 (27%)	57 (56%)
ALS technician	1 (3%)	26 (25%)
Others	10 (29%)	9 (9%)

DISCUSSION

It appears that Eisenberg's criteria provide a useful tool for isolating those cases with the best chance of survival, as 57% of all survivors of cardiac arrest fell within this group. The 33% survival rate for those patients who fit the Eisenberg criteria is compatible with the 29% survival rate reported by Eitel et al[3] and below the 60% survival rate reported by Eisenberg.[2] The 60% rate was calculated based on ALS within eight minutes; however, a recalculation of our figures based on this eight-minute parameter yielded no significant change. The 12% survival rate for all witnessed arrests of cardiac origin is comparable with the 8% noted by Eitel et al[3] but lower than the 26% save rate noted by Eisenberg.[4]

The longer mean on-scene time for medical cases as compared to trauma cases (Table II) is in keeping with the philosophy that it is appropriate to attempt to stabilize a medical patient in the field, but a trauma patient requires the care that only an operating room can provide.[5] Kellerman et al[6] has indicated that patients with a cardiac arrest of medical origin have an extremely poor chance of survival if stabilization does not occur in the field. All of the 16 Eisenberg patients who survived until discharge were delivered to the hospital with organized rhythms. Of the 32 Eisenberg patients who died, only three had organized rhythms on arrival at the hospital.

Two ALS units treated the nine survivors of the 136 bystander-witnessed arrests. These two units are similar in a number of ways. Both serve densely populated areas and utilize first-responder EMS programs operated by fire departments with two- to four-minute average response times. The other five ALS units had no successful resuscitations of patients in which arrests were witnessed by bystanders. These five units, in general, serve larger, less densely populated areas. First-responder fire department EMS programs are generally not available. These units did have 11 bystander-witnessed arrests that fit the Eisenberg criteria, but none of these patients survived.

The above observations may be a reflection of the system that must be in place to aid successfully the victim of a bystander-witnessed cardiac arrest. The first component

TABLE II. Average Advanced Life Support On-scene Time in Minutes by Type of Arrest

Type of Arrest	(n)	On-Scene Time (mean ± SD)
Medical	(416)	23 ± 11
Trauma	(34)	10 ± 6
Eisenberg	(48)	25 ± 10
Eisenberg survivors	(16)	27 ± 9

is the citizen. This person must be educated in how to access the EMS system and perform CPR promptly. Early CPR is vital to enlarge the window of time in which defibrillation and other ALS therapies can benefit the patient. It was noted in this study that the civilian is the best tool for achieving initial CPR within four minutes (Table I). The process of civilian recognition of the emergency, activation of EMS, dispatching, and travel time makes inappropriate the reliance on trained EMS first responders to begin CPR within four minutes of an arrest. Less than 20% of patients suffering a bystander-witnessed arrest of presumed cardiac etiology received initial CPR from a civilian. Additional civilian CPR training in Monroe County is needed.

First responding EMS units do, however, provide vital services. These units are usually fire department vehicles manned by firefighters cross-trained in EMS. There are many more fire stations than ambulance bases, and thus fire department first-responders are often the first trained helpers to arrive at the scene of an emergency. These units provide manpower to take over the physically tiring task of CPR from civilians; they also provide oxygen therapy and suction devices to assist with airway management.

There are challenges yet to be met to allow the ALS component to be fully effective in dealing with out-of-hospital sudden cardiac death in Monroe County. These include improving civilian education on how to access the EMS system, increasing the number of civilians who can perform CPR, increasing the number of first responders, and improving ALS coverage.

This study demonstrates the effectiveness of the emergency medical services system operating in Monroe County when all components of the EMS system function in a rapid and coordinated fashion to assist victims of cardiac arrest.

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Paraesophageal hernia

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ABSTRACT. We have reviewed our experience with eight patients with paraesophageal hernias surgically repaired at this institution during an 18-month period. Three of these patients required emergent operation, and although the morbidity was higher in this group than in those undergoing elective surgery, no mortalities occurred in either group. The potential for the development of life threatening complications warrants immediate surgery on patients who are diagnosed to have paraesophageal hernias, even if asymptomatic. We recommend a transabdominal approach for the performance of a crural repair and an antireflux procedure.

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Paraesophageal hernia is an uncommon disorder of the diaphragmatic hiatus. The clinical picture and associated complications are quite distinct from the more commonly encountered sliding hiatal hernia. The true incidence is unknown, since most paraesophageal hernias are initially asymptomatic and remain undetected until complications arise. It is estimated that paraesophageal hernias represent approximately 2% of the hiatal hernias demonstrated radiologically[1] and from 3% to 8% of the hiatal hernias that require surgical repair.[1–3] Paraesophageal hernias are characterized by the essentially normal position of the esophagogastric junction, in contrast to sliding hiatal hernias, in which the esophagogastric junction is displaced into the chest. As a result, patients with paraesophageal hernias infrequently present with symptoms of gastroesophageal reflux.

The indication for surgical repair of a paraesophageal hernia is the diagnosis itself and should not be predicated on the presence or absence of symptoms. Failure to intervene early may result in the life threatening complications of gastrointestinal bleeding or strangulation with obstruction, which are associated with an unacceptably high mortality and morbidity.[2–4]

The surgical repair advocated for paraesophageal hernias ranges from simple closure of the hiatus to crural repair and routine use of an antireflux procedure. This report reviews eight cases of paraesophageal hernias surgically repaired at this institution during an 18-month period.

PATIENTS AND METHODS

All charts belonging to patients with the diagnosis of paraesophageal hernia who underwent surgery at this institution from January 1986 through May 1987 were retrieved for review. Eight such patients were identified and constitute the basis of

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TABLE I. Presenting Complaints in Eight Patients with Paraesophageal Hernia

Upper gastrointestinal hemorrhage	2
Acute exacerbation of abdominal pain	2
Gastric outlet obstruction	1
Acute abdomen	1
Dyspnea on exertion	1
Chronic anemia	1
Total	8

this report.

These eight hernias were found in five women and three men. The mean age of patients was 67 years (range, 55–76 yr). Three patients underwent emergency surgery for acute abdomen, upper gastrointestinal bleeding, or gastric outlet obstruction. The remaining five patients underwent elective surgery once the diagnosis of paraesophageal hernia was established.

RESULTS

The chief complaints at the time of admission are listed in Table I. Of the two patients who presented with an upper gastrointestinal hemorrhage, one was being treated for peptic ulcer disease, and the other was given a diagnosis of hiatal hernia. Pertinent past history is shown in Table II. Seven of the eight patients had previous complaints of epigastric pain. Of interest is the fact that 62.5% of these patients were diagnosed as having hiatal hernia and were being treated with antacids and H_2 blockers at the time of admission.

The chest film was diagnostic for paraesophageal hernia in two of eight (25%) patients (Fig 1). An upper gastrointestinal series (UGI) was obtained in seven of eight patients and was diagnostic in all (Figs 2, 3). Upper endoscopy was performed in seven of eight patients, and the correct diagnosis was made in six of seven (85%) of the cases. The patient in whom the endoscopic procedure was inconclusive was noted to have "distorted anatomy"; however, the endoscopist failed to make the diagnosis, which was subsequently made by UGI.

Exploration revealed that the majority of patients had stomach and greater omentum in the hernial sac. The patient who presented with an acute abdomen was found to have strangulated transverse colon within the paraesophageal hernia. In no instance was difficulty encountered in reducing the hernial contents via an abdominal approach.

The surgical procedures performed are listed in Table III. Antireflux procedures were performed in five of eight (62.5%) of the patients (four Nissen fundoplications, one Belsey-Mark IV). Simple crural repair without an antireflux procedure was performed in the remaining three patients. A thoracic approach was used in two of eight patients. The diversity of procedures performed reflects each surgeon's preference.

Most of the complications occurred in those patients requiring emergent operation (Table IV). Of note, the only complication encountered in the group undergoing elective surgery was pneumonia, which resulted in eventual tracheostomy for ventilatory support; this complication was related to the use of a thoracic approach for repair of the paraesophageal hernia. No mortalities occurred in either the elective or emergency group.

TABLE II. Previous History in Eight Patients with Paraesophageal Hernia

	n (%)
Epigastric pain	7 (88)
History of hiatal hernia	5 (63)
Postprandial fullness	4 (50)
History of peptic ulcer/gastritis	4 (50)
Chronic anemia	3 (38)
Chest pain	2 (25)
Dyspnea on exertion	1 (13)

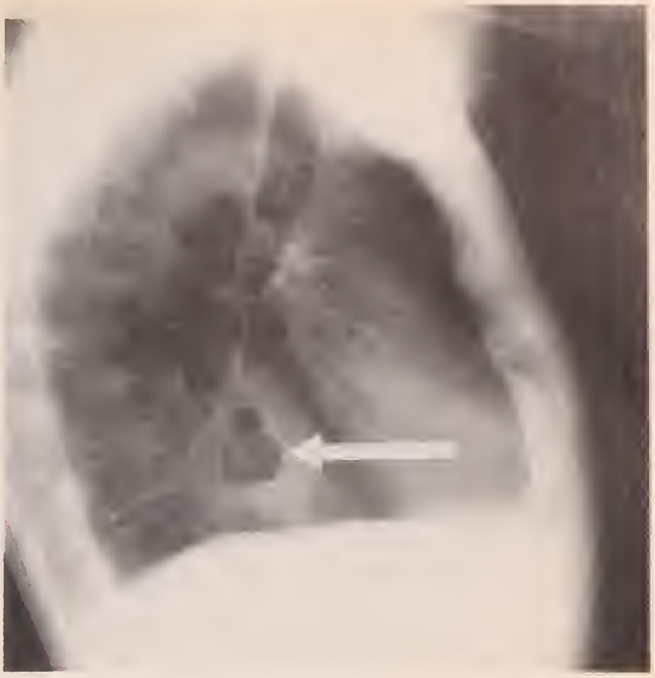

FIGURE 1. Lateral view of the chest reveals the presence of an air-fluid level in the retrocardiac area (arrow). The esophagogastric junction appears to be in a relatively normal position.

DISCUSSION

The etiology of paraesophageal hernias remains speculative; however, the rarity with which these hernias occur in patients younger than 40 years suggests that this is an acquired rather than a congenital condition. The average age of the patients in this report was 67 years.

Paraesophageal hernias should be clearly distinguished from the very rare parahiatal hernia. This latter condition

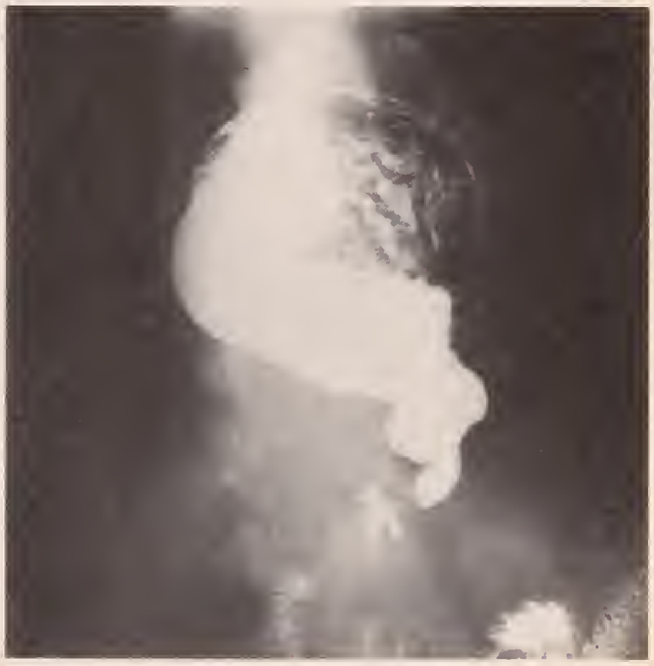

FIGURE 2. Typical appearance of a paraesophageal herniation of the stomach.

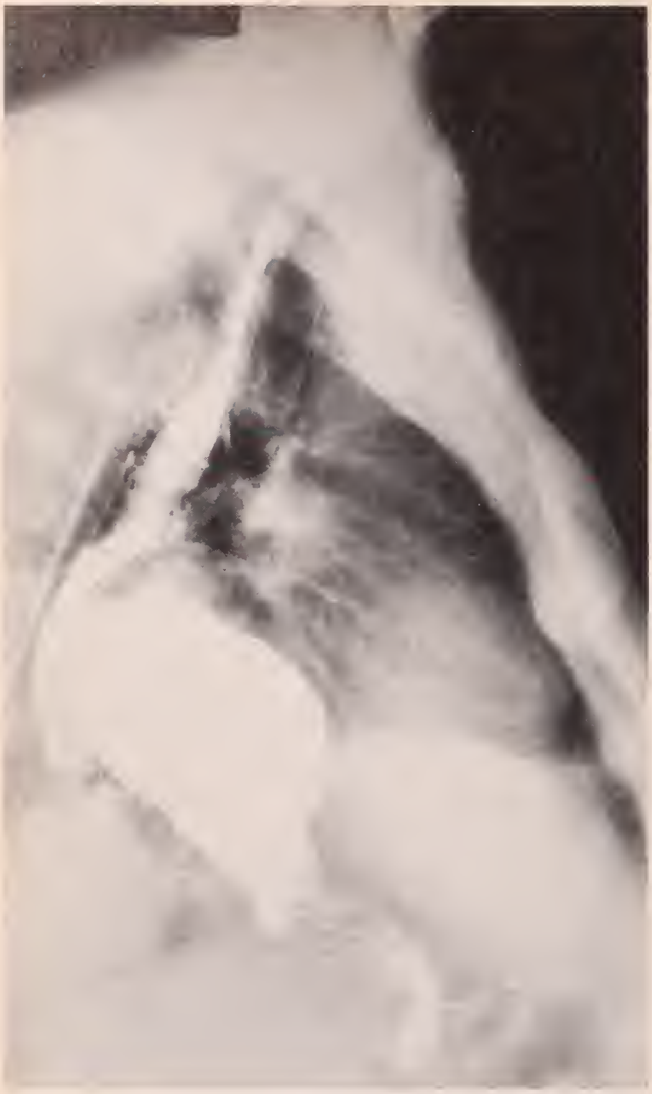

FIGURE 3. Lateral view from the upper gastrointestinal series allows visualization of the paraesophageal hernia as well as the position of the esophagogastric junction.

has been characterized as a diaphragmatic defect which occurs in proximity to but quite separate from the esophageal hiatus. Whether, in fact, parahiatal hernias exist has been questioned by some.[1,5]

The mechanism resulting in the formation of a paraesophageal hernia is presumed to be related to a relative increase in abdominal pressure, resulting in a defect between the diaphragmatic crura, alongside the esophagus and esophagogastric junction. As this defect enlarges, the most mobile portions of the stomach, the body and antrum, are pulled toward the mediastinum in response to the negative intrathoracic pressure developed with each inspiration. This repetitive action eventually weakens the pyloric and duodenal attachments, with gradual displacement of the stomach into the chest. As the stomach continues this pendulum motion between the abdominal and thoracic cavities, the posterior attachments of the esophagogastric junction may become weakened, allowing the esophagogastric junction to migrate cephalad, the basis of the mixed form of paraesophageal hiatal hernia.[5] The inverted position of the stomach in the chest with a relatively nondisplaced esophagogastric junction, as occurs with paraesophageal hernias, should be clearly distinguished from the nonrotated intrathoracic stomach associated with sliding hiatal hernias. The potential complications of the former, which do not occur with the latter, include organoaxial rotation, volvulus, and obstruction of the stomach.

TABLE III. Surgical Procedures Performed in Eight Patients with Paraesophageal Hernia

Laparotomy, crural repair, Nissen	3
Laparotomy, crural repair, vagotomy and pyloroplasty	1
Laparotomy, crural repair, colectomy	1
Laparotomy, crural repair, vagotomy and pyloroplasty, Nissen, splenectomy	1
Left thoracotomy, crural repair, Belsey Mark IV	1
Left thoracotomy, crural repair	1
Total	8

TABLE IV. Complications of Surgery in Eight Patients with Paraesophageal Hernia

Emergent Surgery (n = 3)	
Splenectomy	1
Wound infection	1
Hemorrhagic gastritis	1
Elective Surgery (n = 5)	
Pneumonia with tracheostomy	1

Despite the degree of anatomical distortion that occurs, many patients are either asymptomatic or have relatively vague complaints until a major complication develops. Borchardt described the triad of epigastric pain, the inability to vomit, and the inability to pass a nasogastric tube, which is pathognomonic for paraesophageal hernia.[2] Unfortunately, Borchardt's triad was present in only 37% of the patients described here, which emphasizes the difficulty in establishing the diagnosis on clinical grounds alone.

The most reliable study in our experience was the UGI, which established the diagnosis in all cases in which it was performed. Although not as reliable as the UGI for diagnosing paraesophageal hernias, endoscopy is a valuable adjunct in evaluating associated mucosal pathology. Anemia or occult gastrointestinal blood loss occurring in association with paraesophageal hernias is not uncommon and is secondary to a gastritis of the herniated portion of stomach or a gastric ulcer where the stomach is compressed by the esophageal hiatus.[1,2]

The natural history of paraesophageal hernias is such that if left untreated, catastrophic complications will occur. This was clearly demonstrated by Skinner and Belsey[3] in their longitudinal study of 21 asymptomatic patients with paraesophageal hernias in which six (30%) died from complications directly related to the hernia itself. Similarly, three of eight (37.5%) of the patients described here presented with life threatening complications (gastric hemorrhage, gastric outlet obstruction, strangulated hernial contents). Our experience is consistent with that reported in the surgical literature in general.[2,5] Once the diagnosis of paraesophageal hernia is made, expeditious surgical repair should be performed, regardless of the absence of any symptoms.

Although it is well accepted that paraesophageal hernias should be repaired, some controversy exists with regard to the approach as well as the preferred procedure to be used. Review of our experience suggests that an abdominal approach is associated with less morbidity than a thoracic approach. The hernia is usually bounded by a well-formed sac, and no difficulties are encountered in reducing its contents into the peritoneal cavity through a laparotomy incision. In contrast, a thoracic approach, particularly in this older patient population, may result in significant pulmonary complications.

The main issue with regard to the procedure itself revolves around the necessity for an antireflux procedure in addition to a crural repair. Hill and Tobias[1] advocated a simple crural repair and emphasized that mobilization of the esophagogastric junction should be avoided to prevent the subsequent development of a sliding hiatal hernia. However, Walther et al[6] demonstrated that 60% of 16 patients with paraesophageal hernias and nondisplaced esophagogastric junctions had incompetent lower esophageal sphincters, as assessed with 24-hour esophageal pH monitoring. We agree with these authors who suggest that an antireflux procedure should be performed in all emergent cases of paraesophageal hernias and in those situations in which preoperative lower esophageal sphincter studies cannot be obtained.

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PRESENT STATUS AND RECENT ADVANCES IN SURGERY OF THE ESOPHAGUS

...Hernias through the esophageal hiatus usually have been divided into three types—the sliding hernia, agreed to be the most common; the para-esophageal, implying that the esophagogastric junction remains at the proper level but the fundus forms the herniated part, and the short esophageal type. Considerable discussion has centered on the frequency of occurrence of the short esophageal type, some authors maintaining that it is hardly ever seen, others meeting it fairly frequently. According to Humphreys and his associates, the true congenital short esophageal hernia is characterized by the fact that the intrathoracic portion of the stomach derives its blood supply from the thoracic aorta but not from the branches of the celiac artery. Some evidence has been forwarded that the short esophagus encountered with symptomatic hiatus hernias is the result of peptic esophagitis rather than the cause of it....

The evaluation of the patient with hiatus hernia must include endoscopy to prove or disprove the presence of esophagitis, in addition to ruling out symptomatic disease in the organs already enumerated. Once satisfactory evidence exists that a symptomatic hiatus hernia is present, surgical correction should be recommended.

For the uncomplicated hernia the best results have been obtained utilizing a transthoracic approach as described by Allison. For the cases which have an esophagus which is too short to allow reduction of the stomach below the level of the diaphragm, Effler and Groves recommend transplanting the esophagus anteriorly into a new hiatus combined with crushing of the phrenic nerve. Collins recommends splitting the cardia to form a gastric tube distal to the esophagus allowing reduction of the remainder of the stomach below the diaphragm. Fisher and Johnson report 7 patients with a short esophageal hiatus hernia which they treated by distal gastric resection, with the result that the hernia spontaneously disappeared, apparently from the reduction achieved in the acid-peptic factor. Strictures secondary to hiatus hernia and esophagitis introduce another factor difficult to overcome. Lindskog and Kline advise distal esophagectomy combined with vagectomy, extensive proximal gastric resection, and gastrojejunostomy for irreversible stricture formation. Others advocate resecting the involved area along with the proximal part of the stomach and interposing a jejunal segment between the esophagus and pyloric part when stricture formation has occurred.

Whatever the procedure chosen for irreversible stricture formation, it has to fulfill two conditions: it must eliminate the problem of recurrent esophagitis and leave sufficient food reservoir to insure adequate nutritional status. Both these conditions are difficult to fulfill, and in some ways this is still an unsolved problem....

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(*NY State J Med* 1959; 59:280–288)

The crisis in New York City's perinatal services

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Infant mortality continues to be a priority on the public health agenda. Although rates have generally declined during the past decade, they remain high across the nation. In 1987, New York City's rate increased for the first time in 14 years, from 12.8/1,000 live births to 13.1/1,000.[1]

High infant mortality rates, both nationally and locally, have promoted a variety of responses, most of which have focused on expanding access to prenatal care. While access to prenatal care is essential, a comprehensive system of maternal and infant services, with adequate capacity, access, and quality, is necessary to reduce infant mortality. Examination of New York City's perinatal health care system reveals a crisis in both capacity and access—a system on a collision course with the growing reproductive health needs of the citizenry.

The evidence documents that the "crisis" could have been predicted and is probably just beginning. Increases in the rates of poverty, drug abuse, and homelessness, and the resulting increase in morbidity and mortality; rising rates of AIDS, sexually transmitted diseases, tuberculosis, and hepatitis; increases in births, birth rates, and birth-related illness and deaths; and changing practice patterns resulting in more labor intensive and technologically advanced intensive care, are all factors in the increasing demand for maternal and infant services in New York City. This increased need has coincided with cutbacks in services and funds, reductions in numbers of beds, and staff shortages.

THE "DEMAND" SIDE

Birth Rates. A critical factor in defining need for perinatal services is the number of births. The number of New York City births, and the birth rate per population, have risen every year since 1980 (Fig 1). There was a 19% increase between 1980 and 1987, nearly 44% of which occurred in the past two years.[1] This pattern of continuing increase prevails nationally.

The increase in number of births has been true for all racial, ethnic, and age groups in New York City except teens, although the magnitude of the increase varies. Birth rates have increased by 5% among whites and Hispanics and 10% among blacks. While teen birth rates declined between 1985 and 1986, there was a 14% increase in births to women over the age of 30 among both black and white women. The increase has been most striking in some communities, typically those that are also experiencing an increase in a range of social, economic, and health status problems. For example, between 1980 and 1987, Central Harlem experienced a 51% increase in number of births, and other poor communities experienced increases between 30% and 49%. The smallest increases were found in some of the city's white middle class neighborhoods, several of which had increases of less than 15%.[1]

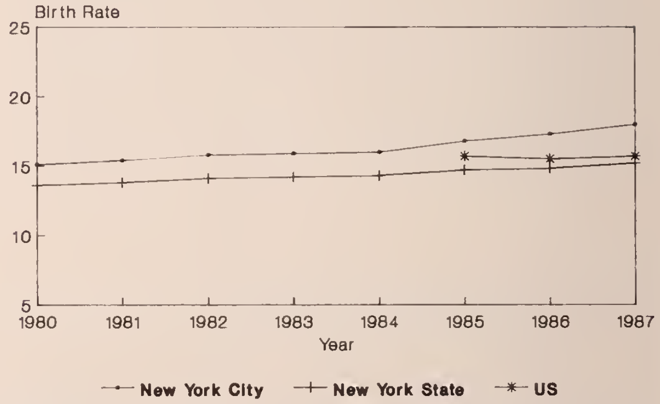

FIGURE 1. Birth rates, New York City, New York State, and United States.

Neither city nor state projections anticipated the magnitude of this increase. Projections for the ethnic and age composition of New York City's population vary by source, making birth rate projections inconsistent (Figs 2,

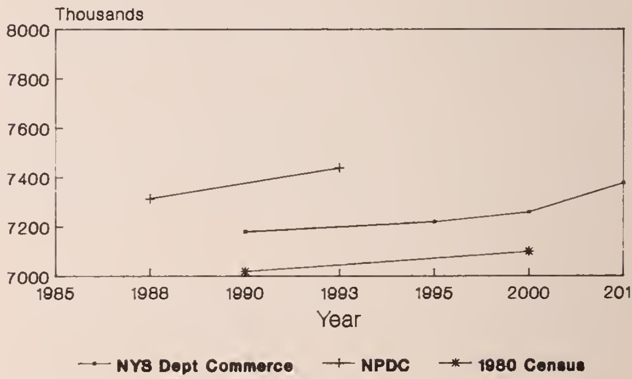

FIGURE 2. Projected total population by year, New York City.

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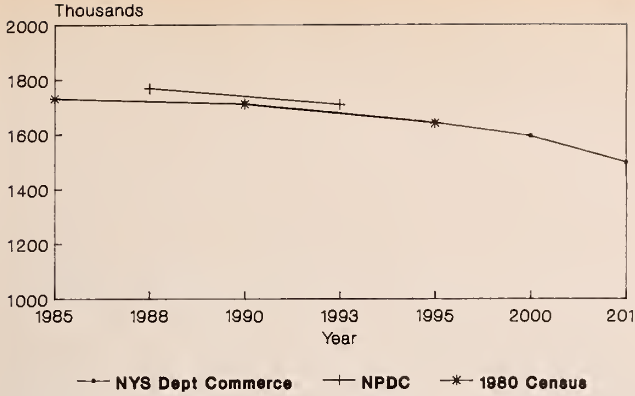

FIGURE 3. Projected female population, ages 15–44 by year, New York City.

3). The New York State Department of Commerce projected a 3.4% increase in the number of women of childbearing age in New York City between 1980 and 1990,[2] but did not project any increase in the expected number of births.[3] (It is important to note that the New York City Health Systems Agency (HSA) used these projections in determining service needs.) The New York City Planning Commission, on the other hand, predicted that the birth rate would rise between 1980 and the year 2000, although the number of births would decline. For the decade 1980 to 1990, the commission projected 1,040,000 births.[4] As there were more than 920,000 births in New York City between 1980 and 1987, the commission's projection for the decade was surpassed by the end of 1988.

The number of women of childbearing age (15–44 yr) has been increasing throughout the decade, as "baby-boomers" have come of age. The 1980 Census reported 1.69 million women in New York City in this age group. However, the city, the state, and the National Planning Data Corporation (NPDC) concur that this group will have peaked in size at some point between 1985 and 1990 (reaching somewhere between 1.72 and 1.77 million), although these forecasts differ in the rate, magnitude, and starting point of the decline in the number of women of childbearing age. New York City has projected a total of 1.46 million women in this age range in the year 2000. New York State estimates the figure to be closer to 1.6 million.[2,4,5]

Also predicted to change between 1980 and 2000 is the racial and ethnic mix in the city. Blacks, Hispanics, and Asians were reported to be 47% of the population in 1980. The Year 2000 Report projects this proportion to rise by 13% by the end of the decade.[6] According to NPDC, the majority of the increase in the racial and ethnic minority population will be among Hispanics, as the black population is projected to decline. As fertility rates have been higher for racial and ethnic minorities, these projected changes, if accurate, will result in an increase in the proportion of these births. In 1986, 28% of all births were to Hispanics, 33% were to black non-Hispanics, and 33% were to whites.

Looking primarily at the declining cohort of women of childbearing age, it would appear that the total of births could begin to decline in the next few years in New York City, and therefore the need for maternity and infant services might also decline. However, the growth in the rate of increase in the last two years suggests that caution should be applied to any such assumptions, as dramatic changes in birth rates may result from factors such as changing immigration patterns or patterns of sexual behavior.

Poverty. It is no news that the poor suffer greater health and social problems than the general population and have less access to services. The number of poor people, particularly the number of poor women and children, has grown significantly in New York City in the 1980s. Twenty-five percent of New York City residents now live below the federal poverty level.[6] In some neighborhoods, that proportion rises to 50%. Two of every five children in the city are poor.[7] The economic status of female-headed households also deteriorated, with over 200,000 of these families, close to 65%, living below the poverty level. These figures are all higher than those for the state or the nation, and they are continuing to rise.[6,7] Also rising is the number of homeless men, women, and children living in the streets, shelters, and welfare hotels of the city, currently estimated at over 70,000 people. Approximately two thirds of the hotel and shelter population, about 19,000 persons, belong to single-parent households headed by women (personal communication, Coalition for the Homeless, New York, NY, April 1988).

The proportion of unmarried women giving birth, a marker for social risk, rose from 34.6% in 1980 to 40.3% in 1987. Approximately 45% of births are to women who qualify for Medicaid or have no health insurance. After reaching a low of 8.4% in 1984, the proportion of infants weighing less than 2,500 grams at birth has risen steadily in each subsequent year. Provisional New York City data indicate that 9.4% of infants born in 1987 weighed less than 2,500 grams. Poverty and homelessness have been shown to be associated with diminished access to health care, and poor health status and pregnancy outcomes. Thus, it is not surprising that a recent study by the United Hospital Fund (UHF) found the bulk of the increase in hospital utilization, between 1985 and 1987, to be by residents in low-income communities, with a continuing decline in utilization by the more affluent. In areas with more than 40% of the population living below the poverty level, UHF found a 4.6% increase in patient days, while patient days were down 3.3% in areas with levels below 10% poverty.[8]

AIDS and Substance Abuse. AIDS has emerged as a significant factor in New York City's perinatal morbidity and mortality profile. An increase in the number of AIDS-related infant deaths was observed in 1987, with 2.2% of infant deaths citywide considered to be AIDS-related.[9] AIDS-associated maternal deaths have also been reported.[10] Recent New York City and New York State Health Department studies found 1.5% of all newborns in New York City seropositive for HIV.[11]

While 75% of all pediatric AIDS cases were the offspring of intravenous drug users, the problems of substance abuse for women and infants extend beyond AIDS. Maternal substance abuse has a variety of well-known harmful physiologic effects on the women, the fetus, and pregnancy outcome.[13-15] A recent birth and death certificate review found infant mortality rates among babies with reported maternal substance abuse to be nearly 2.5 times higher than the citywide rate. Only 42% of these women were reported to have received prenatal care, compared to 75% citywide. Thirty percent of infants weighed less than 2,500 grams, more than triple the citywide proportion.

Since 1981, there has been a dramatic increase in the reporting of maternal substance abuse on birth certificates, from 6.7 per 1,000 live births in 1981 to 20.3 in 1987. The largest increase was seen between 1985 and 1986.[16] In 1985, there were 1,227 birth certificates reporting maternal substance abuse; in 1986, the figure was 2,069.

Reports from chiefs of neonatology around the city indicate that the number of babies born with in utero crack exposure is continuing to rise rapidly. At a Brooklyn hospital, 15% of the women in prenatal care say they use cocaine (personal communication, chiefs of obstetrics and gynecology, New York, NY, March 1988). At a Bronx hospital, an anonymous urine toxicology screen of 200 women in labor revealed 21% (42) to be positive for cocaine.[17]

Although human immunodeficiency virus (HIV) infection of women and newborns has serious long term consequences for their health and need for medical care, in New York City it is through the association between HIV and drug use that the impact on perinatal services is chiefly manifested. Drug-exposed newborns are using more neonatal intensive care unit (NICU) and normal nursery days because they are more likely to be of low birth weight, to experience withdrawal symptoms, and to be the subject of prolonged negotiations concerning custody and placement.

The United Hospital Fund reports a 14.8% increase in newborn nursery days between mid-1985 and mid-1987, largely due to an increase in the number of newborns needing prolonged intensive care. Drug withdrawal was found to be one of the ten most common secondary diagnoses among "long stay" babies (ie, babies staying more than 21 days), affecting one out of seven of these infants. From mid-1985 to mid-1987, UHF found a 50% increase in the number of long stay baby discharges (from 457 to 687) and a 3% increase in the number of short stay baby discharges (from 16,136 to 16,850).[8]

While a minimal abstinence syndrome has been reported in crack-exposed newborns, the major medical reason for increased length of stay in these infants is low birth weight. Both MacGregor et al[18] and Chervkuri et al[19] have reported significant increases in proportions of crack-using women who deliver prematurely, and whose infants are low birth weight and/or growth retarded compared to controls. Habel's birth certificate analysis confirms the elevation in the low birth weight proportion.[16]

When the first impact of the crack epidemic was felt in 1985, the demand for placement for drug-exposed babies removed from maternal custody increased dramatically, as evidenced by the number of "boarder babies" in hospitals awaiting placement. The problem peaked in the winter of 1986, causing severe strains on nurseries and NICUs. New York City's Special Services for Children (SSC) reported 239 infants without "special needs" awaiting placement in hospitals in February 1987. To meet this growing problem, the placement system was rapidly expanded, with recruitment of foster parents and the establishment of "congregate care" facilities. By the end of October 1988, SSC reported 70 such infants awaiting placement. However, the number of infants with "special needs" increased during the same time period, from 34 to 68. While the response to the crisis effectively reduced the number of boarder babies in the city's hospitals, if the number of drug-exposed babies doubles as projected, the boarder baby crisis will resurface, adding significantly to the strain on already overcrowded NICUs.

Sexually Transmitted Diseases (STDs). The rapid rise in the incidence of sexually transmitted diseases represents another sign of the deteriorating health status of poor women and infants. Between 100,000 and 150,000 cases of STDs were reported in New York City last year.[20]

The gonorrhea rate has been rising since 1979. Between 1984 and 1986 alone, there was a 46% increase in the number of cases reported.[1] The incidence of syphilis is also rising dramatically—more than 60% between 1986 and 1987.[20] Postulated causes of the increase are increases in antibiotic resistant strains of gonorrhea and syphilis, lack of adequate follow-up, association with the HIV virus, an increase in the exchange of sex for drugs, and a shift in resources from STD prevention to AIDS.

An alarming increase in congenital syphilis has also been reported both nationally and locally. In New York City, the number of reported cases has more than tripled since 1984[20]; there is significant under-reporting according to neonatologists. In more than 40% of the reported cases, the mothers had not received prenatal care.

While the rise in sexually transmitted diseases is a national problem, New York City accounted for nearly one third of the total national increase in reported cases of infectious syphilis between 1986 and 1987. As with AIDS and substance abuse, it is overwhelmingly a problem among the poor: rates for blacks and Hispanics have increased 150% and 73%, respectively. The proportion of females has also increased from 27% to 34% of the total, with an actual increase of over 160% between 1986 and 1987.[20]

The sequelae of untreated STDs include premature rupture of membranes, premature births, pelvic inflammatory disease (PID), and ectopic pregnancies. A number of New York City hospitals are reporting an increase in admissions for PID. One Brooklyn hospital reported a 150% increase in monthly PID admissions between 1981 and 1987.[21] Ectopic pregnancy is now the fourth leading cause of maternal death in New York City. Between 1975 and 1980, the estimated total of ectopic pregnancies rose to 2,080.[22] Between 1985 and 1986, there was a 13.6% increase in ectopic pregnancies.[23]

Changing Patterns of Practice. Over the past several years, perinatal medicine has become increasingly more technologically sophisticated. Interventions are occurring earlier and more frequently. Additionally, the political climate, as typified by the Baby Doe case, has increased the pressure on neonatologists and nurses to employ heroic measures. At the same time, defensive medicine has become common practice, particularly in obstetrics, in which malpractice rates are skyrocketing. Cautious obstetricians are giving more care, and more intensive care. For example, the rate of cesarean sections has risen from 16.5% in 1980 to 24% in 1986 nationally.[24,25] These changes, along with the increase in births and in the number of high-risk mothers and infants, place increased demands on the delivery system and its workers.

NEED BECOMES DEMAND: PATTERNS OF UTILIZATION

The confluence of factors described has resulted in a dramatic increase in demand. In obstetric services, the citywide occupancy rate has risen steadily since 1981 (with a dip in 1986) to its highest level, 86.6% in the third quarter of 1987.[26] The recommended occupancy rate for maternity services is 75%. Many of the city's busiest obstetric services are experiencing occupancy rates over 100%. One public hospital's occupancy rate in obstetrics reached 165% in the third quarter of 1987. A voluntary hospital serving a large AIDS and drug-using population saw its occupancy rate rise 12% between the second and fourth quarters of 1987. Numerous other facilities are reporting rates in the 90%+ range. Throughout the city, while occupancy rates fluctuate, they remain extremely high (more than 10% higher than the state as a whole) (Table I).[26]

Almost every hospital in the city with an obstetric service experienced an increase in the number of births between 1980 and 1987. However, the increase was greater in the public sector. Births to women delivering in the city's municipal hospitals increased nearly 30% between 1980 and 1986, while the voluntary sector experienced an 11% increase.[1] In a "double bind," the municipal hospitals can be fined if care does not conform to standards, but as "the provider of last resort," they can also be cited for turning people away, even if they are filled and have concerns that care will therefore be inadequate.

While maternity services throughout the city are at high levels of occupancy, the situation is more severe for neonatal services. In neonatal units, occupancy rates are over 100%. In 1987, the NICU occupancy rate in New York City increased from 105.3% in the first quarter to 111.2% at the end of the year. One hospital after another reports rates of 127%, 139%, 104%, etc, with one reaching 297.2% in the third quarter of 1987.[26]

From mid-1985 to mid-1987, newborn discharges rose 4.4%, and unlike obstetrics (for which length of stay declined 5.7%), length of stay increased 9.3%, and patient days increased 14.8%. The biggest increase in days was among "long stay babies" (those staying longer than 21 days), who represented only 4.1% of newborn discharges in 1987, and used 35% of all newborn days. Both the number of long stay babies and the length of their stays have increased since 1985.[8] Given the overwhelming demand, it is not surprising that many of these NICUs closed their services to admissions numerous times during 1987.

The Supply Side. While increased demand is contributing significantly to the crisis in maternal and infant services, a declining supply is also taking its toll. A New York State policy of "downsizing" the state's hospital system resulted in the elimination of more than 4,000 acute care beds since 1981. As recently as 1987, the position of the New York State Commissioner of Health has been that New York City had too many hospital beds. It was not until December 1987, when faced with a citywide occupancy rate of 95%, that the commissioner called for an immediate reversal of this policy, and requested proposals from the hospitals for the reopening of acute care beds.[27]

Maternity beds have been cut by approximately 7% (compared to a 9% overall reduction in acute care beds between 1981 and 1986). The total number of obstetric services has declined significantly, from 53 in 1980 to 44 in 1987. The New York State Department of Health reports no change in the number of certified NICU beds between 1984 and 1987 for the city, although the Health and Hospitals Corporation (HHC) reports a 2.7% loss during this period.

Availability of ambulatory services can be inferred through waiting time for the first appointments. The waiting time for a first prenatal or gynecology appointment is as long as 60 days in some outpatient clinics in New York City (personal communication, chiefs of obstetrics and gynecology, New York, NY, 1988).

With growing crack and AIDS epidemics, drug treatment programs have become an important component of the maternal services delivery system. In 1985, the New York City Health Systems Agency estimated the number of additional treatment slots needed in New York City to be 30,000. This estimate was developed before the crack epidemic. At present, New York City has little to offer a pregnant woman with a drug habit because of the shortage of treatment spaces, which are financed and designated by the state. There are waiting lists in most programs, and few are geared to address the growing percentage of drug abusers using cocaine. In addition, many drug treatment programs do not accept pregnant women or those with small children. Those few that do accept pregnant women have traditionally not integrated the necessary health care services into their programs, either through on-site provision of prenatal care or through firm linkages with medical providers. Thus, pregnant women motivated to get off drugs may find themselves with no place to go for drug treatment; pregnant women receiving drug treatment may remain unconnected to a prenatal care

TABLE I. Occupancy Rates: Neonatal, Medical/Surgical, and Obstetrics, New York City (NYC) and New York State (NYS), 1985–1987*

	Neonatal		Medical/Surgical		Obstetrics	
	NYC	NYS	NYC	NYS	NYC	NYS
1985	94.8	96.5	84.4	81.6	82.4	74.9
1986	104.6	101.6	81.1	78.7	83.5	73.3
1987	109.5	103.2	85.1	84.3	81.4	71.9

* Source: New York State Department of Health Quarterly Occupancy Reports, 1985–1987.

provider.

Staff Shortages. Compounding the bed and ambulatory care shortages is the growing shortage of health care workers. The problem is particularly acute for maternal and infant services because of shortages of obstetricians and nurse midwives.

The American College of Obstetrics and Gynecology (ACOG) surveyed practicing obstetricians in 1985 and again in 1987 to assess the impact of increased malpractice costs and liability claims on practice patterns. ACOG found that 41% had changed their practice patterns as a result of malpractice considerations, that 12.4% of those affected no longer practice obstetrics, and that 12.9% had decreased the number of deliveries. With premiums rising every year—up 238% in the past five years—a diminishing supply of obstetricians appears likely to be an ongoing problem.[28]

Compounding the problem in New York City is the dramatic decline in the number of obstetrics/gynecology residents in New York City. From 1975 to 1985, there was a drop of 20.2%. (In contrast, the total number of residents for all specialties in New York City decreased 1%.)[29]

The Report of the New York State Commission on Graduate Medical Education found that the proportion of Ob/Gyn residents trained in New York State is lower than the overall proportion for all residencies.[30] Ironically, regulations resulting from this report are expected to further reduce the number of residency slots in the city. New accreditation requirements for training programs, and reorganization of these programs, with more control going to major medical centers, is expected to further cut HHC's supply of physicians. The report's recommendation to reduce the supply of foreign medical graduates (FMG) in New York City will exacerbate the shortage. The commission proposes using nonphysician providers as substitutes, despite the fact that the supply of midwives, physicians' assistants, and nurse practitioners is already inadequate.

The Report of the New York State Ad Hoc Advisory Committee on Emergency Services, commonly known as the Bell Commission report, recommends limiting the number of consecutive hours that residents can work.[31] The state has indicated that it will pay for any increases in ancillary staff required by these regulations. Budget deficits and staff shortages make this unlikely.

The growing shortage of registered nurses in New York City and across the nation has been discussed extensively. Low salaries, inadequate career advancement, and difficult working conditions have made recruitment and retention a problem for most facilities. While most New York City obstetric and neonatal services are reporting nursing vacancies, municipal facilities experience disproportionate difficulty recruiting and retaining nurses. Nursing vacancy rates in municipal hospitals are nearly twice as high as those for the voluntary hospitals.

In an effort to address the nursing shortage, hospitals have had to hire more per diem nurses. These nurses (some of whom are hospital nurses working extra shifts) are more costly than the permanent full-time staff they are hired to replace.

A recent contract between New York City and the New York State Nurses' Association should go a long way in closing the gap between the voluntary and the municipal hospitals. This contract provides for parity in salaries with 14 selected voluntary hospitals, increases in longevity payments, and improvement in shift assignment procedures. The New York City Nursing Task Force, with representatives from the health and education sectors, is also examining ways to improve recruitment of nurses and other health care workers.

In addition to physician, housestaff, and nurse shortages, maternity services report high vacancy rates among other workers as well. Many also state that the approved positions, even if filled, would be inadequate to handle current workloads.

The New York State Department of Health is reporting significant problems throughout the state in recruiting respiratory therapists and pharmacists. HHC reports a shortage of physical therapists, pharmacists, health aides, and technicians.[32] Both New York State and the American Medical Association (AMA) report a decline in enrollment in training programs for most of these professions. The AMA reports that between 1986 and 1987 alone, there were declines of over one third in enrollment for lab technicians, medical technologists, medical assistants, and respiratory therapists.[33]

While the expansion of nurse midwifery could increase the availability of maternity services, the New York State Department of Health reported a high vacancy rate for CNM positions in New York City. There are 400 certified nurse midwives in New York State, although no data are available on how many are practicing in New York City. As only 30 certified nurse midwives graduate annually in New York State, it could be years before these vacancies are filled, and much longer before nurse midwifery capacity can be expanded.

CONCLUSIONS

The factors described above, which are contributing to the need for intensive perinatal services in New York City, are likely to persist or increase in the coming years, with the possible exception of birth rates. The current supply of services is inadequate to meet this need.

The perinatal care system in New York City is at a critical juncture. Needed are both immediate actions to alleviate the current crisis, and long term solutions, some of which may require systemic and policy changes.

Without an increase in the supply of health care workers, expansion of inpatient and outpatient services will be very difficult. Unfortunately, the shortage of health care workers is likely to worsen in the face of rising need, unless significant changes are made in recruitment and retention policies. Creative approaches could include adjustment of salary levels, benefits, career ladders, and training opportunities, as well as the development of day care and flextime options. Recruitment efforts that target minority communities might better succeed if attached to supportive funding for recipients. The development of joint programs with health care unions to offer upgrading and training for union members offers another route for filling mid-level positions.

The malpractice crisis has contributed to diminishing the supply of obstetricians. Obviously, the malpractice crisis requires resolution. Innovative approaches that address quality assurance as well as providing resources for babies with perinatally acquired handicaps might provide a starting point. Virginia has been a pioneer in establishing a no-fault compensation system for birth-related neurologic injury, which also encourages physician acceptance of Medicaid patients. Rosenbaum and Hughes[34] have suggested that states establish subsidized umbrella malpractice plans through joint underwriting associations for obstetricians caring for public sector patients. They also advocate that state insurance commissions closely scrutinize the practice of setting malpractice insurance rates.

Obstetrician shortages have been further exacerbated by cuts in approved residency slots in New York City. A major factor in the reduction of these slots has been concern about the adequacy of gynecologic surgical experience. National consensus would be required to reassess these educational requirements in the light of changing patterns of practice and need. New York City's leading obstetric educators could serve as gadflies to stimulate such a review.

Lastly, perinatal care must concern itself with the current socioeconomic realities and integrate a broad spectrum of services into the delivery system. While perinatal care clearly cannot resolve underlying societal issues such as homelessness, poverty, and drug use, it can incorporate recognition of these factors into the design of clinical services. For example, obstetric and drug treatment providers could forge programmatic links that would not only ensure coordination of all medical components but plan for supportive parenting training and pediatric developmental assessment in cases in which supervised family reunification can be anticipated. San Francisco has initiated a multi-agency collaboration toward this end which includes representation from the Department of Public Health, Child Protective Services, Community Substance Abuse Services, Day Care, and Housing Police (personal communication, Geraldine Oliva, MD, MPH, Director of Family Health, San Francisco Dept of Public Health, December 1988). Another such example could consist of strengthening the link between obstetric and gynecologic care (past a single postpartum visit) in order to intervene medically prior to a subsequent high risk pregnancy, as well as in recognition of the STD epidemic.

New York City is currently demonstrating the effects of an inadequate response to signs of a growing crisis in perinatal services. The cost of allowing the current trends to continue could be reversal of prior decades' gain in maternal-infant morbidity and mortality. Short term cost savings may prove to be extremely costly in the long run.

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The management of respiratory failure in infants and children

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Respiratory failure occurs when an illness or impairment of the respiratory system results in inadequate oxygenation, inadequate ventilation, or both. In these situations, the pulmonary system is unable to meet the metabolic demands of the body. Respiratory failure is a common medical emergency in infants and children and is encountered in almost every type of pediatric practice. The problem may present in a variety of clinical settings. It may present as an exacerbation of chronic underlying lung disease or as an acute process occurring in previously healthy lungs.

The clinical assessment of respiratory failure is difficult and often unreliable. The diagnosis is most easily made by the physician who has a high index of suspicion and is aware of the clinical situations in which respiratory failure is likely to be a complication. This article reviews the management of acute respiratory failure in the pediatric patient excluding the neonatal period.

DEFINITION

There is no uniform clinical definition of acute respiratory failure that includes all of its causes. The primary functions of the respiratory system are the elimination of carbon dioxide from and the delivery of oxygen to the pulmonary capillary bed. In the absence of an intracardiac shunt, acute respiratory failure is present if arterial oxygen tension (PaO_2) is less than 50 torr in a patient breathing room air, or the arterial carbon dioxide tension ($PaCO_2$) is greater than 50 torr.[1,2] These values are arbitrary and are not diagnostic of any particular disease.

It is important to keep in mind that a patient may have a normal PaO_2 and be in profound respiratory failure if he is breathing air containing a high oxygen concentration. Furthermore, patients with $PaCO_2$ values in the 40 torr range may have profound acidemia if they have preexisting bicarbonate loss. Thus, they may have a life threatening problem in the presence of this lesser degree of hypercapnia. Other clues to the existence of acute respiratory failure are the presence of dyspnea, acidemia, or both.

This definition of respiratory failure allows for the division of the condition into two types. Type I is a failure of oxygenation alone and is characterized by a low PaO_2 and a normal or low $PaCO_2$. The major cause of type I respiratory failure is usually a disease process involving the lungs themselves. Type II failure is primarily a failure of ventilation and is manifested by a low PaO_2 and a high $PaCO_2$. It usually results from either a decrease in minute ventilation or an increase in total dead space.[3]

Classifying respiratory failure as either type I or type II offers only limited clinical information and reveals nothing about the specific disease entity involved or its severity. However, the classification does offer insight into the basic pathophysiologic processes involved and may perhaps point toward appropriate therapeutic modalities.

ETIOLOGY

The various conditions that may result in acute respiratory failure in children are best considered according to the location of the defect. A properly working respiratory system requires that three of its divisions be intact: the central nervous system respiratory centers; the chest bellows and its components; and the lungs, including upper and lower airways. Therefore, a significant disturbance affecting any one of these components may lead to respiratory failure. Some of the major diseases and conditions that may lead to respiratory failure are listed in Table I. It is important to realize the diverse nature of these causes.

TABLE I. Causes of Respiratory Failure in Children

- Central nervous system dysfunction
  - Drug overdose (narcotic, sedative)
  - Trauma
  - Mass lesions
  - Infection
  - Central hypoventilation syndromes
- Respiratory bellows
  - Congenital neuromuscular disorders
  - Acquired neuromuscular disorders (infections, postinfections, toxins)
  - Trauma
  - Pleural effusion
  - Extreme obesity
- Lungs and airways
  - Foreign body aspiration
  - Congenital anomalies
  - Infections (supraglottitis, bronchiolitis, pneumonia)
  - Asthma
  - Cystic fibrosis
  - Obstructive sleep apnea
  - Pulmonary edema
  - Trauma

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CLINICAL MANIFESTATIONS

The clinical signs and symptoms of respiratory failure in children are often nonspecific. The manifestations of respiratory failure are not always clinically evident, and some of the symptoms may be due to nonrespiratory causes. The clinical assessment alone of hypoxemia and hypercapnia is not reliable.[4] Clinical judgment is required as to when a blood gas determination should be obtained. Signs and symptoms that may be related to hypoxemia, hypercapnia, and acidosis are summarized in Table II. Mental status changes, ranging from restlessness and agitation to coma, are common to all three. Cyanosis is especially unreliable as a guide to a low PaO_2. Cyanosis may not appear until hypoxemia is severe, especially in the anemic patient. And, probably most important in assessing a patient for cyanosis, there is large interobserver variability.

Hypoxemia interferes with brain metabolism, depresses the myocardium, and causes pulmonary hypertension. Hypercapnia depresses the central nervous system, and acidemia depresses myocardial function. Therefore, on first evaluation, any child who is comatose, obtunded, confused, or appears cyanotic should undergo a blood gas evaluation. In addition, the presence of severe or unexplained dyspnea, restlessness, or unexplained somnolence probably warrants assessment of blood gases.

PATHOPHYSIOLOGY

The basic mechanisms of abnormal gas exchange are hypoventilation, ventilation-perfusion imbalance, intrapulmonary shunt, and diffusion impairment (Table III). All four of these abnormal mechanisms are potential causes of hypoxemia in a child at rest and breathing room air at sea level. An individual with respiratory failure may have abnormal arterial blood gases due to any one or a combination of these mechanisms.[5,6]

Alveolar hypoventilation occurs when ventilation of the lungs does not remove all the carbon dioxide produced by cellular metabolism. Alveolar hypoventilation is the characteristic abnormality in patients with depressed central nervous system function, or those with neuromuscular or skeletal abnormalities that result in inadequate movement of the chest bellows. Hypoventilation may result from either a decrease in tidal volume or respiratory rate, or when there is an increase in total dead space. Causes of hypoventilation include depression of the respiratory center caused by drug overdose, paralysis of the respiratory muscles, muscle fatigue, and central hypoventilation. Both hypoxemia and hypercapnia usually result, and, if the process is acute, acidosis will usually be present.

TABLE II. Clinical Manifestations of Respiratory Failure

Condition	Symptoms	Signs
Hypoxemia	Restlessness	Arrhythmias
	Agitation	Tachycardia
	Confusion	Tachypnea
	Dyspnea	Cyanosis
Hypercapnia	Headache	Diaphoresis
	Dizziness	Tachycardia
	Somnolence	Hypertension
	Coma	
Acidosis	Coma	Arrhythmias

TABLE III. Causes of Hypoxemia

Respiratory causes	Ventilation-perfusion imbalance
	Pulmonary shunt
	Diffusion barrier
	Hypoventilation
Nonrespiratory causes	Low inspired oxygen concentration
	High altitude
	Intracardiac shunt

Ventilation-perfusion imbalance occurs when there is a mismatch of ventilation to blood flow in the lungs affecting oxygen uptake. The matching of ventilation to perfusion becomes increasingly abnormal as the lung disease becomes more severe. This is a characteristic of most abnormalities that affect the lungs in children, such as an acute asthma attack or mucoid obstruction of the airway with resultant atelectasis. The hypoxemia caused by mild mismatching of ventilation and perfusion can usually be corrected by increasing the oxygen concentration of the inspired air. Acidosis may or may not occur, depending on the presence or absence of hypercapnia and the severity of hypoxemia.

Intrapulmonary shunting results from perfusion of large areas of nonventilated or underventilated alveoli. This shunting is a major cause of hypoxemia in children with acute lung injury, including both cardiogenic and noncardiogenic edema. Hypercapnia is usually not present. However, hypercapnia may develop in late stages of ventilation-perfusion imbalance, especially when respiratory muscle fatigue becomes increasingly significant. If hypoxemia is severe, acidosis may occur as cellular metabolism becomes increasingly anaerobic leading to increased lactic acid production.

Diffusion impairment, which is the failure of the pulmonary capillary blood to fully equilibrate with the alveolar gas, is a very uncommon cause of hypoxemia in children. Additionally, because of its tendency to develop slowly, diffusion impairment is rarely the sole cause of acute respiratory failure and generally results in hypoxemia but does not produce hypercapnia.

MANAGEMENT

A clinical diagnosis of respiratory failure can be made by use of findings from a complete history, physical examination, and determination of blood gases and other routine laboratory tests. From these data, an adequate assessment of oxygenation, ventilation, and acid-base balance can be made as well. Table IV lists important questions one should consider in the assessment of a patient with respiratory failure. As soon as possible, treatment should be instituted to correct the underlying disease process and to ensure adequate ventilation and oxygenation.

Table V outlines a sequential approach to the evaluation and management of respiratory failure. Frequently, respiratory failure will progress slowly over a period of hours or even days. This allows adequate time for diagnosis and institution of therapy. However, some patients, when they are first brought to medical attention, have severe respiratory distress with cardiorespiratory arrest imminent, or they may even exhibit respiratory or cardiac arrest. Once resuscitation is achieved, the best method for

TABLE IV. Questions to Consider When Assessing Oxygenation, Ventilation, and Acid-Base Status in Acute Respiratory Failure

- Oxygenation
  - Is the patient adequately oxygenated?
  - Are the lungs working properly to effect oxygenation?
  - How much oxygen should be administered and by what route?
- Ventilation
  - Is the patient's alveolar ventilation adequate for the level of CO_2 production?
  - Is the patient working too hard to maintain the level of alveolar ventilation?
  - Does the patient need ventilatory assistance?
- Acid-base status
  - Does the patient have an acid-base disorder?
  - What is the acid-base disorder?
  - What are the underlying problems causing the acid-base disorder?

determining the need to increase the inspired oxygen concentration is by the direct measurement of the arterial oxygen tension.

Even in the absence of cyanosis, the presence of hypoxemia may be inferred by symptoms such as restlessness, confusion, and coma. When these findings are present, administration of oxygen should be instituted immediately and blood gas tension measurements obtained as soon as possible.

Cyanosis is a poor indicator of hypoxemia because it depends on the hemoglobin concentration. Cyanosis will be evident only when there is about 5 g of reduced hemoglobin per 100 mL of whole blood.[7,8] Therefore, mild cyanosis may be difficult to detect in the presence of anemia. When polycythemia is present, cyanosis may be present even though adequate oxygen content is available to satisfy tissue demands. Nevertheless, the presence of cyanosis requires oxygen administration until blood gas tensions are assessed.[9]

OXYGEN ADMINISTRATION

There are few respiratory diseases associated with hypoxemia in which administration of oxygen is not indicated. As a general rule, oxygen therapy will have little or no effect in a hypoxic child when desaturation is due to right-to-left cardiac or intrapulmonary shunting. Although there are few contraindications to oxygen when it is properly used, there are important precautions to take in its administration. Oxygen should be considered a drug that has proper doses and routes of administration for providing maximum benefit and minimum toxicity.

TABLE V. Evaluation and Management of Respiratory Failure

- Suspect diagnosis
- Confirm diagnosis
- Determine arterial blood gases: classify patient as type I or type II respiratory failure
- Evaluation
  - Arterial blood gas
  - History
  - Physical exam
  - Chest x-ray
  - Routine laboratory data
  - (ECG, electrolytes, blood count, etc)
  - Other procedures
  - (cultures, drug [toxicology] screen, etc)
- Management
  - Oxygenation
  - Ventilation
  - Fluids
  - Chest physiotherapy
  - Treatment of specific etiology
  - Close observation and monitoring

The use of oxygen therapy in infants and children is associated with some adverse effects.[10] In patients with chronic hypercapnia, excessive use of oxygen may cause further respiratory depression by decreasing the frequency and depth of breathing.[8] This is uncommon in infants and children, but it occasionally occurs in patients with cystic fibrosis. Another possible adverse effect of oxygen therapy is alveolar collapse, which may develop during inhalation of air containing high concentrations of oxygen. Nitrogen, an inert gas that is relatively insoluble in blood, maintains a residual volume in the alveoli. If oxygen replaces nitrogen, the gas volume within the alveoli is reduced because oxygen is rapidly absorbed into the blood. This is particularly likely in alveoli in which there is mismatching of ventilation and perfusion.[10]

Side effects of oxygen administration in animals were recognized for many years. Oxygen toxicity is of concern primarily in intubated patients who require the delivery of continuous, high concentrations of supplemental oxygen for long periods. Oxygen toxicity may affect the lungs, central nervous system, retina, hematopoietic system, and endocrine organs.[11] Normobaric oxygen toxicity occurs when oxygen is delivered to the patient at normal atmospheric pressure. This condition is most often manifested by pulmonary abnormalities and is therefore of greatest clinical importance. Lastly, to minimize the drying effects on respiratory epithelium, oxygen must be warmed and humidified.

OXYGEN THERAPY EQUIPMENT

Until recently, oxygen therapy given by nasal cannula was not tolerated well by infants and young children. The prongs had large external diameters that were uncomfortable and irritating. In some cases, these large prongs were dangerous when modified for young children. Cannulae are now specifically designed for infants. They are convenient, they are relatively noninvasive, and they cause only slight restriction of the child's movements.[12] Eating, drinking, and communication with family and staff are not inhibited. Children rarely experience claustrophobia and feelings of helplessness and suffocation with the use of nasal cannulae, as often occurs when other forms of oxygen therapy (such as a face mask, hood, or tent) are used.

The concentration of oxygen in the inspired air varies greatly with changes in flow, minute ventilation, and cannula position. Nasal cannulae frequently are not the ideal device to use for active or restless children, because cannulae are easily dislodged from the nares. In addition, they should not be used in patients with nasal obstruction. The lowest flow rate that keeps the PaO_2 in a safe range is the one to use. Oxygen flow rates greater than 6 L/min should not be used, because such high gas pressures are associated with gastric retention and regurgitation, and with possible penetration of frontal sinuses by gas, leading to frontal headaches.

Oxygen hoods are clear plexiglass domes or boxes of variable sizes placed over the head and neck of the patient. Designed for use with infants, small sized hoods are capable of achieving oxygen concentrations approximating 95% to 100%. As with the treatment of newborns, oxygen hoods for use in older infants or children restrict access to the head for activities including facial care and feeding. Older children feel confined and are too active to stay in these hoods. While using a hood, it is important to ensure against accumulation of Co_2 by utilizing oxygen flow rates of 8 to 10 L/min or greater and by continuously monitoring the inspired oxygen concentration.

When a more precise oxygen concentration is indicated, a face mask may be used. A wide variety of masks are available for infants and children. Regardless of type, however, all masks should be soft and pliable and made of transparent material. A flow of at least 3 to 6 L/min, depending on the type of mask, is necessary to prevent Co_2 accumulation. All masks impose several difficulties including impaired communication with the patient, discomfort, a feeling of claustrophobia, and interference with feeding and expectoration of secretions.

There are several types of oxygen masks that meet specific needs. The simple oxygen mask can provide oxygen concentrations from about 35% to 55%, depending on the patient's inspiratory flow rate and tidal volume in relation to the oxygen flow rate into the mask. For example, if the patient's flow rate exceeds the oxygen flow into the mask, the oxygen concentration is diluted by room air. The oxygen concentration also varies if there are any leaks around the ports or perimeter of the mask. The partial rebreathing mask contains a reservoir bag and may achieve an inspired oxygen concentration between 65% and 95%. The reservoir bag should never be allowed to completely deflate during inspiration. If this occurs, the oxygen flow rate is insufficient to handle patient needs or there is a significant leak in the system. A non-rebreathing mask utilizes one-way valves and a reservoir bag and is commonly used to deliver an oxygen concentration approaching 100%.

Venturi mixing masks are designed to deliver a specific oxygen concentration to the patient. They are especially useful in the treatment of chronic lung disease, and for treating those diseases that require a fixed inspired oxygen concentration between 24% and 50%.[12,13]

The most popular method of providing infants and children with an oxygen concentration up to 50% is by use of a tent. However, the oxygen concentration in a tent is directly proportional to the oxygen flow rate and the number of leaks in the system. Although the popularity of the oxygen tent has diminished somewhat recently, it is still very useful for the child who will not keep a cannula or mask in place. However, the tent, commonly referred to as the "croup tent," may be unsuitable for active or agitated patients. Entry into the tent quickly reduces the oxygen concentration of the inspired air; thus, children requiring frequent nursing care should receive oxygen or humidity by another device.

TRACHEAL INTUBATION

In general, patients who have type I or borderline type II respiratory failure usually do not need mechanical ventilatory assistance. They require only oxygen administration, adjunctive therapy, or a combination of the two, to improve airway function. In contrast, the presence of type II respiratory failure with a high level of hypercapnia, especially when there is a rapidly rising $Paco_2$, is a clear indication for instituting mechanical ventilation. In addition, mechanical support may be necessary even in the absence of evidence of type II respiratory failure. This would be the case when it is clinically evident on overall evaluation that the child is likely to hypoventilate and develop serious acidosis and hypoxemia.

Assessment of the patient's clinical parameters, including the degree of cerebral, cardiac, and respiratory dysfunction, together with the blood gas evaluation will determine the need for tracheal intubation. When possible, the patient should be given 100% oxygen prior to intubation. Sterile polyvinyl chloride tubes are used, with their approximate size based on the patient's age (Table VI).

The preferred route of intubation will frequently vary with the personal preference of the physician. Nasal intubation, which may be used in older infants and children, should only be considered once an oral tube is in place. This route offers the advantages of improved oral and pharyngeal hygiene; it also provides a more stable fixation of the tube, which reduces the chances of tracheal erosion and accidental extubation. Nasal intubation is the preferred route when an adequate artificial airway is needed for one to three weeks. The complications associated with the use of endotracheal tubes in pediatric patients are well known (Table VII). Great care and close monitoring of the artificial airway will minimize the risks of several of

TABLE VI. Orotracheal Tube Specifications*

Age Group	Internal Diameter (mm)
Newborn (<1.0 kg)	2.5
Newborn (>1.0 kg)	3.0
1–6 mos	3.5
6–12 mos	4.0
12–18 mos	4.5
18–36 mos	5.0
3–4 yrs	5.5
5–6 yrs	6.0
6–7 yrs	6.5
8–9 yrs	7.0
10–11 yrs	7.5
12–14 yrs	8.0

* Modified from Pagtakhan and Chernick.[3]

TABLE VII. Complications of Intubation

- Loss of airway and hypoxia
- Aspiration
- Tube obstruction
- Trauma to orificial structures
- Nasopharyngeal hemorrhage
- Sinusitis
- Laryngeal injury
- Tracheal ulceration
- Tracheitis
- Subglottic edema, stenosis
- Introduction of nosocomial infections

these complications. Cuffed tubes are rarely required in children less than eight years of age.

MECHANICAL VENTILATORY SUPPORT

A variety of conditions require respiratory intervention and mechanical support in the infant and older child. These vary from apnea due to neurologic depression to conditions associated with acute or impending respiratory failure. The adequacy of ventilator support can best be assessed by serial blood gas determinations.

The ultimate goal of mechanical ventilation is to provide effective gas exchange with minimum circulatory impairment and minimum trauma to the lung. This may be accomplished by a ventilator that moves gas into the lungs, holds it there for a predetermined amount of time, and then releases it to a preset baseline. Most ventilators used in the care of children with respiratory failure are either volume-cycled or time-cycled.[14] Volume-cycled ventilators end their inspired cycle when a predetermined volume of gas has been delivered. The volume of gas delivered to the patient is independent of the pressure that the machine develops. An increase in ventilator pressure may be the result of several factors, including decrease in lung compliance, kinked ventilator tubing, malpositioned endotracheal tube, water condensation in the ventilator tubing, and increased airway resistance due to accumulated secretions or bronchospasm.

Time-cycled ventilators change from the inspiratory phase of the breathing cycle to the expiratory phase only after a preset time has elapsed. If the gas flow is constant, tidal volume is the product of flow and inspiratory time. However, some volume is lost due to expansion of ventilator tubing as well as from leaks within the system. The optimal tidal volume and minute ventilation in infants and small children can be determined by observing chest expansion, auscultation for air entry, measuring exhaled volumes, and by verifying adequacy of gas exchange with analysis of blood gases.[15] Volumes of air exchanged are fairly constant, but changes in airway resistance and lung compliance can cause a portion of each breath to be lost within the ventilator circuit.

To avoid delivering excessive pressures to the patient, the ventilator chosen should have an upper pressure limit above which it will vent any excessive pressure to the atmosphere through a pressure-relief device. Both volume-cycled and pressure-cycled machines have a pressure limiting feature as a safety device.

There are many ventilators available that are capable of providing respiratory support for the older infant and child. In children with body weights greater than 15 kg, a volume-cycled machine is most commonly used. There are several parameters on the ventilator that must be determined. Each control setting should be based on the disease being treated, the patient's metabolic demands, and his or her size.[14] One should always ventilate in a manner that is consistent with the patient's usual pattern of blood gas values. For example, children with chronic carbon dioxide retention are usually ventilated so that the pH remains normal despite the fact that $PaCO_2$ is elevated.

Most patients will tolerate a tidal volume that is slightly more than that observed while breathing spontaneously. Thus, a tidal volume in the range of 12 to 15 mL/kg may be chosen. If the patient is assisting the ventilator, it may be necessary to alter the delivered volume so that the minute ventilation does not become excessive. The assist mode on the ventilator may be used if the patient's work of breathing is not excessive. Otherwise, the patient should be sedated and possibly paralyzed with a muscle relaxant and ventilated at a controlled rate consistent with metabolic requirements.[16]

The setup of a mechanical ventilator for a patient requires several considerations other than minute ventilation, that is, tidal volume and rate. These additional considerations include pressure limit, oxygen concentration, inspiratory gas flow, inspiratory to expiratory ratio, and positive end-expiratory pressure. Depending on the patient's disease, condition, and age, several ventilation strategies may be considered. While it is beyond the scope of this article to discuss all strategies, in-depth discussions are available elsewhere.[12-14]

Intermittent mandatory ventilation (IMV)[17] is the most common mode of ventilation in children. The patient is permitted to breathe spontaneously between ventilator-administered breaths. Total ventilation is achieved by a combination of ventilator positive-pressure breaths and spontaneous negative-pressure breaths. This has the effect of decreasing mean intrathoracic pressure and therefore enhancing venous return and cardiac output. A potential problem with such a mode of ventilation, however, is that the machine does not always synchronize with the patient's breathing pattern. Newer ventilators, such as the Siemens 900C, have a synchronous IMV (SIMV) capability. Thus, the ventilator will not produce a positive pressure during a patient's spontaneous breath but continues to supply a fresh gas flow.

As the patient's condition stabilizes and improves, weaning from mechanical ventilation is accomplished in a step-wise manner. The inspired oxygen level generally is maintained at 50% or less before positive end expiratory pressure (PEEP) is lowered. PEEP is usually decreased in increments of 2 cm H_2O as long as oxygenation is maintained, until a level of 4 cm H_2O is reached. The ventilator rate is gradually lowered according to arterial pH and $PaCO_2$, allowing the patient to assume a greater proportion of the work of breathing. Maintenance of optimal nutrition is an integral part of the weaning process. Once the patient is able to maintain an acceptable blood gas level of 40% or less inspired oxygen, PEEP of 4 cm H_2O, and IMV of 2–4, removal of the endotracheal tube is considered.

High-Frequency Ventilation. High-frequency ventilation provides a radically new concept in ventilation. The technique of using a ventilator that incorporates a pulsing jet effect as well as high inspiratory cycle rates allows for small tidal volumes and low peak inspired pressures. This technique has been used in selected clinical problems involving infants and older children who are refractory to the established modes of ventilation. To date, controlled studies using this technique in pediatrics are limited, and further clinical trials are necessary before high frequency ventilation is ready for general clinical use.[18]

ADDITIONAL THERAPY

Management of respiratory failure in children often requires additional treatments, depending on the etiologic or pathophysiologic features of the illness. Adequate fluid balance plays an important role in the management of acute respiratory failure. With a few exceptions, patients in respiratory failure benefit from being kept on the dry side (slightly underhydrated). Reduction of hydrostatic pressure in the capillary bed will diminish leakage of water into the lungs. Increase of extravascular lung water can cause decreased pulmonary compliance, which may result in difficulty in achieving adequate ventilation without high pressures. Keeping the patient optimally dry or underhydrated will lower pulmonary vascular pressures and will diminish the amount of extravascular lung water in both the interstitium and the alveoli. "Optimally dry" implies avoidance of the complications of dehydration such as hypotension and renal insufficiency.

Pharmacologic agents play a major supporting role in the management of respiratory failure. Thus, these drugs are effectively used as bronchodilators in patients with status asthmaticus or other respiratory tract disorders accompanied by severe bronchospasm.

Chest physiotherapy and suction techniques are frequently indicated in cases of respiratory failure in which an excess of pulmonary secretions cannot be removed by the normal clearance mechanisms of ciliary activity and cough. These techniques are also indicated in the treatment of atelectasis.

MONITORING OF PATIENT STATUS

Close observation of the acutely ill child requires regular inspection and continuous monitoring of vital laboratory parameters, such as analysis of blood gases.[19] Modern electronic technology provides accurate and noninvasive methods of continuously monitoring oxygen and carbon dioxide. Transcutaneous monitoring not only correlates well with determination of PaO_2 and $PaCO_2$, but it also reflects other physiological parameters such as general circulatory conditions and peripheral blood perfusion.[20] It has been used extensively in neonates as a reliable trend monitor. In older children it appears to be less effective.

COMPLICATIONS OF TREATMENT

There are many potential complications associated with treatment of acute respiratory failure.[21] These complications may involve any organ or system of the body. Significant advances in the intensive care of these critically ill patients have produced longer survival. But, unfortunately, this progress has also resulted in a further increase in the number of complications. Common problems that are encountered are discussed in the following sections.

Complications of Airway Pressure Therapy. The clinical goal of airway pressure therapy is to provide optimal pulmonary ventilation while minimizing physiological stress. It is imperative to understand not only how and when to apply airway pressure therapy, but also to know how and when the therapy may be detrimental or contraindicated.

Pulmonary barotrauma basically consists of lung rupture secondary to excessive airway pressure. The consequences of this trauma include interstitial and subcutaneous emphysema, pneumomediastinum, pneumothorax, and pneumoperitoneum.[22] The incidence of barotrauma varies from 1% to 5% among patients receiving positive pressure airway therapy.[23] Positive end-expiratory therapy of 8 to 10 cm of water or greater is associated with a greater risk of barotrauma (5% to 7%).[23] The diagnosis of barotrauma is based on clinical suspicion, findings on radiologic evaluation, and the finding of physiologic alterations in pulmonary function.

Venous Return Compromise. Use of airway pressure therapy in any form potentially decreases venous return. Hemodynamic monitoring, intravenous fluid therapy, and the administration of drugs that alter cardiovascular function significantly affect the use of airway pressure therapy.

Pulmonary Atelectasis and Infection. Segmental and lobar atelectasis are common complications of airway pressure therapy. Complete reexpansion may be possible with the use of bronchodilator aerosols together with chest physiotherapy and suction. If these modalities are unsuccessful, fiberoptic bronchoscopy may be used. This technique has been shown to be of great value for treating persistent atelectasis in infants and children.[24] Pulmonary infection is a common complication of intubation. Nosocomial infections caused by microbial strains resistant to antibiotics are a constant threat to these patients. The occurrence of these infections may be minimized by use of optimal pulmonary hygiene and the meticulous care of the artificial airway and respiratory care equipment.

Extra-Pulmonary Complications. Organs other than those of the pulmonary system may be at risk for impairment of function secondary to airway pressure therapy. Impairment of renal function has been shown to be directly related to airway pressure therapy. In general, positive airway pressure decreases urinary output and causes retention of sodium and water. These effects may be partly attributable to increased levels of antidiuretic hormone, to changes in the distribution of intrarenal blood flow, or to the combined effects of both factors.[25]

Gastrointestinal bleeding due to stress ulcers, gastric distention, and pneumoperitoneum are commonly observed in patients being treated with positive airway pressure.[26] Most bleeding appears to be due to acute ulcerations of gastric and bowel mucosa. Antacid regimens that maintain the pH of gastric fluid over 5.0, as well as early and frequent enteral feedings, have been successful in decreasing the incidence of gastrointestinal bleeding.

There are other factors not related to airway pressure therapy that significantly contribute to the morbidity and mortality of acute respiratory failure. The complications are too numerous for this discussion. One or multiple organs and systems may be involved. One such factor is the increased susceptibility to malnutrition. In addition to basic nutritional requirements, these patients may have increased metabolic rates related to accelerated protein turnover in support of host defense or tissue repair.

If significant lung injury occurs in a malnourished patient, or if the illness becomes protracted, serious loss of lean body mass develops. The end result of protein-caloric malnutrition in a setting of critical illness is increased morbidity and death, commonly related to infection. Severe muscle weakness can also occur, making weaning from ventilatory support very difficult. Repletion of malnutrition or maintenance of normal nutritional status can be accomplished with either parenteral hyperalimentation or enteral alimentation.[27]

Many complications can ensue from total parenteral nutrition, including the metabolic complication of excessive carbohydrate loads.[28] It appears that patients with acute respiratory failure are at risk of developing hypercapnic failure when given large carbohydrate loads. Therefore, caloric replacement should not be greater than the resting requirement and, if possible, some nonprotein calories should be given as lipid.

Other complications of acute respiratory failure include hepatic, neurologic, and hematologic dysfunctions. In addition, major psychological stresses frequently lead to abnormal behavior patterns.

CONCLUSIONS

Acute respiratory failure in infants and children is a life threatening condition with a myriad of causes. It is characterized by a failure of oxygenation, ventilation, or both. Hypoxemia is common to all causes of respiratory failure, whereas $PaCO_2$ levels may be normal, decreased, or elevated. These abnormalities result from several pathophysiologic processes, including ventilation-perfusion mismatch, intrapulmonary shunt, hypoventilation, and diffusion impairment.

The clinical manifestations of acute respiratory failure are usually nonspecific. For this reason, a high index of suspicion and early analysis of blood gases are essential to successful management. Many complications can occur during the management of respiratory failure and may involve multiple organs. Thus, early diagnosis, aggressive treatment, and prevention of complications should reduce morbidity and mortality of acute respiratory failure in the young patient.

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Animal experimentation for medical research*

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Throughout history, animals have played an important role in man's understanding of himself and his environment. Over the years, a wide variety of animals have made invaluable contributions to the understanding, treatment, and cure of human diseases. These animals provide essential tools for studying a broad spectrum of infectious, neoplastic, hereditary, metabolic, and other human disorders. Much of our knowledge of our own bodies and many medical discoveries stem from experiments with laboratory animals.

Humane considerations in the use of experimental animals have been discussed, often with considerable vigor, for more than a century. Medical and scientific research workers whose studies involve experiments on animals have been criticized because they are said to needlessly repeat and duplicate experiments, subject animals to unnecessary pain and distress, use too many animals, and obtain nonsignificant information.[1] Scientific groups counter by asserting that experimental procedures are designed to minimize animal pain and distress, that while some experiments are productive this fact cannot be determined in advance, and that confirmation of experimental results is an essential part of the scientific process.

The place and importance of the experimental animal in medicine today is hotly disputed by antivivisectionists and the scientific community. While abolition of animal experimentation is improbable, unduly restrictive regulation, severely inhibiting medical research, is a growing threat.

HISTORICAL BACKGROUND

In Britain, concerns about animal experimentation began early in the 19th century with the establishment in 1824 of the Royal Society for the Prevention of Cruelty to Animals. Its main focus was to stop painful animal research. In 1870, partially in response to this concern and because vivisection had become a raging controversy, the British Association for the Advancement of Science developed guidelines for conducting physiological experiments, including steps to minimize animal suffering and to discourage animal experiments that were not scientifically justified. In 1875, Queen Victoria urged the establishment of a Royal Commission to investigate the use of live animals for experiments, to study the cruelty that might be involved, and to suggest possible ways to prevent it. The report of the commission resulted in the subsequent passage in 1876 of the now famous Cruelty to Animals Act. The main features of the act include the licensing of experimenters, the establishment of an inspectorate, and the "pain rule." Pain to the animal had to be minimized, experiments had to have the goal of alleviating human suffering, and experiments on domesticated animals such as cats, dogs, and horses required special certification.

The immediate impact of the Cruelty to Animals Act was the reduction of animal experimentation. In response, in 1882, the British medical and scientific communities established the Association for the Advancement of Medicine by Research. Its goal was to promote research and to seek a just implementation of the 1876 Cruelty to Animals Act. The administrative approval of applications for licenses to experiment on living animals was transferred from the home secretary to this association, which issued licenses to large numbers of qualified scientists. Experimental science and medicine expanded rapidly. The controversy in Britain did not abate and many antivivisectionists and humane societies are to this day trying to abolish most, if not all, animal experimentation. The 1876 Cruelty to Animals Act was in effect until 1986. After several years of hearings in the House of Lords, the Animals (Scientific Procedures) Act was passed, with full implementation to take place over three to five years.[2]

In the United States, the American Society for the Prevention of Cruelty to Animals (ASPCA) was founded in 1866. In the following year and again in 1880, antivivisection bills were presented to the New York State legislature. Both failed. In 1883 the first antivivisection society was founded in Philadelphia. It was not until the end of the 19th century, when scientific disciplines found animal experiments to be necessary for the education of physicians, that protests against the use of animals for experimentation became organized. Activities by American animal protection groups have increased since that time and culminated in proposed federal legislation which, if passed, would not only restrict the use of animals for research but would also restrict the kinds of research that

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could be conducted. Present laws relating to research animals include the Federal Laboratory Animal Welfare Act (1966), later changed to the Animal Welfare Act (1970), and its amendments (1976).

The Animal Welfare Act (1966) covered the transportation, procurement, and care of cats and dogs, providing minimal standards for housing and adequate veterinary care. Amendments in 1970 and 1976 broadened coverage to include other warm blooded animals, and strengthened the concept of adequate veterinary care to include appropriate use of anesthetics and tranquilizers. Still exempted was interference with actual experimentation with animals at the research facility. In 1985, National Institutes of Health policy for the care and use of laboratory animals was revised and the policy was enacted into law by Congress as the Health Research Extension Act, which became the new Public Health Service (PHS) policy.

The main feature of the new PHS policy was the requirement that all institutions receiving federal funding establish an institutional animal care and use committee. The committee, which must include a veterinarian and a nonscientist, is charged with reviewing and approving all protocols using animals prior to submission to a federal agency. Shortly thereafter, the Animal Welfare Act was again amended, putting into law a requirement that every research facility covered by the act establish an institutional animal committee. That committee must include a veterinarian and an unaffiliated member representative of community interests concerning proper care and treatment of animals. Its major function is to assess animal care, treatment, and practices in experimental research with animals. These regulations are applicable to the majority of United States animal researchers.

BENEFITS TO MANKIND

In 1988, the Council on Scientific Affairs of the American Medical Association prepared a glossary describing the indispensability of animals in research.[3] The glossary cites review articles which reference more than 800 original scientific manuscripts that delineate medical advances that were dependent on the use of experimental animals.

The discovery and identification of hormones and vitamins and the testing of most antibiotics and chemotherapeutic agents would have been impossible without animal experiments.

Dogs were used in the classical experiments of Banting and Best that identified insulin as an important hormone in treatment of the ocular and vascular complications of diabetes. Procedures for transplantation of the pancreas and other organs were developed using dogs and other animal models. The world's first hepatitis-B vaccine depended on research with chimpanzees and other primates, since the virus cannot be transmitted to other laboratory animals.

The rat has been used extensively for the study of infectious diseases caused by bacteria, fungi, mycoplasma, rickettsia, viruses, and parasites. Dogs have been used to study rickets and calcium and vitamin D deficiency. Dogs have also been used to develop effective therapies for common human ophthalmological diseases such as glaucoma, cataracts, and uveitis, which also occur in dogs. The pump oxygenator used during heart and lung operations was developed using dogs. Pasteur's original work on rabies vaccines was done through study of passage in rabbits. Rabbits were also used to prove that tuberculosis was transmissible by both inoculation and ingestion.

The rat has been used in important studies of reproductive endocrinology, including the physiology and control of the menstrual cycle. Primate research has delineated hormonal and other biological mechanisms responsible for ovulation, steroid feedback, gonadotropin secretion, adrenarche, puberty and adolescence, sperm maturation and transport, embryonic and fetal development, fetal exemption from the maternal immune response, and the onset of labor. Basic research studies carried out in the primate have led to the development of fertility control methods.

Alternatives to in vitro fertilization have now been developed from primate research studies. These include low tubal transfer of eggs and surrogate embryo transfer.

The identification of rhesus factor (Rh) was an early breakthrough in the understanding of the immunology of pregnancy and resulted from tests on primates. If thalidomide had been tested properly in pregnant monkeys in the 1950s, its potential for causing limb and other deformities in the fetus would have been recognized, and much human suffering would have been avoided.

The dog was an important animal model for the development of the use of hypothermia in prolonged neurosurgery, and for many cardiovascular advances. A variety of autoimmune diseases such as thyroiditis, arthritis, myasthenia gravis, and renal disease have been elucidated in the rat. Basic information on genetic mechanisms has been tested in mice models and has led to understanding of mRNA transcription, polypeptide chain production, glycosylation, immunoglobulin molecule assembly, cell secretion, and immunoglobulin transport and action at target sites.

The rat has been the major model used in toxicology screening for carcinogenic compounds. Potential therapeutic techniques for a variety of cancers have been studied in the cat. The mouse model of human lymphoma holds great promise for future research. One of the first links between diet and atherosclerosis was established in rabbit studies. These examples, and many others that could be cited, clearly demonstrate that "animals have proven invaluable in the pursuit of knowledge in the life sciences, and the knowledge gained often benefits both animals and humans."[3]

THE LAY PRESS

On Friday, November 13, 1988, an animal rights advocate was arrested in Norwalk, Connecticut, on charges of planting a bomb outside a company that uses dogs in medical research. Hours after her arrest, members of a New York City Police Department bomb squad entered her apartment and found three more pipe bombs, a shotgun, and animal rights literature.[4] Although many animal rights advocates assail the use of force,[5] break-ins at research laboratories, with the release of animals and the destruction of research files and other property, are frequent.[6,7]

In an op-ed essay in *The New York Times*, Zak[8] argues against the use of animals in research, saying that

> many experiments are frivolous and waste taxpayers' money. Many could be conducted with microorganisms, computer simulations, or other surrogate subjects. But the paramount fact is that many experiments inflict severe pain on animals and are morally objectionable.[8]

Zak presented his views again in a letter in *The Wall Street Journal* a few months later.[9] Responding to Zak's strong opposition to animal experiments, Forman[10] categorizes such opposition as

> skillful but highly misleading propaganda . . . .The nation's biomedical research laboratories are not the sadistic torture chamber conjured up by Mr. Zak and other "antivivisection" activists. The biomedical research community is deeply concerned about animal welfare and aware of the moral responsibilities inherent in conducting animal research. Animals are used in research only when necessary because there is no meaningful substitute.

Another writer[11] states that: "Everyone agrees that animals should not be exposed to unnecessary pain. But neither should scientists be hamstrung by the requirement to use anesthesia in every animal experiment that might cause pain."

When a baboon heart was successfully transplanted into a human infant at Loma Linda University in California in 1984, it heralded a new era in the long history of animal contributions to medical science and the cure of human illness. Yet great debate ensued. Some people argued that baboons have rights and should not be treated as a resource for exploitation by humanity. Others countered by saying that on an ethical value scale, we will always place human beings ahead of subhumans, especially in a situation in which people can genuinely be served by animals.[12]

The most rational and prevailing view, in spite of vocal and well-financed contrary opinions by animal rights groups, is that "most major advances in medicine would not have been possible without research and testing on animals . . . many key discoveries in the past twenty-five years in understanding the cause of cancer, arthritis, heart, lung and kidney disease came from animal research."[13]

SCIENTIFIC LITERATURE

Recently, three of the most prestigious American medical journals have published major articles dealing with the use of animals in medical research. The *New England Journal of Medicine* published a special report on the principles, policies, and practices of animal research at Stanford University.[14] The report considers why animals are used in biomedical research; reviews past achievements and predicts future advances in understanding, preventing, and treating diseases as a result of animal research; and focuses on institutional protection of animal research subjects, including practices at Stanford University.

In the *Archives of Internal Medicine*, Smith et al[15] review the historic role of the American Medical Association and the American physician in the use of animals in medical research from 1896 to the present time. The American medical community has always promoted the humane and appropriate use of research animals and attempted to explain to the public and legislators the importance of animal research to medical progress. These authors point out that current initiatives by physicians to preserve biomedical research are a reaffirmation of their traditional role. An accompanying editorial deplores the increasing vigor of those people who are more concerned with the treatment of animals than with advancing the cause of human health.[16]

A commentary on animals in research in the *Journal of the American Medical Association* appeals to American physicians and scientists to assume a primary role in defending the use of animals in biomedical research.[17] The authors conclude that without support and defense of animal research, "medical advances will be compromised. Opponents of animal research may succeed unless an aggressive, organized effort is mounted by the biomedical community. The integrity of research in medicine must be presented . . . ." The commentary elicited considerable correspondence, both supportive and critical.[18]

The use of animals for medical education is a different but related issue[19] and will not be discussed in this report.

DO ANIMALS HAVE RIGHTS?

In the controversy about the treatment of animals in biomedical research, the claim is frequently made that animals have "rights." An alternate view is that the word "rights" does not apply to animals. The use of the term "rights" in ethics must be sharply distinguished from its legal meaning. Legal rights can be claimed by individuals to be their due, owed to them by virtue of the law. The implication of a legal right is that it can be enforced—that society has an obligation to fulfill the requirements inherent in the legal right. If a law were passed that required every experimental animal to have its water changed every day, the animal would have a (legal) right to fresh water daily. Inspectors could enforce that right.

Moral rights are distinctly different. Most ethicists are of the opinion that whenever a right exists for one party, a corollary consensual duty or obligation exists for another party.[20] A right automatically entails an obligation. Moral obligations have force because they are thought to be the correct thing to do. It is clear that it is easier to pronounce rights than to ensure obligations, but they remain morally linked.

The term "rights," therefore, implies an obligation and thus sets up the possibility of dispute. The term is also extended to matters of lesser moral weight. Society has obligations to animals, trees, and the environment in general. Does that mean that if animals have "rights," then trees also have "rights"?

The question of whether animals have moral rights has been seriously discussed by philosophers such as Hume, Bentham, and Kant, and modern political philosophers such as Nozick.[21] Whether one ascribes rights to nonpersons depends largely on one's theory of rights and their foundation.[22] The traditional way of distinguishing the moral status of animals from that of humans, from Aristotle to Kant to the present day, is on the basis of man's rationality. However, basing rights on rationality or intellectual capability would deprive of their rights infants or mentally incompetent individuals who have less intellectual capacity than some animals.[23]

Some writers attempt to extend the concept of rights to animals based on the ability of animals to experience pain and suffering.[24] Regan[25] argues that animals have rights because they have beliefs, desires, memory, and a sense of identity. The scientific community rejects these arguments. Even if animals have intrinsic moral worth, their moral worth is not equivalent to that of humans. Although we respect the principle that animal lives have value, we believe that "human lives are generally worthier than animal lives, and the right to life of a human being generally supersedes the right to life of an animal."[23]

RELIGIOUS VIEWS ON ANIMAL EXPERIMENTATION

In Roman Catholic teaching, all life is sacred, both human and animal. Nevertheless, animal experimentation for the purpose of the betterment of mankind is permissible, if done in a humane manner. Thus, when performed to find cures for human diseases, animal experiments may be conducted, but must be done with concern for the animal's comfort. However, animal experiments to develop cosmetics and the like are not acceptable.

Jewish law forbids cruelty to animals and also requires that we have compassion for them, and treat them humanely.[26] Thus, hunting for sport is prohibited in Judaism. At the same time, Judaism teaches that everything created in this world was created to serve mankind. Thus, animals may be used as beasts of burden and for food. Animal experiments designed to yield information that might lead to cure of disease are sanctioned by Jewish law as legitimate use of animals for the benefit of mankind. The animals' pain or discomfort must be eliminated or minimized by analgesia, anesthesia, or other means. Otherwise, the pain does not serve a legitimate human need and its infliction is prohibited. Animal experimentation is permissible only if it benefits mankind, not if it merely satisfies intellectual curiosity.

ETHICAL CONDUCT IN THE USE OF ANIMALS FOR MEDICAL RESEARCH

Statutory law in many countries, including France, Britain, the United States, Germany, and Scandinavian countries, now deals explicitly with various aspects of the use of animals in scientific research. Legislative bodies have given authority to executive departments of government to insure that the manner of treatment of animals does not depend upon the whim of individual investigators.[27]

In Britain, animals subjected to experiments likely to cause them pain were protected by the 1876 Cruelty to Animals Act and are now protected even more under the 1986 Animals (Scientific Procedures) Act.

These laws are an effective instrument for exercising tight and detailed control over animal experiments.[28] Since the use of living animals for the acquisition of manual skills was prohibited by the 1876 Act, British surgeons seeking proficiency in microsurgery had to train abroad or practice on decerebrate animals. Removal of this restriction was one of several modifications which adapted the act* to the needs of present day research.[29]

At a 1983 Geneva conference of the World Health Organization/UNESCO Council for International Organizations of Medical Sciences, international guiding principles for biomedical research involving animals were proposed.[30] The last of the 16 principles states that the overriding consideration is that laboratory animals should be treated with the consideration and care for their comfort and well being that is the duty of man to all sentient beings.

In the United States, the NIH asserts that there are valid scientific as well as humane reasons for giving experimental animals good care and compassionate use. It is the responsibility of those who use animals to care for them. NIH policy requires that recipients of its awards assure NIH that they are carrying out this responsibility. NIH uses the National Academy of Sciences National Research Council "Guide for the Care and Use of Laboratory Animals" to define acceptable animal care standards and a set of 12 "Principles for the Use of Animals" to define humane animal use.[31]

The National Research Council (NRC) recently issued a report on the use of laboratory animals in research and concluded that "animal experiments are still critically important to further improvements in medicine and biomedical science."[32] The NRC emphasizes that scientists are ethically obliged to ensure the well being of animals used in research and to minimize their pain and suffering.

The AMA has supported the proper and humane use of animals in research for more than 100 years and opposes legislative and regulatory initiatives that would inappropriately restrict biomedical research.[16] The most recent position statement of the AMA is the following:

> The AMA maintains that the loss of biomedical animal research would hamper severely the remarkable growth of knowledge this nation has experienced in recent decades. Medical breakthroughs in diagnosis, treatment and prevention of heart disease, cancer, diabetes, measles and many other diseases can be traced directly to the use of animals in research.
>
> Procedures once beyond imagination are commonplace today: open-heart surgery, blood transfusions, organ transplantation, laser neurosurgery. All were made possible by animal investigations. Crucial to control of the AIDS epidemic will be animals used in research.
>
> While cases of abuse have been documented, the evidence demonstrates that the overwhelming use of animals in investigations complies with federal, state, local, and institutional guidelines, and is conducted with strict compliance to ethical guidelines.

The Ad Hoc Committee on Animal Research of the New York Academy of Sciences recently published interdisciplinary principles and guidelines for the use of animals in research, testing, and education.[33] These principles are intended for use in development of experimental design and in establishment of procedures to assure the most humane treatment of animals possible.

The American Diabetes Association (ADA) issued a policy statement on the responsible use of animals in research.[34] The ADA believes that the responsible use of animals is essential to biomedical research and education in the treatment and ultimate cure or prevention of diabetes mellitus. The Society for Neuroscience issued guide-

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*See also the Animal Welfare Act, Title 7, US Code, chapter 54, ¶ 2131–2157, Public Law 89-544 (1966), and amendments (Public Law 91-579 (1970), Public Law 94-279 (1976), and Public Law 99-198 (1985)); the Public Health Service Act, Title 42, US Code, chapter 201 *et seq*, Public Law 78-184 (1944), amended by the Health Research Extension Act of 1985, Public Law 99-158 (1985); NIH Reauthorization, chapter 289(d), § 495(a).

lines for the use of animals in neuroscience research in its 1988 Membership Directory. The Foundation of Biomedical Research has issued a handbook for researchers using animal models,[35] as has the Canadian Council on Animal Care.[36,37] The Association of American Medical Colleges has also issued recommendations for the governance and management of institutional animal resources.[38]

The regulation of experimentation on animals on a state level is reviewed by Favre,[39] who proposes new legislation to reduce the pain and suffering of animals to a minimum while allowing maximum flexibility for the researcher. The laws of the State of New York underscore the idea that animal experimentation is a necessary part of medical progress. The relevant laws are found in sections 353 of the Agriculture and Marketing Laws and 55-1.2 through 55-1.6 of the Administrative Rules and Regulations of the New York State Department of Health. Animals are to be treated as humanely as possible consistent with the goals of the needed experimentation. However,

> Nothing herein contained shall be construed to prohibit or interfere with any properly conducted scientific tests, experiments or investigations, involving the use of living animals, performed or conducted in laboratories or institutions, which are approved for these purposes by the state commissioner of health. The state commissioner of health shall prescribe the rules under which such approval shall be granted, including therein standards regarding the care and treatment of any such animals.

While humanitarian concerns have traditionally played a significant role in animal research in the United States, it is only in the last decade that they have been embodied in detailed written principles and that these principles have become a compulsory part of experimental design and implementation. Before the 1980s, the choice of experimental procedures and the steps taken to avoid or minimize animal suffering were left to the individual investigator, perhaps overseen in an informal and haphazard fashion by the institutional veterinarian or other animal care personnel. Now, all studies involving vertebrates must receive prior approval from an institutional committee composed of scientists, veterinarians, and one or two others from outside the institution.[15,40]

The committee evaluates each proposed study and decides whether it satisfies three requirements. First, the study must have a valid scientific objective and a reasonable probability of achieving that objective. Second, the objective must be achievable only by animal research, and the species and number of animals to be used must be justified. The species should be the lowest on the phylogenetic scale consonant with the scientific objectives, and the numbers should be the lowest needed for statistical validity. Third, the level and duration of animal suffering that will occur must be minimized and must be considered acceptable. The third requirement is clearly the most complex and difficult of the three, and is the origin of the bulk of the questions in animal research protocol forms. In practice, it focuses on four major issues.

First, provisions for housing, feeding, and cleaning must conform to federal standards. Second, all surgical procedures and other traumatic or invasive techniques must be essential to the objective and must be accompanied by acceptable anesthesia, antisepsis, and postoperative care. Prevailing standards have come to match quite closely those followed in human surgery. Third, other likely suffering, such as that due to toxic or infectious agents, should be carefully and frequently monitored, and every possible step taken to reduce it. Thus, terminally ill animals must normally be euthanized to end suffering as soon as their condition is apparent. Death due to the experimental toxin or infection is no longer a routine endpoint. It is allowed only when essential to the objective. Furthermore, significant discomfort or distress must be alleviated or the animal must be euthanized. The standards for acceptable distress, like those for acceptable anesthesia and antisepsis, are coming to match those observed in clinical practice. Thus, a single generalized seizure or mild ascites may be acceptable, while status epilepticus or ascites severe enough to cause respiratory distress requires appropriate therapy or euthanasia. Fourth, and finally, euthanasia must be quick, uniformly effective, and accompanied by very little or no suffering.

Once a study begins, institutional veterinarians and other animal care personnel maintain close contact to ensure that the approved protocol is followed. Research projects which extend over years or have significant changes in goals or procedures must undergo committee reevaluation. In addition, animal research facilities and procedures are monitored by federal inspectors in periodic unannounced visits.

Finally, many scientific journals have adopted specific animal research principles, and have included them in their instructions for prospective authors.[41,42] Studies in which adherence to these principles is absent or doubtful are rejected. This salutary censorship is a final and ultimately very effective check on animal research practices.

THE ROLE OF THE PRACTICING PHYSICIAN

The Committee on Bioethical Issues of the Medical Society of the State of New York firmly believes that physicians should assert themselves as leaders and educate the public, their patients, and their elected public officials regarding the value of animals in research. When discussing treatment modalities, new or old, the role of research using animals should be stressed. When interfacing with the media or the public regarding a new development or an excellent clinical outcome, the animal research that made it possible should be emphasized. Physicians should be aware of the rules governing the humane treatment of animals so they can correct the incorrect statements of protestors and animal rights activists. When inaccurate and provocative statements are made in the press, physicians should write to the editors to set the record straight. Physicians should provide informational materials in their offices describing the value of animal research as it applies to their specialty. Physicians cannot and should not remain silent and let animal research be eliminated or severely impeded by the ill informed, irrational outcries of a minority of the public.

SUMMARY AND CONCLUSION

The New York Academy of Sciences, speaking on behalf of the scientific community, states that:

> The scientific community seeks to understand, improve and sus-

tain life and alleviate suffering. The pursuit of these goals may necessitate the use of animals when new information is sought and older information is to be reassessed for educational purposes, and alternate methods are unavailable. Concern for the welfare of animals used in research, testing, and education has led scientists to adopt more comprehensive standards so that gains in knowledge can be achieved using the smallest number of animals and the most humane methods.[33]

At present, animal research is under assault from a small but very vocal and energetic minority of the public. This attack is a serious threat to biomedical research and to the benefits it yields in improved human (and animal) health.

As highly visible and influential representatives of the scientific community, physicians can make an extremely important contribution to combating this growing danger by educating their patients and the public at large in regard to the great contributions animal research has made to medical care, its crucial role in future progress, and the humane treatment given research animals. The American Medical Association, in a resolution passed at its 1988 House of Delegates meeting, encouraged physicians to "place copies of material describing the medical benefits of animal research in their office waiting rooms" (posters, pamphlets, and advice are available at no cost from the Foundation for Biomedical Research, 818 Connecticut Ave, Suite 303, Washington, DC 20006).

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SPECIAL REPORT

A strategic plan for the elimination of tuberculosis in the United States*

In 1987, the Department of Health and Human Services (DHHS) established the Advisory Committee for the Elimination of Tuberculosis (ACET) to "provide recommendations for the development of new technology, application of prevention and control methods, and management of state and local tuberculosis programs targeted toward the elimination of tuberculosis as a public health problem." In response to this charge, the ACET completed a strategic plan for the elimination of tuberculosis (TB) in the United States with advice and consultation from a large number of persons and organizations. The following is a summary of the plan. The complete plan has been published as an MMWR supplement.[1]

The plan urges the establishment of a national goal of TB elimination (an incidence of <1 case per million population) by the year 2010, with an interim target of an incidence of 3.5 cases per 100,000 population by the year 2000. The plan cites three factors that favor the achievement of this goal: 1) TB is retreating into focal geographic areas and demographically well-defined groups; 2) biotechnology can potentially generate better diagnostic, therapeutic, and preventive modalities; and 3) new computer, telecommunications, and other technologies will enhance the transfer of these new biotechnologies into clinical and public health practice. A three-step plan of action is proposed:

Step 1. More effective use of existing prevention and control methods, especially in high-risk populations;

Step 2. The development and evaluation of new technologies for diagnosis, treatment, and prevention; and

Step 3. The rapid assessment and transfer of newly developed technologies into clinical and public health practice.

Current problems cited in the plan include deficiencies in identifying and reporting TB cases and contacts, the failure to fully use prevention interventions, the failure of many patients to complete prescribed therapy, and the failure to adequately assess the effectiveness of community prevention and control programs.

Recommended priorities for action include 1) identifying and screening high-risk population groups within each health jurisdiction and 2) making adequate and appropriate treatment and prophylaxis more widely available. Elimination of TB in the United States depends on the identification of groups at high risk for infection and disease. These groups vary through time, by place, and by personal characteristics. In 1987, the identifiable groups at high risk included HIV-infected persons, the homeless, immigrants and refugees from high-prevalence countries, intravenous-drug abusers, and residents of correctional institutions and nursing homes. Blacks, Hispanics, and Native Americans are also at high risk; the higher risk in these minority populations appears to be primarily related to socioeconomic status.[2] However, because the epidemiology of TB changes, populations now at high risk may decline in risk over time, and groups not currently identified to be at risk may become at risk. Therefore, the plan urges CDC and state and local health departments to continue and to strengthen TB surveillance activities and to further improve their ability to define groups at high risk for TB.

In addition to identifying high-risk populations, health-care providers must extend TB screening, treatment, and prevention programs to these groups. For such programs to be optimally effective, high-risk groups and health-care providers for these groups should be involved in designing, implementing, and promoting these programs.

To increase the proportion of patients who complete therapy, the plan recommends several actions, including the more widespread use of the newer short-course treatment regimens.[3] In addition, for each new case of TB, a specific health-care provider should be responsible for assuring that patients and their contacts are educated about TB and its treatment, that therapy is continued and completed, and that appropriate contact examination and preventive treatment are conducted. The use of directly observed therapy is strongly encouraged. Quarantine measures, including temporary institutionalization, are recommended only in those rare instances when an infectious patient refuses to comply with self-administered or directly observed therapy.

The implementation of these recommendations will require an increase in the number of health department outreach staff who are members of the populations they serve. During the past few years, this approach has proven successful in public health practice and is more cost-effective than alternative approaches such as long-term hospitalization (CDC, unpublished data).

Intensified use of existing technologies as outlined above is essential in moving the nation toward elimination; however, this strategy alone will not be sufficient to reach the goal. It is crucial that new technologies be developed. The plan points out that recent developments in biotechnology are revolutionizing the diagnosis, treatment, and prevention of other infectious diseases and that, by applying these new techniques to TB, it should be possible to develop the new tools needed to eliminate TB.[4]

The highest priorities for new technology development are 1) the development of alternative approaches to prevention of disease among persons already infected and 2) the development of a more rapid and effective test for identifying persons infected with living tubercle bacilli. Research efforts directed toward developing a more reliably effective TB vaccine, more rapid and accurate diagnostic tests, and more effective and rapidly-acting drugs are also needed.

Finally, new technologies must be assessed and put into clinical and public health practice in a timely fashion. The plan points out that federal agencies; professional societies; and schools of medicine, nursing, and public health all have a role in assessing and implementing new technologies and that both public and private funds will be needed to support demonstration

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projects for technology assessment and implementation.

Health departments, medical and nursing schools, schools of public health, voluntary agencies, professional societies, and minority advocacy groups share responsibilities for educating health-care providers and high-risk groups about the manifestations, methods of diagnosis, treatment, and prevention of tuberculosis. The plan recommends national, regional, and state conferences for health-care professionals to focus attention on TB and to teach modern approaches to its control and eventual elimination.

The plan suggests that advisory committees be established in the states and major metropolitan areas to develop more specific strategies and tactics for TB elimination in each health jurisdiction and to review progress toward elimination. These reviews should include evaluations of morbidity and mortality data, the adequacy of case reporting and casefinding procedures, and the quality of treatment and prevention activities. Interested constituencies, such as lung associations, minority organizations, and professional societies, should be represented on these advisory committees.

The ACET states that it is bringing this plan to the attention of the medical community and the public to stimulate positive and constructive discussion and action, to increase the level of TB awareness, and to encourage a commitment toward the elimination of TB. [Reported by: Div of Tuberculosis Control, Center for Prevention Svcs, CDC.]

Editorial Note: The TB elimination plan developed by the ACET provides a roadmap to guide the TB elimination effort for the next two decades. Consequently, the plan is being distributed to a wide variety of public, private, and voluntary groups with the request that they actively join in identifying and supporting steps essential to eliminating this disease within their respective jurisdictions.

Although the occurrence of TB in the United States has declined during the past 35 years, the disease persists as a public health problem in this country. From 1953 through 1987, the number of reported cases decreased from 84,517 to 22,255, and the annual incidence of TB decreased from 53.0 to 9.3 cases per 100,000 population.[5] The reduction has been substantially greater among whites than among other races; as a result, the proportion of cases occurring in nonwhites has risen from 24% in 1953 to 49% in 1987.[6] Today, TB among non-Hispanic whites is predominantly a disease of the elderly; among minorities, it is primarily concentrated in young adults. In 1987, the median age of non-Hispanic whites with TB was 62 years; for minority patients, the median age was 39 years.[6]

Foreign-born persons constituted 24% of patients in 1987, and the risk among immigrants from Asia is especially high, particularly in the first years after arrival in the United States.[5] The risk for immigrants serves as a reminder that TB persists as a global health problem of enormous dimension. Throughout the world, approximately 7–9 million new cases are diagnosed each year, and the disease is estimated to cause approximately 3 million deaths annually.[7] An estimated two billion persons in the world have latent tuberculous infection (International Union Against Tuberculosis, Paris, personal communication, 1988), making it one of the most prevalent infections in the world. Through the development of new technology, the TB elimination effort in the United States can potentially contribute to the solution of the global TB problem.

In the United States, new cases occur primarily among persons with longstanding *Mycobacterium tuberculosis* infection rather than among persons with recent infection. An estimated ten million persons have longstanding tuberculous infection (CDC, unpublished data). Major progress toward elimination can be achieved by targeting TB screening and preventive therapy programs toward groups of persons with *M tuberculosis* infection who are at high risk for developing clinical disease.

To accomplish this objective, health department TB-control programs must be maintained, strengthened, and continually evaluated to assure the most beneficial use of available resources. CDC will continue to assist health departments by providing technical and financial assistance, training and educational resources, and surveillance and epidemiologic assistance and by conducting applied and operational research. CDC will continue to work with advisory groups, other federal agencies, state and local health departments, minority organizations, and other organizations to develop more specific strategies and tactics for implementing the plan.

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Lead poisoning during home renovation

JOHN P. CURRAN, MD, JORGE R. NUNEZ, MD

In childhood lead poisoning, the major sources of lead have been leaded paint or plaster chips found in deteriorating pre-World War II housing and available for ingestion by children with pica. Only recently has the importance of lead dust which contaminates hands, food, water, kitchen utensils, and air been appreciated.[1–4] Rabinowitz et al,[5] in a 1985 report, noted that refinishing procedures of dwelling surfaces containing lead paint were associated with elevations of blood levels (Pb-B) averaging 69% in infant residents. Amitai et al,[6] and Rey-Alvarez and Menke-Hargrave[7] have reported a total of 17 children who experienced significant increases in Pb-B during procedures undertaken to delead their homes such as scraping, sanding, and burning of paint. We could find no report of childhood lead poisoning that occurred during elective structural modernization (not refinishing) of an old, well-maintained house.

CASE REPORT

A two-and-a-half-year-old boy was injured in an automobile accident and admitted to another hospital with a laceration of the right lower eyelid and minor contusions. A complete blood cell count revealed hypochromic, microcytic anemia (hemoglobin, 7.8 g/dL; hematocrit, 27.8%), which prompted blood lead (Pb-B) and zinc protoporphyrin (ZPP) determinations, reported as 79 μg/dL and 1,572 μg/dL red blood cells, respectively. This established a diagnosis of lead poisoning, Class IV, and the patient was transferred to this hospital for therapy.

The child's parents were both working professionals. He lived with them and two siblings (ages 12 and 9) in a pre-World War II single family brownstone dwelling located in a middle class neighborhood. The house was in good condition, with no deteriorating paint or plaster. For eight weeks prior to admission, extensive renovations had been ongoing, including demolition and replacement of most of the walls and plumbing, which generated large quantities of dust. The child sucked his thumb and drank more than 40 ounces of milk daily by nursing bottle.

Physical examination revealed a well-nourished and developed child who appeared healthy except for a sutured laceration of the right lower eyelid and scattered minor contusions. He was alert, cooperative, somewhat anxious, but not irritable. Neurologic examination was normal.

A complete blood cell count produced the following findings: white blood cell count, 9,800/mm^3 with 34% polymorphonuclear leucocytes, 0 band forms, 55% lymphocytes, 1% monocytes; hemoglobin, 8.4 g/dL; hematocrit, 27.3%, red blood cell count, 5.01 million/mm^3. The serum ferritin level was less than 3 μg/L. SMAC and urinalysis were within normal limits. The Pb-B level was 73 μg/dL; ZPP, 1,398 μg/dL red blood cells. Plain films showed no intra-abdominal opacities. ZPP levels on the parents and two siblings were 22.9, 22.6, 18.9, and 12.6 μg/dL red blood cells, and Pb-B levels were reported as 15, 13, 12, and 12 μg/dL.

Based on the Pb-B and ZPP determinations, the patient received three courses of chelation with Ca-EDTA and dimercaprol (BAL) at monthly intervals.

DISCUSSION

Child inhabitants of deteriorating housing stock, and adult deleading workers, have been reported to have significantly increased Pb-B levels during improper deleading procedures.[5–7] Wolf has reported increased lead absorption during restoration of old homes when various methods of paint removal were employed.[8,9] In this report, the source of lead was apparently dust generated during demolition and removal of interior walls. No scraping, stripping, or burning of paint occurred.

The importance of lead-containing dust as a source in childhood lead poisoning has been recognized. Good evidence was provided by reports of elevated Pb-B levels in children of lead workers for whom the only source of exposure was lead dust, brought into the homes on parents' work clothes,[10] and studies have shown that dust in inner city homes contains significant amounts of lead.[1–4] Since small children have extensive contact with dusty surfaces, wash their hands infrequently, and engage in normal hand-to-mouth activity, large quantities of leaded dust may be ingested over time. Also, during extensive renovation, food, water, and utensils will be contaminated with dust. There was no history of pica in the reported case and no other source of lead was identified.

In view of the Pb-B and ZPP levels in the older children and adults, inhalation of leaded dust was apparently not important. Iron deficiency probably contributed to the marked elevation of ZPP.

In some areas of large cities there is an influx of young, middle class, "upwardly mobile" couples with small children who have purchased housing constructed prior to WW II. Due to the increased availability of home equity loans, these old but sturdy houses are undergoing extensive renovations in increasing numbers, and large quantities of lead are being released into the environment in dust particles.

We believe that the present case demonstrates a hazard not sufficiently appreciated by pediatricians, and affecting primarily children in older middle class or upper class homes undergoing renovation or restoration. The American Academy of Pediatrics has recommended that "children of any age living in older housing where renovation is occurring be screened for lead absorption."[11] And the construction industry involved in such renovations should be aware of the risk to which their workers, and perhaps their workers' children, are exposed.

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Development of multiple carcinomas in a long term survivor of Wegener's granulomatosis treated with immunosuppressive drugs

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We report a case of a patient with Wegener's granulomatosis who was the longest known survivor of this disease (21.5 years). She was one of the first patients to be treated with azathioprine.[1] Squamous cell carcinoma of the skin and undifferentiated large cell carcinoma of the lung subsequently developed, of which she died. We also present a brief review of the literature on the development of second malignancies in various immunosuppressed states, such as homograft organ transplantation, Hodgkin's disease treated with chemotherapy and radiation, acquired immunodeficiency syndrome (AIDS), and Wegener's granulomatosis treated with alkylating agents.

CASE REPORT

A 42-year-old woman first presented at the Lenox Hill Hospital in April 1965, with fever and pulmonary cavitary lesions. Noted was a large necrotic, ulcerated, left para-ocular lesion (Fig 1), which on biopsy revealed Wegener's granulomatosis (Fig 2). There was erosion of the floor of the left orbit on radiography.

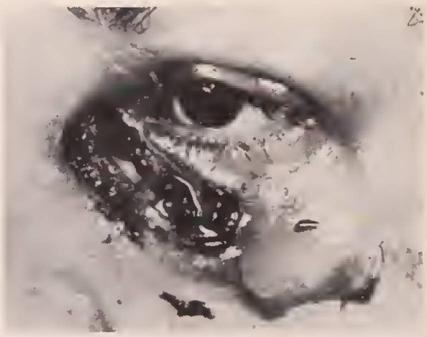

FIGURE 1. The large paraocular ulcerated lesion before therapy.

Initial high dose steroid therapy was unsuccessful; remission was achieved only with the addition of azathioprine to the regimen. The fever abated, the pulmonary lesions disappeared, and the para-ocular lesion shrank to a 1-mm diameter defect.[1] In 1967, because of reports of the greater efficacy of cyclophosphamide in immunosuppression, azathioprine was discontinued and cyclophosphamide was substituted at 50 mg three times a day, together with prednisone, 2.5 to 5.0 mg/day. In general, the disease process was well controlled on this regimen, but between 1967 and 1985, occasional exacerbations occurred when massive steroid therapy was instituted (prednisone, 100 mg/day), with rapid downward tapering. These episodes were characterized by fever, epistaxes, and secondary infections.

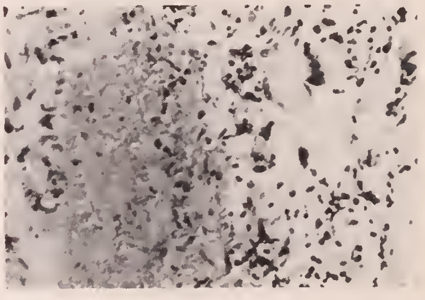

FIGURE 2. Histologic appearance of the left infraorbital lesion. Note the giant cell granuloma and necrotic arterial wall (original magnification ×155).

In 1971, infiltration of the left retro-orbital space led to paralysis of left cranial nerves II, III, IV, and VI. Radiotherapy was unsuccessful.

In 1982, gross hematuria developed, diagnosed through biopsy and attributed to interstitial cystitis associated with long term cyclophosphamide therapy. Cyclosporin A was substituted for cyclophosphamide, but had to be stopped within one week because of the progressive rise of the serum creatinine level. From that point on, prednisone, 2.5-5 mg/day, and azathioprine, 150 mg/day, were resumed. From 1982 to 1985, multiple cystoscopies for fulguration of bleeding points were necessary. In 1983, massive arterial bleeding from the left retro-orbital area was successfully treated with nasal packing and transfusions.

In September 1984, a routine chest film disclosed multiple nodular lesions. An open chest biopsy revealed a large cell, undifferentiated carcinoma of the lung (Fig 3). Staging revealed increased uptake on the bone scan in the lower thoracic and lumbosacral spine. Because of prolonged marrow suppression and lack of agreement on the part of the patient and her family, chemotherapy was not instituted. Low back pain presumed to be due to bone metastases was palliatively treated with local radiotherapy.

In September 1985, an ulcerative lesion of the left leg proved on biopsy to be squamous cell carcinoma. This was excised and did not recur. In October 1985, the patient was transferred to a cancer hospice, where her clinical course worsened, but the patient remained free of pain. On October 22, 1986, she died.

Postmortem findings disclosed Wegener's granulomatosis involving the upper lobe of the right lung; large undifferentiated carci-

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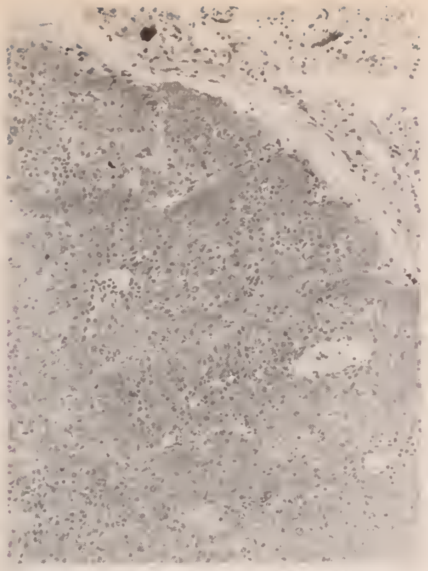

FIGURE 3. Large cell undifferentiated carcinoma of the lung. Note the large, pleomorphic nuclei.

noma of the right lung; organizing thrombosis of the pulmonary artery in the right upper lobe; chronic, although mild, pyelonephritis and hydronephrosis of both kidneys; cholelithiasis and chronic cholecystitis; and Paget's disease involving the lower thoracic and lumbar spine.

DISCUSSION

A causative relationship between immunosuppression and the development of neoplasia has been suspected and reported for more than a decade.[2-13] Valagussa et al[12] found a 7.3% incidence of solid tumors and a 2.4% incidence of acute nonlymphoblastic leukemia (ANLL) in 764 patients with Hodgkin's disease. The solid tumors occurred in patients given radiotherapy with or without adjuvant chemotherapy, and ANLL developed only after treatment with MOPP (mustine, vincristine, procarbazine, and prednisolone). In patients treated with ABVD (adriamycin, bleomycin, vinblastine, and dacarbazine) solid tumors or ANLL did not develop. Armitage et al[13] reported five cases of non-Hodgkin's lymphoid malignant neoplasms in 242 patients initially treated for Hodgkin's disease between 1973 and 1980, an incidence of 2.1%. Deeg et al[14] reviewed delayed complications of bone marrow transplantation in 450 patients and found 17 second malignancies (3.8%) distributed as follows: acute lymphoblastic leukemia (ALL), 3; acute myeloblastic leukemia (AML), 2; sarcoma, 3; Hodgkin's disease, 1; lymphoproliferative disorder, 2; follicular center cell lymphoma, 1; glioblastoma multiforme, 2; and adenocarcinoma of rectum, 2. The patients with glioblastoma multiforme and adenocarcinoma of the rectum had received radiotherapy to the brain and pelvis, respectively. Four hundred fifty-three de novo cancers developed in 432 organ homograft recipients, of whom 425 received kidneys, six had cardiac transplants, and one had a liver homograft.[15] The malignancies are listed in Table I. In a group of 320 patients with Hodgkin's disease followed for 25 years, second malignant neoplasms (SMN) developed in 21 patients; only two neoplasms were acute leukemias.[16] The SMNs are listed in Table II. The concept of second malignancies was further developed by Penn,[17] who found a variable latent period between commencement of treatment and the appearance of a new cancer. This interval is generally 10–15 years after radiation and may be as long as 50 years. For radiation-induced leukemia, the minimum latent period is two years, whereas that for chemotherapy-induced cancers is from a few months to 12 years. Recently, Ioachim et al[18] from our own institution reported 21 cases of lymphoma associated with acquired immunodeficiency syndrome (AIDS).

With respect to Wegener's granulomatosis treated with alkylating agents, there have been reports of five cases of leukemia,[19-22] and three cases of solid tumors—Kaposi's sarcoma,[23] histiocytic lymphoma,[24] and spindle cell cancer of the bladder[25]—have been reported in Wegener's granulomatosis treated with cyclophosphamide. To our knowledge, this is the first report of undifferentiated, large cell carcinoma of the lung and squamous cell carcinoma of the skin developing in Wegener's granulomatosis treated with azathioprine and cyclophosphamide. The oncogenetic mechanism in all these groups is presumed to be decreased immunosurveillance, allowing cloning of non-self, neoplastically transformed cells, with mutagenesis being induced by alkylating agents and radiotherapy.

TABLE I. De Novo Malignancies in Organ Allograft Recipients[*]

Type of Malignancy[†]	No. of Patients
Cancers of skin and lips	674
Solid lymphomas	314
Reticulum cell sarcomas	155[†]
Kaposi's sarcomas	60[†]
Unclassified lymphomas	42
B-cell lymphomas	26
Hodgkin's disease	10
Lymphosarcomas	8
Plasma cell lymphomas	6
Lymphoreticular malignancies	4
Burkitt's lymphoma	1
Histiocytic medullary reticulosis	1
Mycosis fungoides	1
Carcinomas of uterus	
Cervix (111); body (12)	123
Carcinomas of lung	86
Carcinomas of colon and rectum	66
Carcinomas of breast	55
Leukemias	45
Carcinomas of vulva and perineum	40
Miscellaneous	364
Total	1767

[*] Reprinted with permission from Penn.[15]

[†] There were 1,661 patients, of whom 106 had more than one type of tumor. One patient had Kaposi's sarcoma and a reticulum cell sarcoma.

TABLE II. Subsequent Maligancies with Hodgkin's Disease (in 320 Patients Followed for 25 Years)[*]

Type of Malignancy	No. of Patients
Cancer of cervix	3
Cancer of colon	3
Cancer of breast	2
Acute nonlymphoblastic leukemia	1
Acute myeloblastic leukemia	1
Cancer of esophagus	1
Cancer of ovary	1
Mesenchymoma of thigh	1
Melanoma	1
Leiomyoma	1
Sarcoma	1
Cancer of parotid	1
Myeloma	1
Lymphoma of tonsil	1
Cancer of pancreas	1
Astrocytoma, spinal cord	1
Total	21

[*] Reprinted with permission from Rubin P, Zagars G, Chuang C, et al.[16]

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Transient cortical blindness following an acute hypotensive event in the postpartum period

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In this age of advancing medical technology and knowledge about the pathophysiology of cardiopulmonary arrest, many more patients survive this and other hypotensive events. As a consequence, many of the survivors suffer the effects of cerebral hypoperfusion and hypoxia. Several neurologic sequelae have been described.[1,2] This case report describes a previously healthy postpartum woman in whom transient cortical blindness developed subsequent to a pulmonary embolism and profound hypotension.

CASE REPORT

A 40-year-old nullipara woman with no known significant medical history was admitted at 38 weeks' gestation with spontaneous rupture of membranes and no uterine contractions. She subsequently underwent a primary low segmental cesarean section; a healthy baby girl was delivered with Apgar scores of 9 and 10 at five and ten minutes, respectively.

The postpartum course was uneventful until day two, when the patient complained of abdominal pain. A diagnosis of nonobstructive ileus was made, based on physical examination and radiographic findings. Decompression was effected by a nasogastric tube.

On day three, the patient was found to be cyanotic with a systolic blood pressure of 60 mm Hg. An endotracheal tube was immediately introduced, with subsequent cardioversion after ventricular tachycardia developed. An emergency perfusion scan demonstrated multiple pulmonary perfusion defects. In light of an unremarkable chest roentgenogram, a diagnosis of pulmonary embolus was made, and the patient was transferred to the medical intensive care unit; anticoagulation therapy with heparin was begun shortly thereafter.

Approximately six hours later, the patient was observed not to be responding normally to visual cues. Physical examination revealed normal pupillary reflexes, benign fundi, no response to optokinetics, and absent blink reflexes. The severity of the visual impairment precluded the testing of visual fields. Heparin therapy was discontinued. A computerized axial tomographic (CAT) scan of the head at that time was normal. As a small hemorrhage secondary to anti-coagulation therapy could not be completely ruled out, and the need to prevent further embolization was also paramount, a Kimray-Greenfield filter was placed in the inferior vena cava.

The patient's endotracheal tube was removed the following day. The previous visual assessment was confirmed, and visual field testing revealed that peripheral vision was affected less than central vision. Additionally, the patient was found to be mildly confused, with impairment of recent memory. An electroencephalogram (EEG) showed evidence of mild diffuse disturbance of cortical activity with no clear localized or lateralized disturbances; there was no posterior alpha activity (8-12 cycles per second). The predominant posterior activity was an intermixture of 18-20 cycle per second waves and some low amplitude, 4-5 cycle per second waves; these frequencies did not react to eye opening. Photic stimulation induced a driving response at 10 flashes per second. A repeat CAT scan of the head was normal. The patient's vision improved prior to discharge, and the deficit has resolved completely.

DISCUSSION

Cortical blindness is characterized by an inability to see in spite of intact anterior visual pathways. It is often due to a border zone infarct (bilateral) in the distal branches of the posterior cerebral artery.[1] This "watershed" area is one of the first to be affected by generalized hypoxia because of its distance from the origin of cerebral circulation.[3] Other causes include bilateral occlusions of the middle cerebral or posterior cerebral arteries (secondary to cerebrovascular accidents, bacterial meningitis, or meningeal carcinomatosis), toxins, and cerebral edema.[4]

Although cortical blindness following cardiac arrest has been previously described,[4-6] a recent retrospective study suggests that it is most common in surgical patients with cardiovascular

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disease, and that it is only a rare complication of nonoperative cardiac arrest.[7] Postpartum cortical blindness has also been previously described.[8-12] In those reports, the etiology was thought to be a cerebral venous thrombosis. This current report describes a previously healthy woman in whom cortical blindness developed secondary to an acute hypotensive episode related to a pulmonary embolus. The diagnosis was made based on the history of profound hypotension (and presumed cerebral hypoperfusion), as well as physical examination, CAT scan, and EEG findings.

The response to photic stimulation does not preclude a diagnosis of cortical blindess; Aldrich et al[7] found that three of 20 patients with cortical blindness had similar EEG findings. The role of visual evoked potentials and pattern reversal potentials is unclear regarding diagnosis and prognosis in cortical blindness.[7] The absence of headache, convulsions, and focal neurologic signs made a diagnosis of cerebral venous thrombosis less likely.[10] Delay in diagnosis may have occurred because of Anton's syndrome (cortical blindness with denial of the visual defect) or the patient's inability to communicate (secondary to intubation), or because of global impairment of neurologic function.

In summary, many significant hypotensive events have been found to result in several types of neurologic sequelae[1,2]; many of these may go undetected because of several factors (including the transience of symptoms). In light of conflicting reports in the literature regarding the true incidence and etiology of cortical blindness, as well as the potential difficulty in making a prompt diagnosis following a hypotensive event, we believe that a detailed neurologic examination is indicated, even in the absence of symptoms or obvious focal findings. Additionally, further prospective studies may help to define the true incidence of cortical blindness following such events, because of the diagnostic difficulties described above.

Acknowledgment. The authors thank Herbert Diamond, MD, Steven Horowitz, MD, Neil Schaul, MD, and Janet Attanasio for their contributions to the completion of this case report.

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Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

House staff stress and the impact of the new regulations on postgraduate medical education

TO THE EDITOR: The special articles by Drs Walerstein, Rosner, and Wallace[1] and by Reiner[2] dealing with house staff stress and the impact of the new regulations on postgraduate medical education that appeared in the August issue of the *Journal* need dissemination to the public. Doctors have heard it many times over already.

All would agree that curricula need to be revised and changes need to be made in the medical care system to lessen the stress of the work load. Residents put years into their educational training, and must absorb and retain an enormous amount of information. Most people outside of medicine still do not understand the vastness of the knowledge that must be acquired and the enormity of stress incurred. Perhaps the answer lies in developing different training pathways during medical school so that programs in psychiatry, surgery, pediatrics, internal medicine, and so on can be fashioned early on to suit an individual's preference. Acute intensive care medicine, emergency medicine, and ambulatory medicine programs could be similarly fashioned to the individual. This would help, but would result in physicians less exposed to the overall world of medicine. Society might be sorry in the long run. Nonetheless, since medicine has changed, these changes may also be necessary.

The issue of sleep deprivation remains controversial. The new regulations are humane, but they may also be an overreaction by an upset public and an upset department of health. For instance, it is not generally known to the public that at certain levels of their training residents perform some procedures more adequately than more senior physicians. How can we explain this "paradox" to people who are outside of medicine? Can we liken the medical care team to a baseball team where the residents are the players and the more senior physicians are the managers, coaches, and scouts? That analogy also does not seem to fit.

Although the Bell Commission recommendations are commendable they are more idealistic than realistic. Neither the finances, the pool of future quality doctors, nor the availability of the correct type of senior doctors are within the "do-able"—at least for a decade or two. And, it is doubtful that even with creative changes in education and training and a possible resurgence of a desire to go into medicine that we will be able to achieve what the commission has asked for. As Walerstein et al say, "in the future . . . with more economic competition and lower incomes and more regulation and governmental control a likely scenario,"[1] what reasonable, logical, and even empathetic person would choose to enter medicine?

The stress factor is inevitable. Physicians in training must develop a healthy coping mechanism or risk jeopardizing their future career. Perhaps some physicians' attitudes towards their patients and work load are residua from dealing poorly with stress while in training. Currently, more and more residents who finish their training are taking "year-outs" to rethink their future, often taking a temporary position (usually at a high salary) to begin to pay off the financial debts that they have incurred as a result of educational training costs. A future physician must face the dilemma of getting out of financial debt, dealing with the potential for poor psychological and physical health, and possibly being berated by society and not being able to look forward to a reasonable station in life. What other profession goes through this? True, physicians deal with people's lives, not with their homes, cars, rights, or education—but still there is poor understanding, little compassion, and not much encouragement from individuals outside the medical profession to help resolve some of the difficult issues that confront house officers and their lives. The public wants physicians who are knowledgeable, compassionate, and who do not make mistakes, but the regimentation of the system discourages the best of this type of individual from wanting to get involved.

As a member of an internship selection committee, I have interviewed future first year residents. These applicants have expressed concern about and question how the Bell Commission regulations will impact on their learning. There was more concern with this aspect of their future residency programs in New York State than with how the regulations would lessen their work load and shorten their working hours. The regulations will be difficult to implement in New York State. They may drive quality applicants to other states. They may increase the number of "average" applicants who train and remain in New York State. These consequences will be further extended if other states pass similar legislation. The future may find the best medical care skimmed off and lost to the less regulated states. This remains to be seen.

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2. Reiner SC: The impact of the new code regulations on postgraduate medical education in New York State. *NY State J Med* 1989; 89:457-461.

The future of medicine

TO THE EDITOR: I wish to comment on Dr Andrew I. Elkowitz's letter in the August 1989 issue, "The future of medicine: A resident's perspective."[1]

His is a long letter and I want to dispute only a few of his points, although the whole of it goes against my grain. He accuses us older physicians of having "led medicine to its present predicament by our dictums." If indeed American medicine is in a predicament—I am not sure which one he refers to—we have not dictated it, but, alas, let it happen by our passivity. He then goes on and puts a dollar price on human life—be it $208,000 or $8.5 million. This is no way for a doctor to look at health care. The whole idea of equating it with dollars is wrong.

It is true that the resources of our society are finite and limits must be placed on what we as a society can afford for our health care. Dr Elkowitz draws a parallel between health care and automobile manufacture. I disagree! If we are indeed willing to pay $8 million to a prize fighter, then we should be able to afford better care for all people in this country. If triage should ever become necessary, only medical and humane considerations should prevail, never counting the cost.

And that brings me to Dr Elkowitz's six responsibilities:

1. "We should act justly." Of course, everybody should, but he mixes up our profession with the law, in naming this platitude as the very first command.
2. "We should act responsibly." This is again a banal generalization.
3. "We should act ethically." This is diminished by a long parenthetical comment. There is indeed controversy about medical ethics in some details, but the Hippocratic Oath still prevails.
4. "We should treat the poor benevolently." In truth we should not make any distinction in how we treat the rich or the poor and in our (the "old") way we did not.
5. "We should maximize the efficiency of providing care." This translates to "make as much money as possible." I am old-fashioned enough to hate both "maximize" and "efficiency."
6. "We should be good at what we do." This is not a new discovery, either.

I would replace Dr Elkowitz's six points with only one: "Try to help your patients using your medical knowledge, tempered by humane attention to the general circumstances."

The "specter of socialized medicine" haunts Dr Elkowitz, which surprises me in one so young (or is he?). In fact, we are the only industrial nation which has not yet admitted that we need a national health care system such as all nations in Europe have had for years and Canada has on our continent. The mess we have with Medicare, Medicaid, HMOs, and their various offspring leaves millions of our citizens without adequate medical care whatsoever, and that is our predicament!

We do have Social Security and let no one touch it. We need national health care now and we can afford it if we want it.

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1. Elkowitz A: The future of medicine: A resident's perspective [letter]. *NY State J Med* 1989; 89:484–486.

Tombstone erected on grave of Arthur Donaldson Smith, MD

TO THE EDITOR: In 1987 the *Journal* published a series of articles on Dr Arthur Donaldson Smith, a famous physician-explorer.[1] Dr Smith, who died in 1939, was buried in Laurel Hill Cemetery in Philadelphia where his grave was unmarked for half a century. In early 1989, my late husband, Dr H. Spencer Potter, a grandnephew of Dr Smith, and I arranged for a tombstone to be erected on Dr Smith's grave. This was done through the United States Army as Dr Smith had served as a captain during World War I. We also thought it was appropriate to have the subtitle of the *Journal* series about him, "Physician, Explorer, Naturalist, and Diplomat," inscribed as an epitaph on the stone beneath his name, dates, and rank. I thought that this information would be of interest to readers.

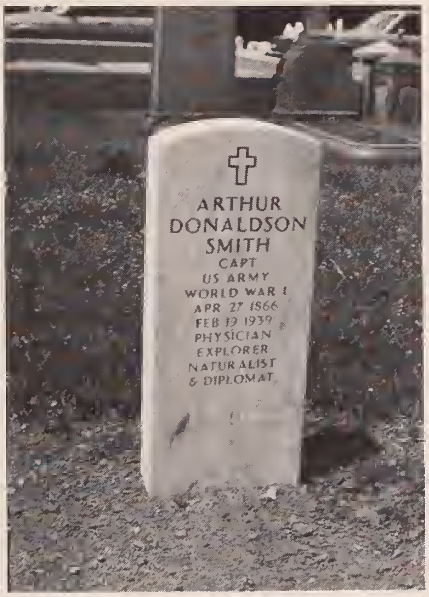

FIGURE 1. Grave of Dr Arthur Donaldson Smith in Laurel Hill Cemetery, Philadelphia.

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1. Imperato PJ: Arthur Donaldson Smith, MD: Physician, explorer, naturalist, and diplomat. *NY State J Med* 1987; 87:90–105; 161–176; 210–225; 334–351; 436–465.

The academic boycott of South Africa

TO THE EDITOR: I wholeheartedly endorse the position of Crawshaw,[1] who condemns the Royal College of Psychiatrists (United Kingdom) for invoking academic sanctions against South African psychiatrists. I recently returned from a lecture tour in South Africa and can attest to the fact that the medical academic communities at the universities of Cape Town and Johannesburg (Witwatersrand) are in the forefront of the fight against apartheid. At great personal risk, many of the faculty members speak out, write, and demonstrate against South Africa's racist policies. The University of Cape Town is fully integrated in terms of students, faculty, and employees and the Groote Schuur University Hospital also has full integration of patients and patient facilities. The University of Witwatersrand is following the example and lead of its sister university in Cape Town.

The academic boycott of South Africa, coupled with the "brain drain" of talented academicians leaving the country will, in the long run, hurt South Africa and its people, both black and white, much more than economic, cultural, sports, or other boycotts. Let us not throw out the baby with the bath water. Let us maintain academic ties with our medical colleagues in South Africa and support them in whatever ways we can in their struggle to overcome apartheid.

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1. Crawshaw R: Academic sanctions against South African psychiatrists by the Royal College of Psychiatrists [letter]. *N Engl J Med* 1988; 318:1624.

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Lyme disease surveillance in New York State

Lyme disease is currently the most common arthropod-borne disease reported in the United States to the Centers for Disease Control (CDC). In the northeastern United States, Lyme disease is transmitted by the bite of an infected *Ixodes dammini* tick, commonly known as the deer tick. The etiologic agent of the disease is a spirochete, *Borrelia burgdorferi*. Lyme disease is named for Lyme, Connecticut, where it was first recognized as an infectious disease in 1975.[1]

The disease has been divided into three clinical stages that are not always distinct.[2] Primary or nondisseminated Lyme disease usually manifests as a unique skin lesion, erythema migrans (EM). EM typically appears about one week after the tick bite (range 3 to 32 days) and often begins as a macule or papule surrounded by an area of redness. The lesion expands to an appreciable size (median diameter 15 cm; range 3 to 68 cm).[3,4] As the lesion expands, it often clears or fades in the center. EM is often described as a burning sensation or being warm to the touch. Multiple lesions are not uncommon. EM is clearly distinct from ringworm, dermatitis, local reaction to an arthropod bite or other common dermatologic complaints. In some cases, primary Lyme disease may not develop or may not be recognized. In other instances, Lyme disease may be self-limiting and may not progress beyond the early stages.

Secondary Lyme disease is most often characterized by migratory joint pain or brief arthritis in the large joints. Nervous system or transient cardiac involvement may occur. Tertiary Lyme disease is most often characterized by frank arthritis, again typically involving the large joints. Lyme disease should be aggressively treated with antibiotics[5,6] in its early stages before refractory manifestations develop. Treatment failures have occurred with all stages of the disease, though they are more common with the later stages.

Lyme disease first became reportable throughout the state in 1986. The number of reported cases has increased dramatically from 443 in 1986, to 877 in 1987, to 2,553 in 1988 (Fig 1). New York State has been disproportionately affected, comprising almost 40% of the total cases reported to the CDC in 1988. Several factors have contributed to the growing number of case reports, including better surveillance by local health units, increased sensitivity and reporting by the medical community, a heightened awareness among the general public, as well as an increase in Lyme disease incidence.

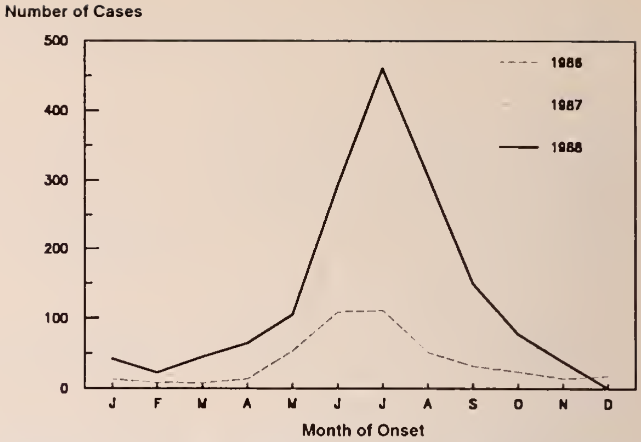

FIGURE 1. Lyme disease cases by month of onset, New York State, 1986–1988 (NYS-DOH surveillance data, case reports through 12/88).

The total Lyme disease cases reported each year do not necessarily represent the true incidence of Lyme disease for that year. Of the 2,395 cases reported in 1988 in New York State, exclusive of New York City, 1,634 (68%) reported onset of symptoms during 1988. However, 454 (19%) reported onset in previous years and 131 (5%) had unknown years of onset.

Supplemental surveillance data forms completed by physicians and local health units were received for 1,656 (69%) of the 2,395 cases of Lyme disease reported in New York State, exclusive of New York City. Further analysis described here is limited to 1,479 (62%) cases that meet the New York State Department of Health surveillance confirmed case definition:

- history of EM or symptoms clinically compatible with late stage Lyme disease and a reactive serologic test;
- history of exposure in an area (county) known to be endemic for the disease.

This differs from the CDC case definition, which requires all confirmed cases to have a history of EM.

In the March 1988 issue of *Epidemiology Notes*, an analysis of 600 serologically confirmed cases surveyed between 1982 and 1985 was presented. The supplemental information was voluntarily submitted by physicians requesting serologic testing by the New York State Department of Health. Strict comparisons of the current and previous data are not possible because of the different methods of sample collection.

The New York State Department of Health defines a county as endemic for Lyme disease if ticks infected with *B burgdorferi* have been found or if confirmed cases of Lyme disease have been reported in county residents who have no history of travel to known endemic areas. Counties considered endemic for Lyme disease prior to 1986 included Nassau, Suffolk, Westchester and Putnam. Since 1986, three additional counties have also been classified as endemic: Dutchess, Orange and Ulster. Uninfected *Ixodes dammini* have been found in seven other counties that border the endemic counties. Surveillance shows that Lyme disease continues to spread throughout Long Island and north along the Hudson River Valley.

Approximately 90% of reported Lyme disease cases come from Suffolk and Westchester counties each year (Table I). Incidence rates are not uniform within any given county; the highest incidence of Lyme disease is found in eastern Suffolk and northern Westchester counties. In 1988, 94 of the 2,395 upstate cases were reported in counties not currently considered to be endemic for Lyme disease; most of these cases reported a history of travel to an endemic area.

EM and arthritis were the most common manifestations reported. A history of EM was reported by 760 (51%) of the cases, arthritis by 508 (34%), arthralgia (but not arthritis) by 446 (30%) and neurologic manifestations by 157 (11%). These included peripheral neuropathy, Bell's palsy, meningitis, encephalitis and radiculitis. Cardiac manifestations were reported by less than 1% of the cases.

A total of 822 (56%) of the confirmed cases reported onset of symptoms during the months of June, July or August (Fig 1). EM was most common during the summer. Seventy-four percent of those cases reporting EM reported the onset as occurring during those summer months. In contrast, arthritis was the most common symptom reported during the winter. A history of tick bites was reported by 421 (28%) of the cases. Tick bite was more commonly reported by those with EM alone than those with later stage symptoms of the disease. Hospitalization was reported for 7.5% of the cases.

Caution is advised when interpreting laboratory results as serologic testing for *B burgdorferi* antibodies is not diagnostic of Lyme disease without consideration of the clinical presentation of the patient. Serum drawn in the early stages of EM may be negative. Positive IgG titers indicate infection at an unspecified time or may be a false positive due to cross-reactivity with other organisms antigenically similar to *B burgdorferi*. Lyme disease serologic testing is further complicated by variability among different laboratories and the different test methods available.

TABLE I. Lyme Disease Incidence in Endemic Counties of New York State, 1986–1988

	No. of Cases/Incidence per 100,000 Population[a]					
County	1986		1987		1988	
Dutchess	3	1.2	11	4.2	66	25.4
Nassau	7	0.5	23	1.7	131	9.9
Orange	3	1.0	8	2.8	43	14.9
Putnam	22	26.6	20	24.2	72	87.2
Suffolk	197	14.9	446	33.8	1,238	93.8
Ulster	2	1.2	1	0.6	10	6.0
Westchester	182	21.0	253	29.2	741	85.6
All non-endemic counties (except NYC)	3[b]		7[c]		93[d]	
NYC	24	0.3	108	1.5	158	2.2
NYS (except NYC)	419	4.0	769	7.3	2,395	22.7
Total NYS	443	2.5	877	4.9	2,553	14.3

a) Estimated 1988 population, NYS DOH Bureau of Biometrics
b) Residents of 3 non-endemic counties
c) Residents of 4 non-endemic counties
d) Residents of 29 non-endemic counties

Lyme disease prevention consists of the following strategies:

- Wear light-colored clothing so that unattached ticks may be easier to see and tuck pants into socks. Stay in the center of paths in tick infested areas.
- Check yourself and your children for ticks every two to three hours while outside and make a full body check while inside.
- Use repellants containing the compound DEET (diethyl toluamide) which is effective against ticks. Follow the instructions for use.
- Use tweezers to remove attached ticks by grasping them across the mouth parts and pulling upward and outward with a firm, steady pressure. Be careful not to squeeze, puncture or crush the tick.
- See a physician if the symptoms of Lyme disease develop.

In summary, Lyme disease surveillance depends upon a broad spectrum of elements, including review of specific clinical and laboratory data, information regarding travel or possible exposure to disease bearing ticks and collection and surveillance of ticks themselves. The New York State Public Health Law requires physicians to report all cases of Lyme disease to their local health department.

For more information about Lyme disease, or to receive a public information pamphlet, contact the New York State Department of Health, Arthropod-borne Disease Program, ESP Corning Tower, Room 651, Albany, NY 12237.

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Birth outcomes in New York State

This article represents the first part of a two part series that summarizes the findings of the New York State Department of Health Birth Outcomes Study. Birth outcomes that were examined in this study include fetal and infant mortality, low birthweight, congenital defects, newborn screening results, frequency of miscarriages and abortions, inclusion in selected neonatal diagnosis related groups (DRGs) and inclusion in the Infant Health Assessment Program (IHAP). In addition, sociodemographic variables such as maternal age, race, ethnicity, education, marital status and use of prenatal care were studied in conjunction with these outcomes. These analyses are strictly descriptive in nature and, thus, cannot fully explain the complex interactions of host, agent and environmental factors. During 1987, there were 271,355 births to New York State residents with an additional 145,264 induced abortions and 2,442 spontaneous fetal deaths.

Fetal Mortality

The rate of spontaneous fetal deaths (20+ weeks of gestation) has decreased within the last decade. In 1977, the rate was 11.9 per 1,000 and fell to 8.9 per 1,000 in 1987, a reduction of 25.2%. This reduction was evident in both the New York City population as well as the rest of the state. The nonwhite spontaneous fetal death rate (11.6 per 1,000) was 58.9% higher than the white rate (7.3 per 1,000) in 1987. The spontaneous fetal death rate increased from 1986 to 1987 from 8.8% to 8.9%.

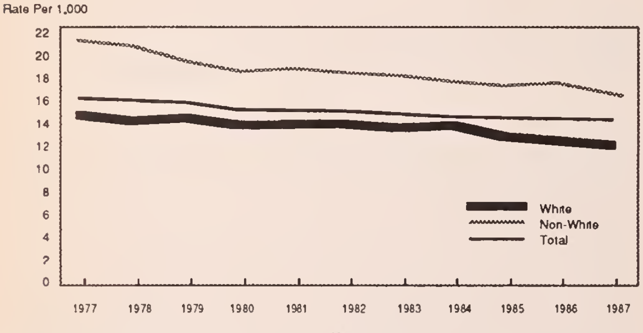
Infant Mortality

The infant mortality rate in New York State for 1987 was 10.7 per 1,000 live births. This rate increased 0.1 per 1,000 after a long period of continuous decline and was higher than the provisional national rate of 10.0 per 1,000. New York City residents had an infant mortality rate that was 41.1% higher than for residents in the rest of the state (12.7 versus 9.0 per 1,000).

The difference in infant mortality rates between white and nonwhite residents has not changed substantially from 1977 to 1987 (Fig 1). Nonwhite residents of the state had a rate 77% higher than their white counterparts (14.0 versus 7.9 per 1,000). Hispanics had the lowest infant mortality rate (6.8 per 1,000) compared to non-Hispanic white (7.9 per 1,000) and non-Hispanic black (17.5 per 1,000) births.

Cause of Death

In 1987, there were 2,893 infant deaths to New York State residents (Fig 2). Of these deaths, 68.6% (1,985) occurred during the neonatal period (first 28 days of life); 31.4% (908) were postneonatal (between 28 days and one year). Of the neonatal deaths, 18.8% were due to congenital anomalies (International Classification of Diseases (ICD) 740-759), 20.1% due to short gestation-low birthweight (ICD 765), 11.2% to respiratory distress syndrome (ICD 769) and 38.2% to other conditions originating in the perinatal period (ICD 760-764, 766-768, 770-779).



Office of Public Health

FIGURE 1. Infant mortality rates by race, New York State, 1977–1987 (from the Office of Public Health).

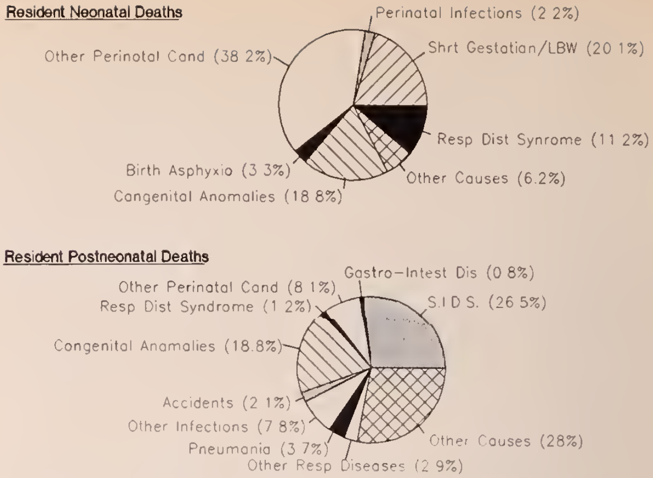


FIGURE 2. Cause of neonatal and postneonatal deaths, New York State, 1987 (from the Office of Public Health).

For postneonatal deaths, the leading cause was sudden infant death syndrome (ICD 798) comprising 26.5% of the total. Congenital anomalies was the second leading cause at 18.8%, followed by other conditions originating in the perinatal period (8.1%).

Low Birthweight

The condition that poses the greatest threat to the health of newborn infants is low birthweight. Fully two thirds of deaths in the first four weeks of life occur among infants weighing less than 2,500 grams at birth. For residents outside New York City, the infant mortality rate in 1987 for those less than 2,500 grams was 71.4 per 1,000, compared to approximately 4.9 per 1,000 for babies greater than or equal to 2,500 grams.

Changes in the rates of low birthweight births within the last decade have not paralleled the changes in the infant mortality rate during this time period. The rate of births less than 2,500 grams declined from 7.8% of all live births in 1977 to 7.6% in 1987. There was a 7.4% increase in the occurrence of births less than 2,000 grams in the same decade, from 2.7 to 2.9%.

As with the infant mortality rate, the white, nonwhite differential is also evident when comparing the percentage of low birthweight infants. The rate for nonwhite residents of New York State was 12.0%, double the rate for white residents. Although Hispanic births had the lowest 1987 infant mortality rate, when compared to non-Hispanic white and black births, this pattern did not reoccur with low birthweight. Non-Hispanic whites had the lowest percentage of low birthweight infants (5.3%) in 1987 as compared to Hispanic (8.4%) and non-Hispanic black (13.6%) births.

The premature birth (less than 37 weeks gestation) rate has remained fairly constant through the last decade. The rate of 9.9% for 1987 is slightly higher than the rate of 9.4% in 1977. The pattern for small gestational age (SGA) infants, defined as births with 37 or more weeks gestation and weighing less than 2,500 grams, is almost opposite that for premature infants. There has been almost a constant decrease in SGA births from 1977 to 1984 (4.0% to 3.3%), with an increase from 1984 to a rate of 3.7% in 1986. The SGA rate dropped to 3.4% in 1987.

Congenital Malformations

The Congenital Malformations Registry collects clinical and demographic data on every child born with a congenital malformation that is diagnosed by the time the child is two years old. The registry now has data on its first and second cohort, children born in 1983 and 1984. In 1984, 17,228 children were born with a reported congenital malformation, or 6.9% of all live births. More than 80% of these children were identified as having one malformation. The rate of malformations was highest among black children (7.5%), followed by white (6.7%) and other nonwhites (6.1%). Of the 22,580 malformation reports received, the organ system most frequently affected was the musculoskeletal (22.2%), followed by the cardiovascular (18.7%) and integument (12.8%) systems.

The second part to this series will focus on those maternal variables that have been associated with poor birth outcomes. Specifically, the variables that were examined include maternal age, race, socioeconomic status, level of education, parity and absence or inadequacy of prenatal care.

This article was adapted from a report entitled *Birth Outcomes in New York State*. Limited copies of the report may be obtained by contacting the New York State Department of Health, Office of Public Health Information Group, Corning Tower, Room 890, Empire State Plaza, Albany, NY 12237.

FROM THE LIBRARY

EMPATHY AND SENSIBILITY OF HEART

When I was a medical student before the era of antibiotics, it was generally considered that among the highest qualities of the physician's sensibilities was his skill at the art of physical diagnosis. The doctor who could detect an ailment from the odor of a patient's breath or from his facies or from the position in which he held his body or turned his eyes and who, walking into a room, was heard to mutter, "fetor hepatica," risus sardonicus," or "uremic fetor," moved on an ethereal plane.

The physicians' reliance on his physical senses in place of the highly complex diagnostic tools and laboratory technics was not merely a lingering nostalgia for the days of their teachers' teachers, but it was a reaction to and almost a disdain for the growing medical reliance on technology and specific drugs. Perhaps it was the voice of physicians of all ages saying, "Remember yourself, and use yourself in the diagnosis and care of your patients."

As the years passed and medicine has grown even more mechanically complex and drugs more marvelously miraculous, there has simultaneously developed a deeper interest in the human side of medical practice, the personal relationship between patient and physician. We have come to a time when the therapeutic effectiveness of the physician himself is being given its deserved attention.

Without the quality of empathy the physician, except in emergencies and acute illness, may be only a highly effective technician. Physicians often consider the popular alarm about the state of the doctor-patient relationship in terms of economics. I believe that it is not the rising costs of the practice of medicine that account for the popular anxiety but a fear at the passing of the "doctor" who, relying on his senses and his belief in himself, gave greater relief of patients' anxieties than all his drugs, laboratories, and chromium-plated equipment and accessories. These things have no heart; they are only "things"....

In 1772 John Gregory, professor of medicine at the University of Edinburgh, wrote, "I now come to mention the moral qualities peculiarly required in the character of a physician. The chief of these is that *sensibility of heart* [italics mine] which makes us feel for the distresses of our fellow sufferers, and which of consequence incites us in the most powerful manner to relieve them....

Sensibility of heart is an ability to perceive or receive sensations, a responsiveness to stimuli, a refined sensitiveness in emotions. The professor was writing about the *physician's heart*. So much interest has been devoted to the psychology of the patient that the psychology of the physician has been somewhat neglected. But often it is *his* attitudes and feelings which determine the patient's attitude, behavior, and indeed recovery....

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(*NY State J Med* 1957; 57:2410–2413)

A PIECE OF MY MIND. AMERICA'S DOCTORS SHARE THEIR MOST DRAMATIC, INSPIRING, AND MOVING EXPERIENCES

Edited by Bruce B. Dan, MD, and Roxanne K. Young. 256 pp. New York, Feeling Fine Programs, Inc, 1988. $18.95 (hardcover)

In Zen Buddhism neophyte monks learn to meditate upon a single thought until, in a mind splitting instant of enlightenment ("Satori"), the heavens split and a bolt of light illuminates their minds, producing a moment of total clarity and profound understanding. I would hope that most of us involved in caring for people would have had such an experience in our medical lives. For me, such an experience occurred with the bare-handed delivery of my infant daughter with the surprised collaboration of my laboring wife. It was a moment of the surest terror and joy, a moment since unduplicated.

In *A Piece of my Mind*, subtitled *America's Doctors Share Their Most Dramatic, Inspiring, and Moving Experiences*, Bruce B. Dan and Roxanne K. Young, medical writers and editors, "bring together the best of the searingly honest, immensely popular column called "A Piece of my Mind," which has appeared weekly for the last eight years in the *Journal of the American Medical Association*."

The book is composed of 80 vignettes, each more compelling than the last, that detail such moments of enlightenment in the careers of medical students, nurses, and physicians. The content is pure emotional dynamite. I found it hard, despite the brevity of each of the pieces, to read more than a few at each sitting; the experience was too emotionally devastating. In the piece entitled "Sudden Intimacies," the intense quality of physician-patient interaction is described as "those moments when the human barrier cracks open to reveal what is most secret and inarticulate. These revelations can fuse together desperate lives and unexpected kinship."

In "Not On My Shift" successive teams of intensive care unit residents battle to hold off the imminent death of a hemorrhaging patient. At sign-out each resident pronounces, in deadly serious tones, that this patient's impending demise will "not be on my shift!" A gem of a piece entitled "Internship—July 1962" pits the steel-haired nursing supervisor against the "wet-behind-the-ears" intern on July 1 and should stand as a monument to those nurses who have helped generations of house staff. In "The Music and the Medicine" a gastroenterologist pipes his patients to sleep with simultaneous intravenous anesthetics and gentle harmonica music.

Although each of the vignettes in this volume are worthy of mention perhaps one of the most soul shattering is "A Medical Student On Call." A student spends weeks drawing blood and attending to a comatose, obese, brain tumor victim with many physically repulsive problems. Despite the patient's comatose state this student alone, acting from heartfelt emotions, and in the midst of rough and insulting handling by other house staff, talks to, strokes, and comforts the seemingly unresponsive coma victim. Weeks later, the patient emerges from the twilight and is able to recount nearly every word uttered in the room, every thoughtless and painful procedure imposed on her, and most importantly, the loving care of this lone student.

The litany of lessons learned from this volume is so simple, so straightforward, as to constitute aphorisms of the human condition: the nobility of the elderly; the brutal honesty of children; the inestimable value of the gentle human touch. The goal of the authors of producing "a testament to the redeeming qualities of the human heart, a record of needs and desires, of frailty, courage, frustration and hope" has clearly been met.

This book has already been adopted by my hospital department as required reading for fourth year students and residents in family medicine. I would highly recommend it for all members of the caring professions, and I can think of a few practitioners I have met along the road who should be locked in a room and made to read it.

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THE DOCTOR AS A PERSON

Edited by Jan Van Eys, MD, and John P. McGovern, MD. 151 pp, illustrated. Springfield, Ill, Charles C. Thomas, Publisher, 1988. $29.75 (hardcover)

There has been an exponential increase in basic scientific knowledge since World War II, particularly in the last 20 years. Physicians can now diagnose, understand, and rationally treat diseases that were previously "untreatable" or uniformly fatal. Yet in some ways this has been a mixed blessing.

What logic suggests should have increased the patient's respect and admiration for the physician has actually had the opposite effect. Where once there was relatively widespread agreement as to medicine's social and scientific utility, there is now a plethora of ethical and philosophical problems. *The Doctor as a Person* asks what changes in the behavior of the physician and the values of society might revitalize this relationship.

The essays in this book were first presented as papers at the University of Texas' M. D. Anderson Hospital and Tumor Institute, during the Eleventh Annual Mental Health Conference, May 22–23, 1986, Houston, Texas. The 12 contributors are all experts in their fields. Most are senior faculty members engaged in clinical research and in regular contact with patients and their families. They are thoughtful, scholarly individuals, ideally positioned to track the continual, sometimes wrenching changes in American health care. They agree, or seem to agree, that technology has contributed to a widespread perception of physicians as "super mechanics," technocrats who operate miraculous machines but who have lost sight of the uniqueness and dignity of their patients. To the degree this is true, it is tragic. Patient problems inevitably have two dimensions, emotional as well as organic. Science, history and experience all demonstrate that these are inseparable. The physician who concentrates on the technological aspects of disease and treatment must inevitably neglect the emotional aspects, and the patient-physician relationship suffers. *The Doctor as a Person* suggests that both parties suffer as human beings as well.

The authors use a coined term: "doctor-person." This means a "care-giver" who pays special attention to the emotional needs of the "care-receiver." An ideal doctor-patient relationship is described as one in which the physician truly cares and the patient perceives this caring. In this exchange, both gain important benefits, even if the patient is not cured. The authors identify a spectrum of role possibilities for the "doctor-person." These range from that of a secular holy man who forsakes all to minister to the sick, to a more pragmatic physician who consciously chooses to spend as much time caring for the emotional and spiritual needs of patients as seeing to their physical and scientific treatment.

The authors admit that many factors beyond technology have contributed to changing the physician-patient relationship. The litany is familiar: government intervention, third party payers, the business community which pays health insurance premiums, the corporate practice of medicine, consumerism, advertising by doctors, the growing priority of cost-containment, and the rationing of medical care. The book offers no consideration of other factors that would also seem to motivate the practice of impersonal, technology-based medicine, such as the debt medical graduates accumulate, obligations to spouses and children, the need for continuous learning, and perhaps other factors not yet apparent.

Efforts to help physicians be better "doctor-persons" are addressed. One essay inquires as to whether increasing the study of the humanities in the medical curriculum will lead to tangible changes in how patients are treated. It concludes there is more lip service than new and productive approaches.

On consideration, it seems the term "person" in "doctor-person" is probably best expressed by the Yiddish word, *mensch*. The word has a double meaning. A *mensch* is, in general, any human being. A *mensch* is, in particular, an individual of integrity and honesty and one who has a true love for all human beings—true love because it is unselfish, demanding no reward.

The Doctor As a Person is not casual reading. It is deep and stimulating, and requires frequent pauses to reflect on the issues discussed. This reviewer urges medical students, house officers, medical administrative personnel, nurses, and physicians to undertake this intense and rewarding effort. Many of us with MD degrees could greatly benefit ourselves and our patients by becoming better "doctor-persons."

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DATA ANALYSIS FOR CLINICAL MEDICINE: THE QUANTITATIVE APPROACH TO PATIENT CARE IN GASTROENTEROLOGY

Edited by Thomas C. Chalmers, MD. 217 pp, illustrated. Cheshire, England, International University Press, $49.00 (hardcover)

This unusual monograph addresses the application of quantitative methods to the practice of gastroenterology, including the contribution of clinicians as well as those from specialists in computers, epidemiology, and decision analysis. Edited by Thomas Chalmers, MD, its 13 chapters are uniformly well-written and well-referenced, and come with the added bonus of a 5¼″ floppy disk for use with IBM-compatible personal computers. Its contents are the result, in part, of a recent meeting, ROMA 88: The XIII International Congress of Gastroenterology and the VI European Congress of Digestive Endoscopy. Topics covered include biostatistics for the clinician, principles for using and interpreting epidemiologic data, clinical decision analysis, and clinical trials, to name a few. A series of case histories with a "gastrointestinal" theme is then presented, where the material covered in these chapters is put to use. Two additional chapters that would be of particular interest to physicians regardless of specialty include one on the use of computers in clinical practice, and one on access to the medical literature. The enclosed 5¼″ floppy disk contains several programs which would be of interest to both the novice and experienced personal computer user.

The chapter by Dr Chalmers, "Clinical Trials," is a nice overview of the faults of clinical trials and how to be aware of their existence, as one attempts to read through the many journals that our profession requires. A chapter on meta-analysis, the combining of results of several related studies, is quite well written and informative.

My only criticism of this book concerns the chapters in which data analysis is applied to clinical problem solving. These tend to be quite technical, are heavily laced with statistics, and result in tedious reading. This sets the stage for the nonstatistically oriented clinician to lose the message of the chapter. This is in part addressed by the extensive use of subtitles posed as "clinical questions" throughout these chapters. These same questions might well be asked by the physician confronted by the clinical scenarios that are presented.

This book should find its way into most medical libraries. It will be of value in teaching clinical decision making to medical students, perhaps as supplemental reading in a formal course. It should be required reading for all gastroenterology fellows, particularly those who are in programs active in clinical research. The chapters on access to medical literature and computers in clinical practice will be of interest to most physicians. Those with an IBM-compatible PC will enjoy the programs provided on the floppy disk.

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PHYSICIAN'S GUIDE TO INSULIN-DEPENDENT (TYPE I) DIABETES: DIAGNOSIS AND TREATMENT

By the American Diabetes Association, Inc. 150 pp, illustrated. Alexandria, Va, American Diabetes Association, 1988. $22.45 (spiralbound)

This volume is an excellent reference which provides the primary care physician with current, comprehensive, practical information on the diagnosis, management goals and complications of type I diabetes mellitus. It is the companion manual to the American Diabetes Association's (ADAs) *Physician's Guide to Non-Insulin Dependent* (*Type II*) *Diabetes: Diagnosis and Treatment* (reviewed below) which provides essential information regarding the care and management of patients with type I diabetes in an easy to read, high quality, well organized format. It represents the collaborative efforts of 13 contributing editors and ten diabetes consultants, all of whom are experts in the research and clinical aspects of insulin dependent diabetes mellitus.

The guide is organized into six main chapters which are tabbed for easy access. Each section is preceded by a highlight summary which lists essential information to be understood in the chapter. Emphasis is placed on the chronicity of type I diabetes mellitus and the life threatening, multi-organ complications that result from the disease. The focus of this manual is on routine management objectives and tools, ie, insulin treatment, monitoring, nutrition, and exercise. The chapter on special problems of diabetic ketoacidosis, hypoglycemia, pregnancy, and surgery in the diabetic patient is a welcome aid to assist the primary care physician in managing these clinically challenging situations.

The primary care physician will find this text a useful companion reference source to the type II manual also published by the American Diabetes Association.

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PHYSICIAN'S GUIDE TO NON-INSULIN-DEPENDENT (TYPE II) DIABETES: DIAGNOSIS AND TREATMENT

Second Edition. By the American Diabetes Association, Inc. 98 pp. Alexandria, Va, American Diabetes Association, 1988. $22.45 (spiralbound)

The intent of this handbook, a second edition and part of the American Diabetes Association Clinical Education Program, is to provide a key reference guide to all health professionals caring for individuals with non-insulin-dependent (type II) diabetes mellitus. It represents the collaborative efforts of 16 renowned research and clinical specialists in the field of diabetes from across the country. Nine contributing editors compiled the most current information on the pathogenesis, diagnosis, and treatment of type II diabetes with emphasis on the clinical application of this knowledge. The information was then reviewed and revised by seven additional diabetes consultants. This effort was led by the editor-in-chief, Dr Harold E. Levovitz, a much respected diabetologist well known for his clinical, research, and teaching expertise.

The book provides a concise yet comprehensive, practical, well-organized, up-to-date reference source for all health professionals treating patients with type II diabetes mellitus. It is a handy, easy to use, and indispensable guide for the primary care physician, as well as a useful addition to the body of medical literature.

The guide is organized into five main chapters which are tabbed for easy access. Each section is preceded by a "highlight" summary which lists the essential information to be understood in the chapter. Emphasis is placed on the role of blood glucose regulation, blood pressure control, and blood lipid normalization as important factors in modifying the chronic complications of diabetes mellitus. The text is easy to read, basic, yet comprehensive. The focus of the manual is on the evaluation and treatment of the patient from the "Diagnosis and Classification" (Chapter 1) to "Pathogenesis" and "Management" (Chapters 2, 3) and finally "Helping the Patient to Cope" (Chapter 4) through the "Detection and Treatment of Complications" (Chapter 5). The bibliography which follows each chapter is complete and up to date. The tables, 28 of them, provide accurate, basic information for the physician who provides well-managed care for the patient with non-insulin-dependent diabetes.

The primary care physician will find this text very useful in the everyday practice of medicine. Its high quality and well organized practical approach make it a must for all health professionals.

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SYNOPSIS OF CARDIAC PHYSICAL DIAGNOSIS

Edited by Jonathan Abrams, MD. 253 pp, illustrated. Philadelphia, Lea & Febiger, 1989. $18.50 (paperback)

In the preface to this book, Jonathan Abrams states, "There is a real danger that modern technology will again deemphasize the role of the stethoscope." To decrease the chances of this happening, Abrams has produced a very useful text illustrating how much information can be obtained by physical diagnosis. This may be particularly important at this time because of concerns about expensive, and perhaps unnecessary, tests which are frequently used in establishing a diagnosis. Dr Abrams correctly believes that the clinician and student with a good background in cardiac physical diagnosis will use the physical examination to its maximum before using more expensive tests. This book serves to bridge the gap between texts in physical diagnosis and clinical medicine. It can be used by medical students to further their knowledge of physical diagnosis and to show them how these physical signs are used in assessing various clinical conditions. The book can also be used by house officers who are more interested in specific clinical conditions, and need a review of the use of physical diagnosis as it applies to these conditions.

Abrams is the Chief of the Division of Cardiology at the University of New Mexico School of Medicine and is highly regarded as a teacher. His contact over the years with medical students and house officers enables him to appreciate the teaching material needed by each group at different levels of training. Several sections of the book seem to be designed for the student to use in responding to questions raised at bedside rounds. An example is seen in the section on mitral regurgitation, where one table lists clues to hemodynamically severe mitral regurgitation, while a separate paragraph lists the differential diagnosis of the systolic murmur of mitral regurgitation.

This text, which is entitled a "synopsis," is intended as a review for the reader who already has knowledge of the subject. The size of the book and its soft cover design suggest that it is best suited for the house officer to carry in the pocket for quick reference. The book is organized into two sections. The first part describes the basic approach to physical examination; this includes discussion of cardiac sounds and the use of the stethoscope. Other chapters review the arterial pulse, the jugular venous pulse, the precordial impulse, heart sounds, ejection sounds, and lastly, cardiac murmurs. The second section is more clinically oriented and discusses the major valvular lesions. In addition to these valvular lesions, there are separate chapters dealing with hypertrophic cardiomyopathy and mitral valve prolapse.

Each chapter in the second half of the text has the same organization. Each starts with a discussion of anatomy and pathophysiology, then discusses physical diagnosis, differential diagnosis, and then finally Doppler echocardiographic diagnoses. There is appropriate use of tables and illustrations. The illustrations of the extra heart sounds and the various types of ejection clicks are particularly useful.

Among the most novel and useful aspects of this text are the "practical points" mentioned by Abrams. These practical points are seen periodically in both sections of the book. They are very useful and set this book apart from other physical diagnosis textbooks. These points are presented as if the experienced professor were on rounds with the reader, giving him or her his little "pearls." The addition of the section on Doppler echocardiographic studies after each section on valvular lesions is useful. Abrams correctly makes the point that it is impossible adequately to evaluate valvular lesions without consideration of Doppler echocardiographic studies. He tries to show how the combination of the physical examination and the echocardiographic study can give an accurate estimate of the severity of many of the valvular lesions.

Abrams states that the purpose of his book is to "provide the clinician and student with a solid background in cardiac physical diagnosis so that the physical examination extracts the maximum diagnostic information before expensive and extensive tests are ordered." The text is well organized and adequately illustrated. I think the author has achieved his goal and produced a text that will be useful for medical students, house officers and practicing physicians.

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DIAGNOSIS AND TREATMENT OF MUSCLE PAIN

Edited by Hans Kraus, MD. 116 pp, illustrated. Chicago, Quintessence Publishing Co, Inc, 1988. $48.00 (softcover)

Dr Kraus has written a book that serves as a basic guide for the clinician in approaching the entity of muscle pain. The reader is left with a general understanding of the diagnostic categories of muscle pain, evaluation techniques and selective therapeutic regimes. Numerous years in academic medicine and clinical research have provided Kraus with a broad spectrum of experience which establishes his qualifications.

In a series of four concise chapters, muscle spasm, muscle tension, muscle deficiency, and trigger points are defined and some therapeutic approaches described. Chapter five presents a brief description of the nationally established YMCA's "The Y's Way to a Healthy Back," adapted from Dr Kraus' program. The succeeding chapters present methods of quantifying muscle pain and craniocervical mandibular disorders, followed by sections discussing the influence of metabolic disorders, the psychological components, and the neurological perspective of muscle pain.

The text is well organized, using basic medical terminology that facilitates easy reading. A blend of diagrams, tables, and case histories displays data clearly and efficiently. The use of contributing authors adds a flavor of the team approach to treatment. A shortcoming of the book is the lack of input from a physical therapist, since a high percentage of clients seen by these professionals are referred specifically for the treatment of muscle pain. The only reference to physical therapy is in the case histories where the approach mentioned was not effective. The use of cryotherapy, heat modalities, deep massage, pulsed ultrasound, and exercise, to name a few modalities, has been quite successful. As professionals educated about muscle pain and therapeutic exercise for years, physical therapists are valuable and resourceful team members.

One area highlighted in the text is Kraus' experience with ethyl chloride in conjunction with immediate controlled mobilization for the treatment of muscle spasm. His data reveal faster recovery, with decreased or no relapses of pain. Kraus hypothesizes that these responses occur from the interruption of the sensory motor chain with the use of ethyl chloride, which in turn blocks muscle spasm. He believes ethyl chloride is more effective than ice as an anesthetic. However, cost and availability of ethyl chloride should be considered, especially when home treatment programs are recommended. For clients tolerating ice, it can be effective and conveniently used with minimal cost to the client. Emphasis is placed on active exercise of the injured part, within the limits of pain, immediately following the application of ethyl chloride to make the treatment effective. This encourages the clinician to think about early mobilization when treating clients with muscle pain.

Another area of interest is the quantification of muscle pain which describes pressure threshold, tissue compliance (new method developed by the author), and thermography as methods of measurement. These objective tools, coupled with physical examination and observation, assist the clinician in accurate objective documentation. They are of prognostic value for the patient's course and allow for data collection in research evaluation. Chapter five is devoted to trigger points, which are common, and which are often unfamiliar to the clinician when treating muscle pain. The author advocates trigger point injection with procaine or lidocaine, followed by exercise, as the treatment regimen. Only brief mention is made of one other method (deep point massage), and the author states that it is rarely effective. This is indeed a controversial point and a broader discussion would have been helpful. The last area that warrants attention is the chapter on craniocervical mandibular disorders. It combines thoroughness and accuracy yet is presented in a concise fashion.

In conclusion, this book provides the clinician with an introductory approach to muscle pain diagnosis and selective treatment regimes. The references provide an additional source of information which should be explored for treatment alternatives and in-depth discussions on areas of interest.

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SURGERY FOR THE MORBIDLY OBESE PATIENT

Edited by Mervyn Deitel, MD. 400 pp, illustrated. Philadelphia, Pa, Lea & Febiger, 1989. $70.00 (hardcover)

In *Surgery for the Morbidly Obese Patient*, Dr Mervyn Deitel and his fellow authors have given us an extensive review of the field of bariatric surgery as it exists today. More than that, this text offers a superb overview of all aspects of the treatment of the morbidly obese patient and the present state of the art. I found the multiplicity of procedures which have arisen over the past ten years to be fascinating and quite succinct in their presentations, both written and pictorial. Obviously Dr Deitel and his authors are well qualified to cover a topic of this vast nature. I would find this to be a useful addition to the medical library of anyone who deals with the morbidly obese patient.

The volume is divided into three major sections: Basic principles of obesity, operations for morbid obesity, and associated care follow-up. The topics in each section give an excellent authoritative review as to the preoperative, operative, and postoperative care of the obese patient.

I was amazed at the ingenuity of the surgical mind regarding the variety of procedures which have progressed from the wiring of the jaw right up to the present-day vertical banding procedures. The most informative section for me dealt with associated care and follow-up of the morbidly obese patient. Obviously the topics in this section are given as a general overview and are covered in more detail in other sources, but I thought the broad nature of these sections was useful in dealing with this subject matter.

The most useful of all was the reference to the National Bariatric Surgery Registry. I believe participation is an absolute must for any surgeon working in this field.

Overall, I found the general organization and technical features of this book to be superb and easy to follow and understand. It obviously places this text as one of the musts and mainstays in the literature for anyone dealing with bariatric surgery. The drawings are excellent and the descriptions of the surgical procedures themselves could be quite useful in their detailed nature. The references are replete with works by both the new and old contributors in a field which has become quite extensive in dealing with the patient with morbid obesity.

I think one point to keep in mind in dealing with this subject matter is whether or not its future truly lies with surgery as was noted in the text. Practitioners in the field of medicine have become frustrated in dealing with these patients and the surgeon has taken over. Whether the future and the answer is in the surgical field remains to be seen, but at least the initiative has been taken to pursue this for future generations.

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FROM THE LIBRARY

MEDICAL LIBRARIES FOR THE SMALLER CENTRES

In the nearly five hundred incorporated villages in the State of New York and in all but seven of its cities there is not a medical reference and circulating library yet to be found. While other kinds of libraries are everywhere springing up, it is still the common practice for each physician and surgeon to purchase his own books, subscribe for his own periodicals, and consequently to limit his professional reading closely to these alone. Such a course necessitates useless duplication of professional works and periodicals in each community, and not only wastes hard-earned funds unduly, but results in each one's being very inadequately provided for, especially the younger men who have the greater share of time and strength with which to make use of a large collection. Moreover, by such a method, or lack of method, the more expensive and more useful books of reference are often not to be found at all in private libraries, or if found, are in possession of the older, more prosperous men, who are thus at once placed at a corresponding advantage over their younger or less prosperous neighbors. For, as it still too often appears, there is not yet everywhere in the ranks of the profession that community of even literary interest which, if present, would make the libraries of individuals freely accessible to all who might need or desire their use without incurring undue obligation or other disagreeable experience.

Affirming, then, that the whole present custom is uneconomical, interfering and unwise, and that it likewise results in great disadvantage, not only to the younger men and women in the profession, but even more so to all who would keep comprehensively abreast of medical progress, it is seen also to be irreparably disadvantageous to those who aspire to prepare papers and get ready for scientific discussion, and otherwise contribute to the profit and enthusiasm of the professional cult. Undoubtedly it is somewhat largely owing to the want of books and an early habit of using them, that we have so frequently to note such arrests of professional development as untimely dropping off of early enthusiasm, falling into everyday intellectual complacence, shiftlessness, routinism, and obvious scientific stagnation and inefficiency where and when enterprise is most needed; and more than all, the admitted inability rightly to cope with quackery and ignorance, under whatever guise these may seek to attract and capture public attention. In fact, so damaging and interfering is all this that the present method whereby isolated and small groups of hard-working and in every way nobler struggling practitioners are inadequately provided with the higher, broader means of intellectual stimulus and most useful information, should be recognized at once and by everybody as absolutely effete, and consequently should be superseded by modern reference and circulating libraries, which in every other kind of professional life have proved useful and stimulating.

But this is by no means all, for it is easily observable that not only is the better prosperity of the profession itself thus involved, but that of the community as well; especially in connection with sources of infection and contagion, and the means and methods required by improved sanitation and hygiene. I venture to say that every unrecognized case of small-pox within the past few years has been largely owing to the fact that the attending physician did not have any one of the several rather expensive books for reference, which, if timely used, might not only have helped him out and thus saved his professional reputation, but also might have saved the community from untold misery and loss. . . .

SMITH BAKER, MD
(*NY State J Med* 1908; 8:361–363)

Seizures are a potential side effect of DEET use

N,N-diethyl-m-toluamide (DEET), a topical repellent that blocks tick attachment, is an important weapon in the prevention of Lyme disease (caused by *Borrelia burgdorferi* through the tick vector *Ixodes dammini*). Between June and August 1989, there were five reports of generalized seizures temporally associated with the use of DEET in New York and Connecticut (*MMWR* 1989; 38:678–680).

Although in none of these cases has DEET toxicity been proven as the cause of the seizures, health officials have advised caution when using repellents that contain DEET. "The optimal concentration of DEET for prevention of tick bites is unknown. However, repellents containing 20%–30% DEET applied to clothing are approximately 90% effective in preventing tick attachment." Recommendations for safe use of DEET include the following: use repellent sparingly—one application will last 4–8 hours, and saturation does not increase efficacy; wear clothing with long sleeves and long pants and apply repellent to the clothing rather than to your skin; avoid inhaling or ingesting repellent and avoid contact with eyes; do not apply repellent to areas of children's hands that may come into contact with their eyes or mouth.

The five reported cases of seizures included four boys, aged three to seven years, and a 29-year-old man. All five patients reported few prodromal symptoms, recovered rapidly, had unremarkable medical histories, had had no previous seizures or neurologic events, and had normal nonfocal neurologic examinations following the seizures. "Each had had topical cutaneous exposure to varying concentrations of DEET . . . The interval between last use of DEET and onset of seizures ranged from 8 to 48 hours."

Previous reports of DEET toxicity have described symptoms such as behavioral changes, ataxia, encephalopathy, confusion, irritability, and insomnia, as well as seizures and/or coma following repeated cutaneous exposure to DEET.

Distribution of experimental AIDS drug DDI increases

Dideoxyinosine (DDI; Bristol-Myers Co, New York, NY) has shown promise in blocking replication of the AIDS virus and boosting the immune systems of AIDS patients. Although DDI has not yet been approved by the FDA for widespread marketing, the FDA and the National Institutes of Health (NIH) have recently allowed for increased distribution of DDI to AIDS patients not participating in supervised clinical trials (*Science News* 1989; 136:231).

The increasing need for effective anti-AIDS treatments, and mounting pressure from AIDS patients and activists to accelerate the availability of experimental therapies, have led to this unprecedented action. At present, azidothymidine (AZT) is the only drug approved for treating AIDS in the United States, and it is associated with a variety of adverse effects. DDI will now be available, through the manufacturer, for AIDS patients who cannot qualify for clinical trials of the drug, and who cannot tolerate or receive no benefit from AZT therapy.

DDI, too, has its side effects, including limb pain and pancreatic swelling, which appear to subside following cessation of the drug. "A daily dose of 500 to 750 milligrams appears effective and relatively nontoxic," according to one developer of the drug. AIDS patients receiving DDI will report regularly to their personal physicians, who will provide information on the drug's efficacy and toxicity to researchers conducting the clinical studies of DDI.

AZT now being distributed free to children with AIDS

On October 26, 1989, US Secretary of Health and Human Services, Dr Louis W. Sullivan, announced that the Food and Drug Administration (FDA) would allow the manufacturer of azidothymidine (AZT), the Burroughs Wellcome Company, to distribute the drug to children with AIDS. Until this announcement was made, AZT had not been approved by the FDA for use in children with AIDS except among those participating in clinical trials. Of the approximately 1,900 children in the US 12 years of age or younger who have been diagnosed with AIDS, 800 have died. Prior to the October 26, 1989, announcement only 200 to 300 were getting AZT in clinical trials. The remainder went untreated (*NY Times*, October 26, 1989, pp A1, A22).

Burroughs Wellcome, which had not applied for approval of AZT for children until a few weeks before Dr Sullivan's announcement, is now making the drug available free of charge to children with AIDS. The company had been under mounting pressure to apply for FDA approval for the drug's use in children from AIDS activists, government health officials, and members of Congress. More than a quarter of all children with AIDS reported to the Centers for Disease Control reside in New York State.

Risk of HIV transmission through blood transfusions is decreasing

In April 1985, the American Red Cross began screening donated blood for the human immunodeficiency virus (HIV). Between April 1985 and December 1988, the risk of infection with HIV from a transfusion of blood or blood components that tested antibody-negative decreased by more than 30% each year. Despite this encouraging finding, there still exists the risk of failing to detect HIV-infected blood products.

There is a period of time, commonly called the "window," between when a person is infected with HIV and when his or her blood will test positive for HIV antibodies. A major goal of the developers of antibody tests such as the enzyme-linked immunosorbent assay (ELISA) and the Western blot assay is to make this window smaller and smaller. Cumming and colleagues have determined this period to range from four to 14 weeks, with the average window being eight weeks or less (*N Engl J Med* 1989; 321:941–946). There is, however, still much uncer-

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NEWS BRIEFS are compiled and written by Vicki Glaser.

tainty concerning the length of the window.

Based on a window of four to 14 weeks, and analyzing data accumulated by the American Red Cross from April 1985 through December 1987, Cumming et al estimated the risk of HIV-infected donor blood passing undetected into the blood supply, and the risk of contracting HIV through a blood transfusion. "We estimate that donor-recruitment practices plus careful education and screening are eliminating 49 out of every 50 donors likely to be HIV-positive," the authors concluded, "and that testing is 92 to 97 percent effective, for a combined effectiveness of 99.9 percent."

Cumming et al estimated that the risk of human error resulting in the presence of HIV-positive blood products in the transfusion supply was less than 0.1%. Therefore, the main risk for HIV-infected blood to enter the blood supply is through the failure of the screening tests to detect HIV antibodies in infectious blood. "Estimating the chance that a donation will be made during this [window] period is an effective approach to defining the risk of HIV infection in transfusion recipients."

Using three window periods—short (4 wk), long (14 wk), and average (8 wk)—the authors estimated that 67, 227, and 131 HIV-positive blood units, respectively, entered the nation's blood supply undetected in 1987. This would lead to a risk of HIV infection from the transfusion of a single unit of 1:300,156, 1:88,267, and 1:153,123, respectively. The risk per transfusion, with each patient receiving an average of 5.4 units, would then be about 5.4 times the risk per single unit.

An analysis of the various donor groups revealed that first-time male donors were at greatest estimated risk of HIV infection, followed by untested repeat male donors, tested repeat male donors, first-time female donors, untested repeat female donors, and tested repeat female donors.

The authors credit education, donor recruitment efforts, and self-screening for eliminating 49 of 50 HIV-positive donors. To further ensure the safety of the blood supply, they recommend increased efforts to recruit and retain female donors, as well as continued and more frequent donations from previously tested donors.

FDA approves new schizophrenia drug

For persons with schizophrenia who do not respond to available antipsychotic medications, the US Food and Drug Administration (FDA) has approved a new drug that has produced marked improvement in nearly one third of a study population of formerly untreatable schizophrenics (*Science News* 1989; 136:245). The drug, clozapine (Sandoz Pharmaceutical Corp; East Hanover, NJ), is not a cure for schizophrenia, yet it appears to reduce the hallucinations, delusions, disordered thinking, social withdrawal and apathy associated with schizophrenia. Many of the participants in the study who responded to clozapine were able to leave the hospitals and move into community-based rehabilitation programs.

In addition to the risk of seizure, clozapine can produce agranulocytosis, a potentially fatal decrease in the number of white blood cells. Agranulocytosis occurs in about 1% of patients who receive clozapine. To screen for early signs of agranulocytosis, patients receiving clozapine must submit to weekly blood sampling, performed by the home health care company that delivers the drug.

Newly published anatomical atlas crowns a lifelong career in medical illustration

Medical illustrator Dr Frank H. Netter, perhaps best known for the *Ciba Collection of Medical Illustrations*, has completed a 576-page color atlas of the human anatomy (*NY Times*, September 19, 1989, p C3). The 514 color plates featured in the book include new illustrations as well as a compendium of his precise, life-like drawings that have appeared in previous volumes of human anatomy.

Dr Netter began his career as a commercial artist, and found good use for his skills in medical school, where he would often draw sketches of human anatomy to supplement his written class notes. Recognizing his innate ability for medical illustration, professors sought his drawing skills in preparing their scientific papers. The growing demand for his illustrations led him to give up his practice as a surgeon in the 1930s.

Since 1948, Dr Netter has focused on the 11-volume *Ciba Collection of Medical Illustrations*, with a new volume now in press, which depicts the full spectrum of human anatomy and embryology. Although human anatomy has not changed, advances in medical technology and novel methods of imaging and manipulating the human body have heightened the need to understand its details.

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Carleton William Bullard, MD, Sun City Center, Florida. Died June 16, 1989; age 91. Dr Bullard was a 1923 graduate of the State University of New York at Buffalo School of Medicine. He was a Diplomate of the American Board of Otolaryngology. His memberships included the American Academy of Otolaryngology, the American Academy of Facial, Plastic, and Reconstructive Surgery, the Pan American Association of Ophthalmology, the Pan American Medical Association, the Medical Society of the County of Cayuga, and the Medical Society of the State of New York.

Edwin H. Douglass, Jr, MD, Newburgh. Died August 31, 1989; age 84. Dr Douglass was a 1930 graduate of the New York University School of Medicine. He was a Fellow of the American College of Surgeons. He was a member of the Medical Society of the County of Orange and the Medical Society of the State of New York.

Leo Herman Elstein, MD, New York. Died August 5, 1989; age 75. Dr Elstein was a 1938 graduate of the New York University College of Medicine. His memberships included the New York Cardiological Society, the American Public Health Association, the American Society of Internal Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Laurence Bruce Felmus, MD, Scottsdale, Arizona. Died September 12, 1989; age 83. Dr Felmus was a 1931 graduate of Yale University School of Medicine. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Nemesio M. Francisco, MD, Oswego. Died August 24, 1989; age 54. Dr Francisco was a 1961 graduate of the Faculty of Medicine and Surgery University of Santo Tomas, Manila, Philippines. He was a member of the Medical Society of the County of Oswego and the Medical Society of the State of New York.

Harold Friedlander, MD, Miami, Florida. Died May 15, 1989; age 87. Dr Friedlander was a 1929 graduate of New York Medical College. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Jacob Savin Garber, MD, Forest Hills. Died August 18, 1989; age 85. Dr Garber was a 1929 graduate of the University of Maryland School of Medicine. He was a member of the American Academy of Family Physicians, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Peter F. Gaston, MD, Forest Hills. Died September 25, 1989; age 75. Dr Gaston was a 1938 graduate of the Orvosi Fakultas Tudomanyegyetem, Budapest, Hungary. He was a Diplomate of the American Board of Psychiatry and Neurology. He was a member of the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Fred William Geib, MD, Hilton Head, South Carolina. Died September 1, 1989; age 86. Dr Geib was a 1928 graduate of the Johns Hopkins University School of Medicine. He was a Diplomate of the American Board of Surgery. He was a member of the New York Academy of Medicine, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

A. Lilian A. Gilbert, MD, New York. Died September 10, 1989; age 85. Dr Gilbert was a 1930 graduate of the Johns Hopkins University School of Medicine. She was a Diplomate of the American Board of Pediatrics. Her memberships included the New York Academy of Medicine, the American Thoracic Society, the New York County Medical Society, and the Medical Society of the State of New York.

Stephen W. Giorlando, MD, Howard Beach. Died September 19, 1989; age 69. Dr Giorlando was a 1943 graduate of the State University of New York Health Science Center at Brooklyn. He was a Fellow of the American College of Obstetricians and Gynecologists and a Qualified Fellow of the International College of Surgeons. He was a member of the American Geriatrics Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

William Howard Godsick, MD, Roslyn. Died March 3, 1989; age 89. Dr Godsick was a 1923 graduate of the New York University School of Medicine. He was a Diplomate of the American Board of Obstetrics and Gynecology, a Fellow of the American College of Obstetricians and Gynecologists, and a Qualified Fellow of the International College of Surgeons. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Edward N. Goldstein, MD, Cranburg, New Jersey. Died July 28, 1989; age 79. Dr Goldstein was a 1936 graduate of the New York University School of Medicine. He was a Diplomate of the American Board of Otolaryngology and a Qualified Fellow of the International College of Surgeons. He was a member of the American Academy of Otolaryngology, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Herman S. Goodman, MD, Bradenton, Florida. Died September 12, 1989; age 88. Dr Goodman was a 1926 graduate of the New York Medical College. His memberships included the American Psychiatric Association, the American Academy of Neurology, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Royal Marwin Howard, MD, Staten Island. Died September 3, 1989; age 67. Dr Howard was a 1945 graduate of the New York University School of Medicine. He was a Diplomate of the American Board of Ophthalmology and a Fellow of the American College of Surgeons. His memberships included the New York Ophthalmology Society, the American Academy of Ophthalmology and Otolaryngology, the Richmond County Medical Society, and the Medical Society of the State of New York.

Erwin Jessel, MD, Floral Park. Died September 7, 1989; age 82. Dr Jessel was a 1931 graduate of the Friedrich-Wilhelms Universitaet Medizinische Fakultaet, Berlin, Germany. He was a Diplomate of the American Board of Ophthalmology. His memberships included the New York Society for Clinical Ophthalmology, the American Academy of Ophthalmology and Otolaryngology, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Eugene Howard Kagan, MD, Syracuse. Died July 5, 1989; age 60. Dr Kagan was a 1956 graduate of the Medizinische Fakultaet der Universitaet Zurich, Zurich, Switzerland. He was a Diplomate of the American Board of Pathology and a Fellow of the American College of Pathologists. He was a member of the American Society of Clinical Pathologists, the Onondaga County Medical Society, and the Medical Society of the State of New York.

Robert L. Klupt, MD, Southbury, Connecticut. Died November 22, 1988; age 85. Dr Klupt was a 1925 graduate of Columbia University College of Physicians and Surgeons. He was a Fellow of the American College of Chest Physicians. He was a member of the New York Cardiological Society, the New York County Medical Society, and the Medical Society of the State of New York.

Harold Koretz MD, Avoca. Died August 9, 1989; age 79. Dr Koretz was a 1934 graduate of Wayne State University School of Medicine, Detroit. He was a Diplomate of the American Board of Internal Medicine. He was a member of the Steuben County Medical Society and the Medical Society of the State of New York.

Joseph Korn, MD, Brooklyn. Died August 25, 1989; age 83. Dr Korn was a 1930 graduate of the New York Medical College. He was a Diplomate of the American Board of Family Practice and a Fellow of the American Academy of Family Physicians. His memberships included the New York Cardiological Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Harold William Lovell, MD, Nokomis, Florida. Died May 13, 1989; age 85. Dr Lovell was a 1929 graduate of the University of Michigan Medical School, Ann Arbor. He was a Diplomate of the American Board of Psychiatry and Neurology and a Fellow of the American College of Physicians. His memberships included the New York Academy of Medicine, the American Academy of Neurology, the American Psychiatric Association, the American Geriatrics Society, the New York County Medical Society, and the Medical Society of the State of New York.

David L. MacDonell, MD, Ocean Beach. Died September 4, 1989; age 65. Dr MacDonell was a 1949 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the American Academy of Family Physicians, the Suffolk County Medical Society, and the Medical Society of the State of New York.

William Francis Mitty, Jr, MD, New York. Died August 9, 1989; age 67. Dr Mitty was a 1946 graduate of the New York University School of Medicine. He was a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons and the American College of Gastroenterology. His memberships included the New York Academy of Medicine, the New York State Surgical Society, the New York Gastroenterological Society, the American Geriatrics Society, the American Trauma Society, the American Gastro-Enterological Association, the New York County Medical Society, and the Medical Society of the State of New York.

James P. Riordan, MD, Orchard Park. Died September 11, 1989; age 79. Dr Riordan was a 1937 graduate of Georgetown University School of Medicine. He was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Asa Paul Ruskin, MD, Brooklyn. Died September 26, 1989; age 60. Dr Ruskin was a 1957 graduate of the Universite de Paris VI, Paris, France. He was a Diplomate of the American Board of Physical Medical and Rehabilitation and a Fellow of the American College of Physicians. His memberships included the New York Academy of Medicine, the New York Society for Physical Medicine and Rehabilitation, the American Academy of Neurology, the American Geriatrics Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Gordon E. Shull, MD, Elmira. Died August 31, 1989; age 71. Dr Shull was a 1944 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the American Academy of Family Physicians, the Medical Society of the County of Chemung, and the Medical Society of the State of New York.

Franklin H. Westcott, MD, San Diego, California. Died July 19, 1989; age 87. Dr Westcott was a 1927 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a Fellow of the American College of Physicians. His memberships included the New York Academy of Medicine, the Pan American Medical Association, the American Academy of Allergy, the American Academy of Compensation Medicine, Inc, the New York County Medical Society, and the Medical Society of the State of New York.

Joel Vaughan Woodruff, MD, Albany. Died September 18, 1989; age 46. Dr Woodruff was a 1969 graduate of the Philadelphia College of Osteopathic Medicine. He was a Diplomate of the American Board of Psychiatry and Neurology. He was a member of the American Neurological Association, the Medical Society of the County of Albany, and the Medical Society of the State of New York.

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INDEX

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ISSUE	NUMBER	PAGES
January	1	1-54
February	2	55-128
March	3	129-194
April	4	195-250
May	5	251-312
June	6	313-380
July	7	381-442
August	8	443-502
September	9	503-552
October	10	553-600
November	11	601-648
December	12	649-712

CLASSIFICATION CODES

CC = Clinical Conference
CPC = Clinicopathologic Conference
CR = Case Report
ED = Editorial Commentary
EP = Essay in Pediatrics
ER = Essay Review
HIST = History of Medicine
LE = Letter to the Editor
LEN = Leads from *Epidemiology Notes*
NF = News Feature

SUBJECT INDEX

Abortion
- The wickedest woman in New York (ER), 296

Abuse
- **(see Human abuse, Child abuse, Elder abuse, Sexual abuse, Spouse abuse)**

Acaricides
- Seizures are a potential side effect of DEET use (NF), 695

Acne
- Isotretinoin—an effective yet teratogenic drug (NF), 374
- Water-soluble vitamin A proves less toxic (NF), 375

Acquired immunodeficiency syndrome
- AIDS among New York State children (LEN), 188
- AIDS educational material may increase rather than reduce fear of AIDS victims (NF), 309
- AIDS in New York State: An update (LEN), 589
- AZT now being distributed free to children with AIDS (NF), 695
- Battling AIDS through behavior modification (NF), 108
- Current challenges of AIDS in New York City, 517
- Designated care programs for patients with AIDS and HIV related illnesses in designated care centers (LEN), 542
- Distribution of experimental AIDS drug DDI increases (NF), 695
- FDA expands testing of anti-AIDS drugs (NF), 548
- HIV partner/contact notification assistance programs (LEN), 636
- Human immunodeficiency virus infection in a cohort of intravenous drug users in New York City: Demographic, behavioral, and clinical features, 506
- Pressure from AIDS patients and a growing market lead to AZT price reduction (NF), 644
- Risk of HIV transmission through blood transfusions is decreasing (NF), 695
- Survey assesses AIDS on college campuses (NF), 438

Acute myocardial infarction
- Strategies for preventing thromboembolism in patients with acute myocardial infarction (ED), 381

Adolescent health
- Abuse of the adolescent, 156

Adult day care
- Adult day care centers for elderly patients (LE), 96

AIDS
- **(see Acquired immunodeficiency syndrome)**

Air travelers
- Illness among commercial air travelers is more common on the ground (NF), 308

Alcohol
- Alcohol abuse: Opportunities for prevention (LEN), 588
- Alcohol and public health: Implications for New York State (LEN), 490
- Public health perspective on alcohol abuse (LEN), 540

Alternative health care delivery systems
- Increasing physician participation in alternative delivery systems (NF), 109

Alzheimer's disease
- Alzheimer's disease and other dementias registry (LEN), 298

American Dental Association
- American Dental Association urges dentists to become more aware of child abuse (NF), 191

American Medical Association
- Unpublished research data may obscure important findings (NF), 438

Amoxicillin/clavulanate potassium
- Cholestatic hepatitis after therapy with amoxicillin/clavulanate potassium (CR), 355

Anemia
- High hopes for new anemia drug (NF), 499

Animal experimentation
- Animal experimentation for medical research (committee report), 671

Anorexia nervosa
- Anorexia nervosa: The destructive pursuit of thinness, 166

Arteriovenous fistula
- Renal arteriovenous fistula in the setting of subacute bacterial endocarditis (CR), 530

Arteriovenous malformations
- Determination of cerebral blood flow in an arteriovenous malformation using SPECT scanning (CR), 627

Arthritis
- Infectious arthritides (LEN), 300

Associated Medical Schools of New York
- Education of physicians and the health of New York: Associated Medical Schools of New York position paper on the mission and responsibilities of medical schools and academic health centers in New York State, 462

Asthma
- Asthma in Timisoara: Diary of a glimpse at the Romanian medical system, 283

Atherosclerosis
- Menopause is associated with an increased risk of atherosclerosis (NF), 308

Azidothymidine
- AZT now being distributed free to children with AIDS (NF), 695
- Pressure from AIDS patients and a growing market lead to AZT price reduction (NF), 644

Babesiosis
- Selected tickborne infections: A review of Lyme disease, Rocky Mountain spotted fever, and babesiosis, 320
- Ticks, Lyme disease, and spotted fever (ED), 313

Bacteremia
- High tech yields new treatment for bacterial infections (NF), 644
- Meningitis following bacteremia with *Streptococcus sanguis* (CR), 625

Bacterial infections
- High tech yields new treatment for bacterial infections (NF), 644

Bicycle helmets
- Bicycle helmets greatly reduce risk of head and brain injury suffered in accidents (NF), 498

Bile duct obstruction
- Common duct stone with associated *Clonorchis sinensis* infection (CR), 480

Birth outcomes
- Birth outcomes in New York State (LEN), 687
- Birth outcomes for infants of drug abusing mothers, 256

Bladder
- Dose-response relationship demonstrated between cigarette smoking and histologic changes in the urinary bladder (NF), 597
- Vesical calculus, lithotrity, and Napoleon III (HIST), 472

Blindness
Transient cortical blindness following an acute hypotensive event in the postpartum period (CR), 682
Blood transfusions
Risk of HIV transmission through blood transfusions is decreasing (NF), 695
Transfusion reactions (CC), 264
Books received, annotations of, 246, 373
Books reviewed
AIDS: A Guide for the Primary Physician (Holmes and Motulsky, eds.), 494
Assuring Quality Outpatient Care for Children. Guidelines and a Management System (Cook and Heidt), 303
Atlas of CO_2 Laser Surgical Techniques (Lanzafame and Hinshaw, eds.), 436
Atlas of Surgical Operations (Zollinger, Jr. and Zollinger), 436
Beginning Child Psychiatry (Adams and Fras), 302
Caring for the Disabled Elderly (Rivlin and Wiener), 370
Child Sexual Abuse: A Handbook for Health Care and Legal Professionals (Schetky and Green), 184
Chinese Medicine: Its History, Philosophy & Practice, & Why It May One Day Dominate the Medicine of the West (Porkert and Ullmann), 545
Clinical Imperatives in Surgery and Orthopaedics (Domisse and Lindeque, eds.), 435
Clinical Problems, Injuries and Complications of Gynecologic Surgery (Nichols, ed.), 436
Colour Atlas of Dermatology (Bhutani), 495
Colour Atlas of Sexually Transmitted Diseases (Bhutani), 546
Common Skin Disorders: A Physician's Illustrated Manual with Patient Instruction Sheets (Epstein), 305
Comprehensive Review in Toxicology (Bryson), 594
Critical Care Obstetrics (Clark, Phelan, and Cotton), 242
Current Trends in Urology (Resnick, ed.), 592
Data Analysis for Clinical Medicine: The Quantitative Approach to Patient Care in Gastroenterology (Chalmers, ed.), 691
Diagnosis and Management of Renal Disease and Hypertension (Mandal and Jennette), 304
Diagnosis and Treatment of Muscle Pain (Kraus, ed.), 693
Diseases of the Colon, Rectum, and Anal Canal (Kirsner and Shorter, eds.), 495
Doctor as a Person, The (Eys and McGovern, eds.), 690
Doctors. The Biography of Medicine (Nuland), 371
Dr Eden's Healthy Kids: The Essential Diet, Exercise, and Nutrition Program (Eden), 641
Drug Interactions Index (Weibert and Norcross), 546
Essential Book of Traditional Chinese Medicine, The. Volume I: Theory (Yanchi), 593
Essential Book of Traditional Chinese Medicine, The. Volume II: Clinical Practice (Yanchi), 593
Exercise and Sport Sciences Reviews (Pandolf, ed.), 496
General Surgery: Review and Assessment (Stillman), 434
Health Services Administration: Education and Practice (Lorentzen and Roemer), 306
Hernia Repair Without Disability (Lichtenstein), 243
Heterosexual Transmission of AIDS in Africa, The (Koch-Weser and Vanderschmidt, eds.), 642
History of Medicine (Greene, ed.), 643
If You Can't Remember Please Don't Forget (Deas), 364
Infective Disease in Primary Care: A Symptomatic Approach (Hull), 243
Intensive Care Medicine (Rippe, Irwin, Alpert, and Dalen, eds.), 105
Interventional Radiology (Castaneda-Zuniga and Tadavarthy, eds.), 544
Judging Medicine (Annas), 427
Life-Sustaining Treatment: Making Decisions and Appointing a Health Care Agent, 97
Linear Growth Retardation in Less Developed Countries (Waterlow), 303
Liver Tumors: Multidisciplinary Management (Hodgson, ed.), 496
Long Term Health Care: Providing a Spectrum of Services to the Aged (Brickner, Lechich, Lipsman, and Scharer), 105
Management of the Frail Elderly by the Health Care Team (Walsh, Tsukuda, and Miller), 369
Manual of Clinical Oncology (Sherman, Calman, Eckhardt, Elsebai, Firat, Hossfeld, Paunier, and Salvadori, eds.), 244
M.D.: Doctors Talk About Themselves (Pekkanen), 487
Medical Ethics and the Elderly: A Case Book (Waymack and Taler), 369
Medical Toxicology. Diagnosis and Treatment of Human Poisoning (Ellenhorn and Barceloux), 304
Moodswing (Fieve), 640
Occupational Back Pain; Spine: State of the Art Reviews (Deyo), 106
Oculoplastic, Orbital and Reconstructive Surgery (Hornblass, ed.), 435
Otolaryngology. Head and Neck Surgery: Principles and Concepts (Templer and Davis, eds.), 242
Pediatric Head Injuries: A Handbook (Amacher), 592
Perfecting the World: The Life and Times of Dr. Thomas Hodgkin, 1798–1866 (Kass and Kass), 497
Physician's Guide to Insulin-Dependent (Type I) Diabetes: Diagnosis and Treatment (The American Diabetes Association, Inc.), 691
Physician's Guide to Non-Insulin-Dependent (Type II) Diabetes: Diagnosis and Treatment (The American Diabetes Association, Inc.), 692
Piece of My Mind, A. America's Doctors Share Their Most Dramatic, Inspiring, and Moving Experiences (Dan and Young, eds.), 690
Post-Traumatic Therapy and Victims of Violence (Ochberg, ed.), 495
Preventing Emergency Malpractice (Fish and Ehrhardt), 643
Principles of Surgical Technique. The Art of Surgery (Wind and Rich), 242
Psychiatric Treatment of Alzheimer's Disease, 371
Psychiatry and Mental Health Science Handbook (Thomas and Lindenthal, eds.), 594
Rationing Medicine (Blank), 371
Rationing of Medical Care for the Critically Ill (Strosberg, Fein, and Carroll, eds.), 595
Rehabilitation of Athletic Injuries: An Atlas of Therapeutic Exercise (Torg, Vesgo, and Torg), 106
Remember Me. A Collection of Poems (Anderson), 244
Renewing the Promise: Medicare and Its Reform (Blumenthal, Schlesinger, and Drumheller, eds.), 544
Research and the Ageing Population (Ciba Foundation), 370
Resources in Medical History. On Insanity and Its Classification (Chiarugi), 245
Responsible Physician, The: Standards of Excellence and the Critically Ill Patient (Wolf, Jr., Hamolsky, Kutscher, and Muraszko, eds.), 595
Self in the System, The: Expanding the Limits of Family Therapy (Nichols), 107
Selling or Buying a Medical Practice (Shaub), 544
Social Learning and Systems Approaches to Marriage and the Family (Peters and McMahon), 306
Solving Common Pediatric Problems: An Algorithm Approach (Margolis), 303
Sometimes God Has a Kid's Face (Ritter), 182
Surgery for the Morbidly Obese Patient (Deitel, ed.), 693
Surgical Infectious Diseases (Howard and Simmons, eds.), 434
Surrogate Parenting: Analysis and Recommendations for Public Policy, 97
Synopsis of Cardiac Physical Diagnosis (Abrams, ed.), 692
Textbook of Diagnostic Medicine (Samiy, ed.), 107
Transcendence of Loss Over the Life Span (Weenolsen), 594
Transplantation in New York State: The Procurement and Distribution of Organs, 97
Trauma (Mattox, Moore, and Feliciano, eds.), 592
Treatment of Psychiatric Disorders, The (Reid), 641
Understanding the Alcoholic's Mind: The Nature of Craving and How to Control It (Ludwig), 494
Unlocking the Family Door: A Systemic Approach to the Understanding and Treatment of Anorexia Nervosa (Stierlin and Weber), 640
When Doctors Get Sick (Mandell and Spiro, eds.), 487
When Living Hurts. Directives for Treating Depression (Yapko), 305
Wickedest Woman in New York, The: Madame Restell, the Abortionist (Browder), 296
Worse Than the Disease: Pitfalls of Medical Progress (Dutton), 547
Breast cancer
Needle localization of occult breast cancer and subsequent management (LE), 361
Occult breast cancer in two community hospitals in New York State, 612
Promising new diagnostic tool for early detection of breast cancer (NF), 644
Role of community hospitals in the detection of occult breast cancer (ED), 602

Breast reconstruction
- Chest wall deformity following expansion of irradiated soft tissue for breast reconstruction (CR), 419

Burn injuries
- Burn injuries and deaths (LEN), 299

Campylobacter fetus
- *Campylobacter fetus* as a pulmonary pathogen in man: Case report of post-pneumonectomy empyema (CR), 90

Cancer
- Cancer control intervention using available data bases (LEN), 104
- Cancer incidence among Native Americans in New York State (LEN), 541
- Cancer incidence in five continents (LEN), 432
- Changing trends in malignant melanoma in New York State (LEN), 239
- Childhood central nervous system tumors (LEN), 638
- Cigarette smoking and passive smoking linked to cervical cancer (NF), 308
- Clinical value of fine needle aspiration biopsy in a patient with medullary thyroid carcinoma and pseudofollicular carcinoma (CR), 527
- Detecting malignant lesions with skin surface microscopy (NF), 499
- Development of multiple carcinomas in a long term survivor of Wegener's granulomatosis treated with immunosuppressive drugs (CR), 680
- Malignant lymphoma presenting with severe hypercalcemia (CR), 533
- Malignant melanoma of the anal canal (CR), 291
- Needle localization of occult breast cancer and subsequent management (LE), 361
- New findings confirm early-warning sign of cancer relapse (NF), 438
- Occult breast cancer in two community hospitals in New York State, 612
- Prophylactic dentistry can help reduce oral complications of cancer therapy (NF), 548
- Role of community hospitals in the detection of occult breast cancer (ED), 602
- Sarcoidosis of the thyroid clinically mimicking malignancy (CR), 578
- Sloan-Kettering targets women in new ad campaign (NF), 108
- Testicular malignancy associated with massive inguinal adenopathy and life threatening hemorrhage (CR), 482

Cardiac arrest
- Resuscitation of cardiac arrest victims by advanced life support units in Monroe County, New York, 652

Cardiopulmonary resuscitation
- Resuscitation of cardiac arrest victims by advanced life support units in Monroe County, New York, 652

Carotid body tumors
- Pulsed Doppler-ultrasonic diagnosis of carotid body tumor (CR), 580

Carpal tunnel syndrome
- Frequency of work-related carpal tunnel syndrome may be underestimated (NF), 598

Cataracts
- Laser may improve early detection of cataracts (NF), 375

Cause of death
- Clinical causes of death of elderly patients in a long term health care facility (LE), 538
- Mortality patterns among a Native American population in New York State, 557

Central nervous system tumors
- Childhood central nervous system tumors (LEN), 638

Cervical cancer
- Cigarette smoking and passive smoking linked to cervical cancer (NF), 308

Cesarean births
- Cesarean childbirth in New York State (LEN), 186

Child abuse
- Abuse of the adolescent, 156
- Abuse of the child and anxiety in the adult, 138
- American Dental Association urges dentists to become more aware of child abuse (NF), 191
- Child abuse: The physician's responsibility, 152
- Child abuse: Where are we going? (ED), 133
- Child sexual abuse (ER), 184
- Clinical picture of child abuse is changing (NF), 190
- Confronting child abuse and neglect in New York State, 163
- Fetal neglect and abuse, 148
- Future effects of abuse differ from child to child (NF), 191
- Health care practitioners face malpractice charges for failure to report child abuse (NF), 190
- Ipecac poisoning: Know the symptoms that suggest child abuse (NF), 190
- Need for physicians to recognize sexual abuse in children (ED), 131
- Physician's management of child maltreatment (committee report), 168
- Public disclosure of private information about victims of abuse (NF), 191
- Secondary syphilis in a prepubertal child: Differentiating condylomata lata from condylomata acuminata (CR), 180
- Sexual abuse of boys (ED), 132
- Training pediatric residents to recognize and manage child abuse (NF), 191
- "While we have time, let us do good" (ER), 182

Children's health
- Acute renal failure in children: Etiology, diagnosis, and management (EP), 336
- AIDS among New York State children (LEN), 188
- AZT now being distributed free to children with AIDS (NF), 695
- Birth outcomes for infants of drug abusing mothers, 256
- Birth outcomes in New York State (LEN), 687
- Child abuse: Where are we going? (ED), 133
- Childhood central nervous system tumors (LEN), 638
- Children and sports: What's healthy, what's not (NF), 548
- Confronting child abuse and neglect in New York State, 163
- Crisis in New York City's perinatal services, The, 658
- Demolishing the lead danger (ED), 651
- Drug use in pregnancy (ED), 255
- Factors affecting the health care of physicians' children (NF), 309
- Lead poisoning during home renovation, 679
- Management of respiratory failure in infants and children (EP), 664
- Need for physicians to recognize sexual abuse in children (ED), 131
- Preschool eye screening gets a boost (NF), 247
- Secondary syphilis in a prepubertal child: Differentiating condylomata lata from condylomata acuminata (CR), 180
- Sexual abuse of boys (ED), 132
- Student participation in mixed gender sports competition, 526

Cholesterol
- GM-CSF demonstrates ability to lower serum cholesterol levels (NF), 109

Cigarettes
- (see Smoking)

Clinical Conferences (series)
- Transfusion reactions, 264

Clonorchis sinensis
- Common duct stone with associated *Clonorchis sinensis* infection (CR), 480

Cocaine
- Cocaine (ED), 384
- Cocaine: General characteristics, abuse, and addiction, 390
- Nonfatal pulmonary edema following cocaine smoking (CR), 415

Commentaries
- (see Editorial Commentaries)

Commissioner of Health of New York State
- MSSNY House of Delegates calls for the removal of David Axelrod, MD, as Commissioner of Health of New York State (NF), 376

Congenital splenic cysts
- Congenital splenic cysts presenting after blunt abdominal trauma (CR), 289

Congestive heart failure
- Hospital costs, resource characteristics, and the dynamics of death for patients with a primary diagnosis of congestive heart failure, 60

Conjunctivitis
- Primary meningococcal conjunctivitis (LE), 634

Contact lenses
- FDA issues warning to contact lens wearers (NF), 247
- Misuse of disposable contact lenses increases risk of corneal infections (NF), 549

Contact notification
- HIV partner/contact notification assistance programs (LEN), 636

Coronary care
- Coronary care in New York City hospitals, 199

Crohn's disease
- Metastatic Crohn's disease (NF), 597

Cyclosporine
- Cyclosporine shows promise against dry eye (NF), 247

DEET
- Seizures are a potential side effect of DEET use (NF), 695

Defibrillators
- Implantable defibrillators undergoing clinical tests (NF), 248

Dementias
- Alzheimer's disease and other dementias registry (LEN), 298

Dentistry
- Prophylactic dentistry can help reduce oral complications of cancer therapy (NF), 548

Diabetes
- New test identifies persons at risk for diabetes (NF), 498

Diarrhea
- Peptic ulcer disease presenting as life-threatening diarrhea following subtotal colectomy (CR), 231

Dideoxycytidine
- FDA expands testing of anti-AIDS drugs (NF), 548

Dideoxyinosine
- Distribution of experimental AIDS drug DDI increases (NF), 695
- FDA expands testing of anti-AIDS drugs (NF), 548

Dolichoectasia
- Treatment of dolichoectasia of the middle cerebral artery with bypass and exclusion (CR), 629

Do-not-resuscitate orders
- Do-not-resuscitate law, The (LE), 425
- New York State's do-not-resuscitate law (LE), 424
- Presumption of consent in New York State's do-not-resuscitate law, 69
- Reflections on the New York do-not-resuscitate law (ED), 57

Drug abuse
- Birth outcomes for infants of drug abusing mothers, 256
- Cocaine (ED), 384
- Cocaine: General characteristics, abuse, and addiction, 390
- Drug use in pregnancy (ED), 255
- Nonfatal pulmonary edema following cocaine smoking (CR), 415

Editorial Commentaries
- Abdominal tuberculosis: A disease poised for reappearance, 252
- Acute renal failure, 314
- Challenge of change, The, 55
- Child abuse: Where are we going?, 133
- Cocaine, 384
- Continuing competence in the medical profession, 443
- Current challenges to the practice of medicine, 553
- Demolishing the lead danger, 651
- Drug use in pregnancy, 255
- Elimination of the requirement for medical libraries in hospitals, 601
- Enigma of extrapulmonary tuberculosis, The, 251
- Health problems associated with urban poverty: A narrowing gap between the Third and First Worlds, 649
- Human abuse through the ages, 129
- Idiopathic retroperitoneal fibrosis—past and present, 503
- Impact of technology on the clinical decision making of house staff, 444
- Mandatory Medicare assignment, 195
- Medical malpractice mediation panel, The, 554
- Modes of administration of heparin in clinical medicine, 382
- Need for physicians to recognize sexual abuse in children, 131
- Perestroika, new mental health legislation, and Soviet psychiatry, 135
- Reflections on the New York do-not-resuscitate law, 57
- Role of community hospitals in the detection of occult breast cancer, 602
- Rural health care challenge in New York State, The, 603
- Sexual abuse of boys, 132
- Smoking and health: 25 years of progress, 1
- Smoking in the workplace: Research needs and potential applications, 2
- Strategies for preventing thromboembolism in patients with acute myocardial infarction, 381
- Tale of Ondine: A curse, a kiss, a clasp, and a comment, 196
- Thyroid needle biopsy: The primary procedure, 504
- Ticks, Lyme disease, and spotted fever, 313
- Typhlitis, 58

Elder abuse
- Abuse of the elderly (LE), 484
- Abuse of the elderly: Misuses of power, 159

Elderly
- Abuse of the elderly (LE), 484
- Abuse of the elderly: Misuses of power, 159
- Adult day care centers for elderly patients (LE), 96
- Clinical causes of death of elderly patients in a long term health care facility (LE), 538

Emergency medical services
- Information statement of the Emergency Medical Services Committee of the MSSNY on conflicts between physicians and the pre-hospital system (NF), 377
- Resuscitation of cardiac arrest victims by advanced life support units in Monroe County, New York, 652

Emergency room
- Emergency room visit time: Changes over a 16-year period, 446

Employees' health
- Evaluating worksite smoking policies: Methodologic issues, 5
- Health effects of involuntary smoking in the workplace, 27
- Implementing nonsmoking policies in the private sector and assessing their effects, 11
- Smoking in the workplace: Research needs and potential applications (ED), 2

Empyema
- Late onset post-pneumonectomy empyema (CR), 582

Endocarditis
- Renal arteriovenous fistula in the setting of subacute bacterial endocarditis (CR), 530

Endodermal sinus tumors
- Endodermal sinus tumor and benign cystic teratoma of the ovary occurring simultaneously (CR), 233

Enterococcal meningitis
- Enterococcal meningitis: A subacute presentation (CR), 92

Environmental Protection Agency
- EPA sets Medical Waste Tracking Act in motion (NF), 375

Epidemiology
- (see **Leads from *Epidemiology Notes* (series)**)

Epoetin alfa
- High hopes for new anemia drug (NF), 499

Errata, 53, 96, 486

Escherichia coli
- Hemolytic uremic syndrome and verotoxin producing *E coli* (LEN), 637

Esophageal dilation
- Meningitis following bacteremia with *Streptococcus sanguis* (CR), 625

Esophageal obstructions
- Heimlich maneuver successful for clearing esophageal obstruction (NF), 439
- Little known hazard of drinking beer (NF), 549

Essay Reviews
- Body and soul: Doctors tell all, 487
- Child sexual abuse, 184
- Family portraits from a family practice, 364
- Judging medicine, 427
- Life and the law, 97
- "While we have time, let us do good", 182
- Wickedest woman in New York, The, 296

Essays in Pediatrics (series)
- Acute renal failure in children: Etiology, diagnosis, and management, 336
- Management of respiratory failure in infants and children, 664

Eye disorders
- Cyclosporine shows promise against dry eye (NF), 247
- Laser may improve early detection of cataracts (NF), 375
- Misuse of disposable contact lenses increases risk of corneal infections (NF), 549
- Preschool eye screening gets a boost (NF), 247
- Primary meningococcal conjunctivitis (LE), 634

Facsimile machines
- FAX machines: Standard medical equipment? (NF), 598

Federation of State Medical Boards of the United States
- Position statement of The Federation of State Medical Boards of the United States on the assessment of licensed physicians, 226

Fetal rights
- Fetal neglect and abuse, 148
- Fetal therapy and surgery: Fetal rights versus maternal obligations (committee report), 80

Food and Drug Administration
- FDA approves new schizophrenia drug (NF), 696
- FDA expands testing of anti-AIDS drugs (NF), 548
- FDA investigations sting pharmaceutical companies (NF), 644

Foodborne illness
- Foodborne illness in New York State 1980–1987 (LEN), 366

Gallbladder
- Common duct stone with associated *Clonorchis sinensis* infection (CR), 480

Gastrojejunal ulcer
- Perforation of a gastrojejunal ulcer into the pericardium (CR), 358

Gynecology
- Trends in American gynecology, 1800–1910: A short history (HIST), 278

Haemophilus influenzae
- *Haemophilus influenzae* type b prevention (LEN), 430

Health care costs
- Hospital costs, resource characteristics, and the dynamics of death for patients with a primary diagnosis of congestive heart failure, 60

Health care professionals
  Health care practitioners face malpractice charges for failure to report child abuse (NF), 190
  Torture and health care professionals, 143
  Training pediatric residents to recognize and manage child abuse (NF), 191
Health care system
  New York State's health care system: Selected major problems and recommended solutions (special report), 606
Health insurance
  Confidential New York State Department of Health Universal New York Health Care (UNY*Care) proposal: Medical Society of the State of New York staff observations, 572
Health maintenance organizations
  Increasing physician participation in alternative delivery systems (NF), 109
Heimlich maneuver
  Heimlich maneuver successful for clearing esophageal obstruction (NF), 439
Hemolytic uremic syndrome
  Hemolytic uremic syndrome and verotoxin producing *E coli* (LEN), 637
Hemorrhagic esophageal varices
  Scrotal ischemia after intravenous vasopressin therapy for hemorrhagic esophageal varices (CR), 583
Heparin
  Full dose subcutaneous heparin therapy, 387
  Modes of administration of heparin in clinical medicine (ED), 382
  Strategies for preventing thromboembolism in patients with acute myocardial infarction (ED), 381
Hepatic failure
  Sustained-release nicotinic acid carries increased risk of hepatic failure (NF), 597
Hepatitis
  Cholestatic hepatitis after therapy with amoxicillin/clavulanate potassium (CR), 355
  Prevention of perinatal transmission of hepatitis B (LEN), 187
  Prevention of perinatal transmission of hepatitis B virus: Prenatal screening of all pregnant women for hepatitis B surface antigen (committee report), 352
Hernia
  Paraesophageal hernia, 654
History of Medicine (series)
  Dietary changes and the decline of scurvy and tuberculosis in 19th century Europe, 621
  History of Kashin-Beck disease, 343, (HIST), 486
  History of the *New York Medical Journal*, 403
  History of the Public Health Association of New York City, 467
  Role of physicians in America's sterilization movement, 1894–1925, 169
  Short history of the quack's materia medica, A, 520
  Trends in American gynecology, 1800–1910: A short history, 278
  Vesical calculus, lithotrity, and Napoleon III, 472
HIV
  (see **Human immunodeficiency virus**)
Home-infusion companies
  Home-infusion company flourishes with doctors' capital (NF), 499
Hormone therapy
  Consensus on postmenopausal hormone therapy (NF), 108
Hospital Association of New York State
  Position statement of the Hospital Association of New York State on the Report of the New York State Advisory Committee on Physician Recredentialing, 223
Hospitals
  Coronary care in New York City hospitals, 199
  Elimination of the requirement for medical libraries in hospitals (ED), 601
  Emergency room visit time: Changes over a 16-year period, 446
  Occult breast cancer in two community hospitals in New York State, 612
  One hospital's experience with a total ban on smoking (NF), 598
  Role of community hospitals in the detection of occult breast cancer (ED), 602
  Smoke-free hospital: A smoke-free worksite, 38
House staff
  (see Medical education, Internship, Residency)
Human abuse
  Abuse of the adolescent, 156
  Abuse of the child and anxiety in the adult, 138
  Abuse of the elderly (LE), 484
  Abuse of the elderly: Misuses of power, 159
  American Dental Association urges dentists to become more aware of child abuse (NF), 191
  Anorexia nervosa: The destructive pursuit of thinness, 166
  Child abuse: The physician's responsibility, 152
  Child abuse: Where are we going? (ED), 133
  Child sexual abuse (ER), 184
  Clinical picture of child abuse is changing (NF), 190
  Confronting child abuse and neglect in New York State, 163
  Fetal neglect and abuse, 148
  Future effects of abuse differ from child to child (NF), 191
  Health care practitioners face malpractice charges for failure to report child abuse (NF), 190
  Human abuse through the ages (ED), 129
  Ipecac poisoning: Know the symptoms that suggest child abuse (NF), 190
  Need for physicians to recognize sexual abuse in children (ED), 131
  Perestroika, new mental health legislation, and Soviet psychiatry (ED), 135
  Physician's management of child maltreatment (committee report), 168
  Public disclosure of private information about victims of abuse (NF), 191
  Secondary syphilis in a prepubertal child: Differentiating condylomata lata from condylomata acuminata (CR), 180
  Sexual abuse of boys (ED), 132
  Spouse abuse, 141
  Torture and health care professionals, 143
  Training pediatric residents to recognize and manage child abuse (NF), 191
  "While we have time, let us do good" (ER), 182
Human immunodeficiency virus
  (see also Acquired immunodeficiency syndrome)
  Designated care programs for patients with AIDS and HIV related illnesses in designated care centers (LEN), 542
  HIV partner/contact notification assistance programs (LEN), 636
  Human immunodeficiency virus infection in a cohort of intravenous drug users in New York City: Demographic, behavioral, and clinical features, 506
  Pressure from AIDS patients and a growing market lead to AZT price reduction (NF), 644
  Risk of HIV transmission through blood transfusions is decreasing (NF), 695
Hypercalcemia
  Malignant lymphoma presenting with severe hypercalcemia (CR), 533
Hypercholesterolemia
  Sustained-release nicotinic acid carries increased risk of hepatic failure (NF), 597
Hyperprolactinemia
  Diagnosis of stress-related hyperprolactinemia: Evaluation of the hyperprolactinemia rest test, 205
Hysterectomies
  Hysterectomies in New York State: 1984–1986 (LEN), 102
Immunization
  Two-dose measles immunization recommendations (LEN), 492
Implants
  Growing controversy on safety of implants (NF), 549
Impotence
  Vascular insufficiency: A significant factor in impotence, 614
Infectious arthritides
  Infectious arthritides (LEN), 300
Injuries
  Bicycle helmets greatly reduce risk of head and brain injury suffered in accidents (NF), 498
  Burn injuries and deaths (LEN), 299
  Lyme disease imitating a sports injury (NF), 498
  Wood burning related injuries, 262
International health
  Asthma in Timisoara: Diary of a glimpse at the Romanian medical system, 283
  Health and health care in Nepal, 272
  Health problems associated with urban poverty: A narrowing gap between the Third and First Worlds (ED), 649
  History of Kashin-Beck disease (HIST), 343, 486
Internship
  House staff stress, 454
  House staff stress and the impact of the new regulations on postgraduate medical education (LE), 684
  Impact of the new code regulations on postgraduate medical education in New York State, 457
Interstitial fibrosis
  Pulmonary hypertrophic osteoarthropathy as the initial manifestation of interstitial fibrosis (CR), 234
Intestinal perforation
  Temporal arteritis and intestinal perforation: Report of three cases (CR), 476

Intravenous drug users
  Human immunodeficiency virus infection in a cohort of intravenous drug users in New York City: Demographic, behavioral, and clinical features, 506

Isotretinoin
  Isotretinoin—an effective yet teratogenic drug (NF), 374

Jugular venous ectasia
  Ultrasound diagnosis of jugular venous ectasia (CR), 532

Kashin-Beck disease
  History of Kashin-Beck disease (HIST), 343, 486
  Kashin-Beck disease (LE), 585

Lead poisoning
  Demolishing the lead danger (ED), 651
  Lead poisoning during home renovation, 679

Leads from *Epidemiology Notes* (series)
  AIDS among New York State children, 188
  AIDS in New York State: An update, 589
  Alcohol abuse: Opportunities for prevention, 588
  Alcohol and public health: Implications for New York State, 490
  Alzheimer's disease and other dementias registry, 298
  Birth outcomes in New York State, 687
  Burn injuries and deaths, 299
  Cancer control intervention using available data bases, 104
  Cancer incidence among Native Americans in New York State, 541
  Cancer incidence in five continents, 432
  Cesarean childbirth in New York State, 186
  Changing trends in malignant melanoma in New York State, 239
  Childhood central nervous system tumors, 638
  Designated care programs for patients with AIDS and HIV related illnesses in designated care centers, 542
  Documentation of maternal mortality, 238
  Foodborne illness in New York State 1980–1987, 366
  Haemophilus influenzae type b prevention, 430
  Hemolytic uremic syndrome and verotoxin producing *E coli*, 637
  HIV partner/contact notification assistance programs, 636
  Hysterectomies in New York State: 1984–1986, 102
  Infectious arthritides, 300
  Lyme disease surveillance in New York State, 686
  Management of head lice infestations, 103
  NYS rabies summary 1988, 431
  Prevention of perinatal transmission of hepatitis B, 187
  Public health perspective on alcohol abuse, 540
  Two-dose measles immunization recommendations, 492

Leiomyoma
  Leiomyomatous tumors: Presentation of a giant gastric leiomyoma and a review of the literature (CR), 416

Letters to the Editor
  Aberman, Ben W., MD, 236
  Abuse of the elderly, 484
  Academic boycott of South Africa, The, 685
  Adult day care centers for elderly patients, 96
  Buck v Bell, 536
  Buttocks blow barotrauma and pneumomediastinum, 361
  Caring and serving, 635
  Clinical causes of death of elderly patients in a long term health care facility, 538
  Complications arising from the use of ophthalmologic medications in an intensive care unit patient, 537
  Confidential Tay-Sachs carrier screening, 585
  Doctors' penmanship, 237
  Do-not-resuscitate law, The, 425
  Empty lifeboat, 95
  Future of medicine, The, 684
  Future of medicine, The: A resident's perspective, 484
  Himself, Mrs Plucker, and the rites of spring, 484
  House staff stress and the impact of the new regulations on postgraduate medical education, 684
  Kashin-Beck disease, 585
  Legislator-physician relationships, 294
  Medical malpractice, 586
  Needle localization of occult breast cancer and subsequent management, 361
  New York Medical Journal and cigarette smoking, 294
  New York State's do-not-resuscitate law, 424
  Omental infarction in pregnancy, 634
  Omphalocele at 15 weeks, 424
  Paget-Schroetter syndrome in a patient on the contraceptive pill, 293
  Penile prosthesis as an adjunct to intermittent self-catheterization in patients with neuropathic bladders, 363
  Physician recredentialing, 484, (LE), 536
  Poor legal representation in a malpractice lawsuit, 237
  Primary meningococcal conjunctivitis, 634
  Professional medical conduct, 293
  Psychiatric teams and the psychiatrist's authority, 236
  Temporary insanity and premenstrual syndrome, 94
  Thallium stress tests and the White House security system, 634
  Tombstone erected on grave of Arthur Donaldson Smith, MD, 685
  Typhlitis, 426
  Universal precautions, 362

Lice
  Management of head lice infestations (LEN), 103

Lithotrity
  Vesical calculus, lithotrity, and Napoleon III (HIST), 472

Living wills
  Living will, The: Does it protect the rights of the terminally ill?, 72

Lyme disease
  Hunt for a vaccine against Lyme disease (NF), 374
  Lyme disease: A review of its clinical features, 566
  Lyme disease imitating a sports injury (NF), 498
  Lyme disease surveillance in New York State (LEN), 686
  Seizures are a potential side effect of DEET use (NF), 695
  Selected tickborne infections: A review of Lyme disease, Rocky Mountain spotted fever, and babesiosis, 320
  Ticks, Lyme disease, and spotted fever (ED), 313

Lymphoma
  Malignant lymphoma presenting with severe hypercalcemia (CR), 533

Malignant lymphoma
  Malignant lymphoma presenting with severe hypercalcemia (CR), 533

Malpractice
  Health care practitioners face malpractice charges for failure to report child abuse (NF), 190
  Medical malpractice (LE), 586
  Medical malpractice mediation panel, The (ED), 554
  Poor legal representation in a malpractice lawsuit (LE), 237
  Prima facie tort rule: A possible medical-legal answer to the medical malpractice crisis, 399
  What's ahead for malpractice premiums? (NF), 438
  When physicians lose their malpractice insurance: What are the options? (NF), 645

Measles
  Two-dose measles immunization recommendations (LEN), 492

Medicaid
  Plan to reform Medicaid system is on the table (NF), 247

Medical care delivery
  Coronary care in New York City hospitals, 199
  Variations in the delivery of medical care in Central New York, 63

Medical education
  Awarding of honors to medical clerks: Results of a national survey, 562
  Education of physicians and the health of New York: Associated Medical Schools of New York position paper on the mission and responsibilities of medical schools and academic health centers in New York State, 462
  House staff stress, 454
  House staff stress and the impact of the new regulations on postgraduate medical education (LE), 684
  Impact of technology on the clinical decision making of house staff (ED), 444
  Impact of the new code regulations on postgraduate medical education in New York State, 457
  JAMA's education issue highlights trends (NF), 645
  New York initiative in medical education, A, 618
  Quality education is essential for quality health care, 213

Medical illustration
Newly published anatomical atlas crowns a lifelong career in medical illustration (NF), 696
Medical libraries
Elimination of the requirement for medical libraries in hospitals (ED), 601
Medical research
Animal experimentation for medical research (committee report), 671
Unpublished research data may obscure important findings (NF), 438
Medical Society of the State of New York
Animal experimentation for medical research (committee report), 671
Caring and serving (LE), 635
Confidential New York State Department of Health Universal New York Health Care (UNY*Care) proposal: Medical Society of the State of New York staff observations, 572
Fetal therapy and surgery: Fetal rights versus maternal obligations (committee report), 80
Information statement of the Emergency Medical Services Committee of the MSSNY on conflicts between physicians and the pre-hospital system (NF), 377
Mandatory Medicare assignment (ED), 195
MSSNY House of Delegates calls for the removal of David Axelrod, MD, as Commissioner of Health of New York State (NF), 376
New York State's health care system: Selected major problems and recommended solutions (special report), 606
Physician's management of child maltreatment (committee report), 168
Position paper of the Medical Society of the State of New York on physician recredentialing, 222
Prevention of perinatal transmission of hepatitis B virus: Prenatal screening of all pregnant women for hepatitis B surface antigen (committee report), 352
Robert D. Brandstetter, MD, awarded William Hammond Citation (NF), 377
Student participation in mixed gender sports competition, 526
William Sharpe, MD, and Leonard Jan Bruce-Chwatt, MD, receive Medical Society's Redway Award (NF), 377
Medical Society of the State of New York, House of Delegates
Board of Trustees 1988–1989, 116
Convention Committees, 122
Councilors 1988–1989, 116
House Committee on Bylaws, 124
1989 House of Delegates Members, 118
Nominating Committee, 123
Officers 1988–1989, 115
Reference Committees, 122
Schedule of sessions, 113
Medical Waste Tracking Act
EPA sets Medical Waste Tracking Act in motion (NF), 375
Medicare
Mandatory Medicare assignment (ED), 195
Medicine
Challenge of change, The (ED), 55
Continuing competence in the medical profession (ED), 443
Current challenges to the practice of medicine (ED), 553
Empty lifeboat (LE), 95
Future of medicine, The (LE), 684
Future of medicine, The: A resident's perspective (LE), 484
Legislator-physician relationships (LE), 294
New York State's health care system: Selected major problems and recommended solutions (special report), 606
Professional medical conduct (LE), 293
Quality education is essential for quality health care, 213
Medicolegal issues
Buck v Bell (LE), 536
Fetal neglect and abuse, 148
Fetal therapy and surgery: Fetal rights versus maternal obligations (committee report), 80
Judging medicine (ER), 427
Life and the law (ER), 97
Living will, The: Does it protect the rights of the terminally ill?, 72
Medical malpractice mediation panel, The (ED), 554
Presumption of consent in New York State's do-not-resuscitate law, 69
Prima facie tort rule: A possible medical-legal answer to the medical malpractice crisis, 399
Proposal concerning products liability, 396
Reflections on the New York do-not-resuscitate law (ED), 57
Temporary insanity and premenstrual syndrome (LE), 94
Medullary thyroid carcinoma
Clinical value of fine needle aspiration biopsy in a patient with medullary thyroid carcinoma and pseudofollicular carcinoma (CR), 527
Melanoma
Changing trends in malignant melanoma in New York State (LEN), 239
Detecting malignant lesions with skin surface microscopy (NF), 499
Malignant melanoma of the anal canal (CR), 291
Meningitis
Enterococcal meningitis: A subacute presentation (CR), 92
Meningitis following bacteremia with *Streptococcus sanguis* (CR), 625
Menopause
Menopause is associated with an increased risk of atherosclerosis (NF), 308
Merkel cell carcinoma
Gallium citrate Ga 67 scintigraphy in recurrent Merkel cell tumor, Use of (CR), 356
Minnesota
Nonsmoking/smoking policy implementation at the state level, 34
Miscellaneous
Aberman, Ben W., MD (LE), 236
Asthma in Timisoara: Diary of a glimpse at the Romanian medical system, 283
Caring and serving (LE), 635
Himself, Mrs Plucker, and the rites of spring, 85, (LE), 484
Legislator-physician relationships (LE), 294
91-year-old daughter's memoir of her physician father, Dr Vacil D. Bozovsky (1865–1933), 576
Mitral valve replacement
Pulmonic valve replacement for pulmonic insufficiency one year after mitral valve replacement (CR), 422
Mortality patterns
Clinical causes of death of elderly patients in a long term health care facility (LE), 538
Mortality patterns among a Native American population in New York State, 557
Napoleon III
Vesical calculus, lithotrity, and Napoleon III (HIST), 472
Native Americans
Cancer incidence among Native Americans in New York State (LEN), 541
Mortality patterns among a Native American population in New York State, 557
Needle biopsy
Clinical value of fine needle aspiration biopsy in a patient with medullary thyroid carcinoma and pseudofollicular carcinoma (CR), 527
Thyroid needle biopsy: The primary procedure (ED), 504
Neisseria meningitidis
Primary meningococcal conjunctivitis (LE), 634
Nepal
Health and health care in Nepal, 272
Netter, Frank H., MD
Newly published anatomical atlas crowns a lifelong career in medical illustration (NF), 696
New York City
Coronary care in New York City hospitals, 199
Crisis in New York City's perinatal services, The, 658
Current challenges of AIDS in New York City, 517
History of the Public Health Association of New York City (HIST), 467
Human immunodeficiency virus infection in a cohort of intravenous drug users in New York City: Demographic, behavioral, and clinical features, 506
New York Medical Journal
History of the *New York Medical Journal* (HIST), 403
New York Medical Journal and cigarette smoking (LE), 294
New York State
AIDS among New York State children (LEN), 188
AIDS in New York State: An update (LEN), 589
Alcohol and public health: Implications for New York State (LEN), 490
Birth outcomes in New York State (LEN), 687
Cancer incidence among Native Americans in New York State (LEN), 541
Cesarean childbirth in New York State (LEN), 186
Changing trends in malignant melanoma in New York State (LEN), 239
Confidential New York State Department of Health Universal New York Health Care (UNY*Care) proposal: Medical Society of the State of New York staff observations, 572
Confronting child abuse and neglect in New York State, 163
Education of physicians and the health of New York: Associated Medical Schools of New York position paper on the mission and responsibilities of medical schools and academic health centers in New York State, 462

Foodborne illness in New York State 1980–1987 (LEN), 366
Hysterectomies in New York State: 1984–1986 (LEN), 102
Impact of the new code regulations on postgraduate medical education in New York State, 457
Lyme disease surveillance in New York State (LEN), 686
Mortality patterns among a Native American population in New York State, 557
MSSNY House of Delegates calls for the removal of David Axelrod, MD, as Commissioner of Health of New York State (NF), 376
New York initiative in medical education, A, 618
New York State's do-not-resuscitate law (LE), 424
New York State's health care system: Selected major problems and recommended solutions (special report), 606
NYS rabies summary 1988 (LEN), 431
Occult breast cancer in two community hospitals in New York State, 612
Periodic physician recredentialing proposed in New York State, 209
Presumption of consent in New York State's do-not-resuscitate law, 69
Reflections on the New York do-not-resuscitate law (ED), 57
Resuscitation of cardiac arrest victims by advanced life support units in Monroe County, New York, 652
Rural health care challenge in New York State, The (ED), 603
Variations in the delivery of medical care in Central New York, 63

New York State Journal of Medicine
Our referees, 100
Robert D. Brandstetter, MD, awarded William Hammond Citation (NF), 377
William Sharpe, MD, and Leonard Jan Bruce-Chwatt, MD, receive Medical Society's Redway Award (NF), 377

Nicotinic acid
Sustained-release nicotinic acid carries increased risk of hepatic failure (NF), 597

Nutrition
Dietary changes and the decline of scurvy and tuberculosis in 19th century Europe (HIST), 621

Obituaries
Abrams, A. Leonard, 599
Addonizio, Joseph Carl, 646
Adler, Daniel K., 440
Agrest, Francis A., 550
Ajello, Dominick A., 378
Alexis, Pierre, 310
Ambinder, Nathan, 110
Arkun, Sureyya Naci, 110
Aronson, William, 249
Banas, Charles Felix, 52
Baron, Louis, 52
Barron, Leonard J., 550
Beckman, E. Mark, 646
Beckwith, Irving Harris, 500
Beery, Edwin Newman, 440
Bennett, William Louis, 378
Bettmann, Ernest H., 52
Birnbaum, John Hans, 500
Birnbaum, Morris, 599
Black, Hugh D., 550
Block, Marvin Avram, 378
Blumberg, Arnold G., 550
Boller, Raymond Jerome, 310
Bongiovanni, John Richard, 249
Bonheim, H. Hermann, 646
Bratt, Murray Almon, 310
Brody, Joseph, 500
Brothers, Milton J., 249
Bruce-Chwatt, Leonard Jan, 502
Buckbee, Harold Frederick, 599
Budoff, Benjamin H., 599
Bullard, Carleton William, 697
Bumble, Samuel Herbert, 550
Burke, Addison J., 110
Butera, Daniel Gerard, 310
Cally, Joseph R., 52
Campbell, Coburn A. L., 378
Cancellieri, Remo, 550
Capello, Anthony Thomas, 192
Capozzi, Giovanni, 599
Caputo, Isadore A., 192
Carr, Roland Benedict, 599
Carrington, William Thomas, 500
Carroll, Hubert Francis, 646
Cavaliere, Joseph T., 192
Chorba, William Gaston, 599
Chu, Dong Sun, 249
Cimildoro, Umbert, 310
Claman, Leoni Neumann, 310
Clark, Paul Chester, 440
Cohn, Anni Gerda, 310
Coles, Robert Sohn, 249
Commins, Saul, 110
Conway, William Hynes, 378
Craft, Robert Johnson, 500
Cramer, Irving, 646
Cucchiarella, Armando H., 550
Curran, Frank J., 378
Cutino, Rodolph M., 646
Davis, Hugh Peter, 52
Davis, James Llewellyn, 378
De Bold, Charles, 500
Deboni, Guerrino, 52
Decker, Albert, 192
Del Terzo, J. Robert, 110
Demeter, Emerick, 249
Demuth, Edwin Leopold, 440
DeRook, Frans, 378
Diana, Michael Angelo, 646
DiCapua, Edmund B., 378
Dick, Frank Alexander, 249
DiFrancesco, Armand L., 192
Donoso, Ephraim, 110
D'Orsogna, Edward I., 192
Douglass, Edwin H., Jr., 697
Drabkin, Irving, 110
Dreifuss, Richard L., 110
El Hassan, Nabil D., 550
Eller, Joseph Jordan, 310
Ellis, Harold Lloyd, 440
Elstein, Leo Herman, 697
Engster, Henry Charles, 310
Epps, Gerald Jay, 550
Fahnrich, Leo, 550
Faust, Otto Alois, 440
Felmus, Laurence Bruce, 697
Ferrara, Angelo C., 52, 441
Fisher, Charles, 110
Fisher, Joseph, 500
Fogel, Dorothy, 110
Forster, Philip L., 440
Francisco, Nemesio M., 697
Frank, Samuel Bergman, 500
Freed, Sidney, 500
Friedlander, Harold, 697
Friedman, Abraham H., 550
Galyon, Charles Amos, 110
Gannon, Walter James, 378
Garber, Jacob Savin, 697
Gaston, Peter F., 697
Gaston, Sawnie Renard, 550
Gates, Edwin Wilder, 310
Geib, Fred William, 697
Gerard, H. Charles, 550
Gerber, Isadore Earle, 192
Germain, Alton Anthony, 52
Gersten, David, 110
Gertner, Joseph, 52
Gilbert, A. Lilian A., 697
Gilbert, Louis Jay, 646
Gilbert, Michael L., 249
Gingell, Barry David, 550
Giorlando, Stephen W., 697
Glantz, William M., 440
Glasier, H. Pelham, 110
Glassman, Charles, 550
Godsick, William Howard, 697
Goldenberg, Leon N., 249
Goldman, Meyer Leo, 646
Goldstein, Edward N., 697
Goldstein, Jacob Warren, 249
Golub, Leo, 52
Goodman, Herman S., 697
Goolsby, Flem, Jr., 646
Gray, Rebecca Elisabeth, 192
Green, Gordon Lemont, 192
Greenstein, Louis, 550
Greenwood, M. Allen, 249
Groeschel, August Herman, 500
Grunfeld, Arnold, 440
Gyarmati, Ernest E., 500
Hamilton, George Clifford, 310
Hansch, Paul Allen, 249
Harel, Aron, 500
Harris, Hilbert L., 192
Harris, Raymond, 646
Harrison, David Abraham, 192
Held, Edward Calisch, 52
Herfort, Alan, 378
Hermann, Harold Bernard, 110
Hicks, Hugh Mason, 599
Hillenbrand, James Jacob, 378
Hirsch, Nathan, 378
Hoffman, Abraham Charles, 378
Holcomb, Frederic W., Jr., 646
Honig, Alexander Sandor, 249
Hotchkiss, Robert Sherman, 646
Howard, Royal Marwin, 697
Imperato, Alfred A., 440
Imperato, Louis T., 440
Ipolyi, Francis Xavier, 310
Irvine, Jed Hotchkiss, 599
Itzkowitz, Louis E., 551
Jackson, Lee, 646
Jackson, Mildred M. Wicker, 599
Jaen, Jose E., 647
Jaklitsch, Frank H., 378
Janelli, Donald E., 379
Jessel, Erwin, 698
Johnson, Roy T., 52
Jones, Ralph, 192
Kadar, Laszlo, 250
Kagan, Eugene Howard, 698
Kahan, Leon, 551
Kaplan, Seymour Robert, 192
Kardons, Frances, 551
Kaufman, Harry H., 250
King, S. Edward, 310
Kingsley, Harry Durwood, 192
Klasson, David Howard, 379
Kleefield, Elmer A., 310
Kleinman, Alexander, 440
Klepetar, Charles, 551
Klupt, Robert L., 698

Koenig, Hedwig, 500
Kohn, Robert W., 193
Konterwitz, Harry, 500
Koretz, Harold, 698
Korn, Joseph, 698
Koweity, Steven E., 379
Kravitz, Jacob, 379
Krikun, Ludmila, 110
Kron, Auguste, 250
Kuschner, Isaak, 500
Laitner, Wolf, 379
Lange, Kurt, 647
Langs, Harold M., 311
Lanzetta, Louis A., 111
Larson, Swen L., 551
Leisten, David, 250
Leopold, Harold Sello, 379
Leve, Lloyd H., 250
Levinson, Robert Daniel, 250
Levy, Samuel Kantor, 551
Lew, Abraham, 111
Liebermann, Joel, 440
Linder, David Osias, 250
Linder, Leon, 52
Lipkin, Mack, 441
Liu, John Peter, 647
Livoti, Charles Balthasar, 441
Loizeaux, Leon Samuel, Jr., 111
Lovell, Harold William, 698
Lubowe, Irwin I., 250
MacDonell, David L., 698
Mackta, Stanley, 311
Maglio, Gerard C., 379
Malejka, Joseph Anthony, 111
Malgieri, John Anthony, 501
Maligaya, Norma Lontok, 311
Mannix, Arthur J., Jr., 441
Mantell, Cecil, 311
Margaretten, Frederick M., 551
Margolius, Bernard R., 311
Mascaro, Joseph Raymond, 379
Matturri, Joseph Mario, 193
May, Harold C., Jr., 111
McDonough, James H., 599
McGarry, Thomas, 193
McHugh, Hugh, 500
Mead, De Witt Clinton, 501
Merk, Harvey John, 647
Milgram, Joseph E., 311
Miller, Melvyn Robert, 52
Mitty, William Francis, Jr., 698
Moore, Frank Guy, 111
Murray, Alice E. Bennett, 647
Napolitano, Michael Joseph, 250
Nataloni, Marino, 311
Nauman, Max, 311
Neichin, Sidney N., 111
Nygaard, Kaare Kristian, 501
O'Connell, Cornelius J., 111
Ollenstein, Raphael W., 501
Oppel, Theodore Wright, 501
Papastrat, Constantine J., 111
Papatestas, Angelos, 501
Parillo, Philip, 193
Penner, Abraham, 250
Peress, Isaac Y., 52
Perillo, Peter Andian, 379
Perl, Gizella, 379
Persse, John David, 111
Pineas, Herman O., 379
Poole, Arthur R., 551
Poster, Henry, 501
Pratt, Sylvester H., 551
Pugliese, John, 250
Puglisi, Bernard S., 647
Quinlan, Edward A., Jr., 111
Raisman, Victor H., 551
Rauch, S. Jerome, 52
Reckford, Leo Paul, 53
Regan, William F., Jr., 53
Reich, Arthur, 551
Reich, Carl, 53
Reissig, Arthur J., 193
Reynolds, Francis X., 311
Riordan, James P., 698
Rochman, Norman Davis, 250
Rock, Julius, 501
Rodgers, John Morris, 647
Romanelli, Anthony R., 379
Roncal, Pilar H., 501
Roose, Lawrence Jacob, 501
Rosenfeld, Eugene David, 111
Rosenstein, Irving, 379
Rosenthal, Harold Carl, 311
Rothstein, David, 501
Rubinstein, Henry Steven, 193
Ruskin, Asa Paul, 698
Sabey, Peter K., 193
Sallick, Myron A., 441
Sauer, John J., 193
Saward, Ernest Welton, 551
Schaefer, Walter, 311
Scheider, Francis G., 551
Schindler, Richard D., 379
Schultz, Daniel Perry, 111
Schwartz, Bertram S., 111
Schwippert, Harry, 551
Scorza, Stephen B., 379
Segal, Isidore, 647
Sharma, Mohinder Kumar, 379
Sheridan, Wallace M., 193
Shull, Gordon E., 698
Silberer, Rosine M., 441
Slepian, Solomon, 647
Slovak, Michael, 551
Sokolowicz, Zundel, 501
Sorgi, Salvatore F., 250
Sowka, Hans J., 501
Spitz, L. Jackson, 250
Sprenger, George L., 250
Spring, Maxwell, 53
Squitieri, Salvatore P., 599
Stabins, Samuel Jay, 311
Stein, Irwin Daniel, 53
Steinberg, David, 311
Stern, Henry, 501
Stone, Samuel, 647
Storch, Charles Bernard, 647
Sullivan, Charles Timothy, 193
Sullivan, John Henry, 599
Surrey, David Joshua, 311
Syracuse, Thomas Joseph, 379
Taylor, Richard George, 111
Thompson, James Edwin, 380
Tilley, Charles E., 380
Tocco, John D., 380
Toomey, James J., 311
Totero, Nestor J., 501
Trombetta, George C., 380
Tschorn, Robert E., 111
Tunick, Arthur M., 647
Tuttle, Elmer H., 380
Valinoti, Joseph R., 380
Vandeveer, Rudolph E., 599
Voorhees, Earle W., 53
Vosburgh, Frances Elizabeth, 380
Wakeley, William E., 111
Waldman, Samuel, 193
Wallace, Stanley L., 501, 552
Waltuch, Max, 380
Watson, Tobias M., 501
Weber, Abraham, 551
Weichsel, Manfred, 647
Weil, Rudolf, 193
Weinstein, Saul, 551
Weissman, Jack B., 599
Welch, John E., 250
Werner, Irving G., 551
Westcott, Franklin H., 698
Weymuller, Charles A., 599
Wheat, William D., 380
Williams, Jane V., 552
Wixson, Russell H., 311
Wolberg, Lewis R., 193
Wood, Marshall H., 502
Woodruff, Joel Vaughan, 698
Worcester, Tryphosa R., 502
Yanover, Robert Ralph, 380
Yeager, Robert Lee, 53
Zavoyianis, Savas J., 380
Zimmerman, Harry, 600
Zinner, Kurt, 250
Zolan, Harold, 600

Omental infarction
  Omental infarction in pregnancy (LE), 634
Omphalocele
  Omphalocele at 15 weeks (LE), 424
Oncocytomas
  Pulmonary oncocytoma (CR), 477
Ondine's curse
  Ondine's curse revisited (CR), 228
  Tale of Ondine: A curse, a kiss, a clasp, and a comment (ED), 196
Ophthalmic medication
  Complications arising from the use of ophthalmologic medications in an intensive care unit patient (LE), 537
Oral contraceptives
  Paget-Schroetter syndrome in a patient on the contraceptive pill (LE), 293
Osteoporosis
  New osteoporosis treatment combines fluoride and calcium (NF), 248
Paget-Schroetter syndrome
  Paget-Schroetter syndrome in a patient on the contraceptive pill (LE), 293
Paraesophageal hernias
  Paraesophageal hernia, 654
Parasitic infection
  Common duct stone with associated *Clonorchis sinensis* infection (CR), 480
Pediatric neck masses
  Ultrasound diagnosis of jugular venous ectasia (CR), 532
Pediculus humanus capitis
  Management of head lice infestations (LEN), 103
Penile prosthesis
  Penile prosthesis as an adjunct to intermittent self-catheterization in patients with neuropathic bladders (LE), 363
  Postoperative fistula following insertion of a penile prosthesis in a diabetic patient (CR), 359
Peptic ulcer
  Peptic ulcer disease presenting as life-threatening diarrhea following subtotal colectomy (CR), 231
Perestroika
  Perestroika, new mental health legislation, and Soviet psychiatry (ED), 135
Perinatal care
  Crisis in New York City's perinatal services, The, 658

Pharmaceutical companies
  FDA investigations sting pharmaceutical companies (NF), 644
Physician competence
  Continuing competence in the medical profession (ED), 443
Physician discipline
  Concentrating on the problem physician: Perspectives in medical discipline, 219
  Professional medical discipline: An overview, 216
Physician penmanship
  Doctors' penmanship (LE), 237
Physician recredentialing
  Continuing competence in the medical profession (ED), 443
  Periodic physician recredentialing proposed in New York State, 209
  Physician recredentialing (LE), 484, (LE), 536
  Position paper of the Medical Society of the State of New York on physician recredentialing, 222
  Position statement of The Federation of State Medical Boards of the United States on the assessment of licensed physicians, 226
  Position statement of the Hospital Association of New York State on the Report of the New York State Advisory Committee on Physician Recredentialing, 223
Physician's role
  Child abuse: The physician's responsibility, 152
  Future of medicine, The (LE), 684
  Health care practitioners face malpractice charges for failure to report child abuse (NF), 190
  Physician's management of child maltreatment (committee report), 168
  Role of physicians in America's sterilization movement, 1894–1925 (HIST), 169
Pneumomediastinum
  Buttocks blow barotrauma and pneumomediastinum (LE), 361
Pneumonectomy
  Late onset post-pneumonectomy empyema (CR), 582
Pneumopericardium
  Perforation of a gastrojejunal ulcer into the pericardium (CR), 358
Poisoning
  Demolishing the lead danger (ED), 651
  Ipecac poisoning: Know the symptoms that suggest child abuse (NF), 190
  Lead poisoning during home renovation, 679
Poverty
  Health problems associated with urban poverty: A narrowing gap between the Third and First Worlds (ED), 649
Preferred provider organizations
  Increasing physician participation in alternative delivery systems (NF), 109
Pregnancy
  Birth outcomes for infants of drug abusing mothers, 256
  Birth outcomes in New York State (LEN), 687
  Cesarean childbirth in New York State (LEN), 186
  Crisis in New York City's perinatal services, The, 658
  Documentation of maternal mortality (LEN), 238
  Drug use in pregnancy (ED), 255
  Isotretinoin—an effective yet teratogenic drug (NF), 374
  Omental infarction in pregnancy (LE), 634
  Prevention of perinatal transmission of hepatitis B (LEN), 187
  Prevention of perinatal transmission of hepatitis B virus: Prenatal screening of all pregnant women for hepatitis B surface antigen (committee report), 352
  Social epidemiology of smoking during pregnancy among Mexican women, 15
Premenstrual syndrome
  Temporary insanity and premenstrual syndrome (LE), 94
Preventive health
  Alcohol abuse: Opportunities for prevention (LEN), 588
Professional medical conduct
  Professional medical conduct (LE), 293
Proprietary medicines
  Short history of the quack's materia medica, A (HIST), 520
Pseudofollicular carcinoma
  Clinical value of fine needle aspiration biopsy in a patient with medullary thyroid carcinoma and pseudofollicular carcinoma (CR), 527
Psoas abscess
  Psoas abscess in the neonate (CR), 421
Psychiatry
  FDA approves new schizophrenia drug (NF), 696
  Perestroika, new mental health legislation, and Soviet psychiatry (ED), 135
  Psychiatric teams and the psychiatrist's authority (LE), 236
Public health
  Alcohol and public health: Implications for New York State (LEN), 490
  Health problems associated with urban poverty: A narrowing gap between the Third and First Worlds (ED), 649
  Lyme disease surveillance in New York State (LEN), 686
  Public health perspective on alcohol abuse (LEN), 540
  Strategic plan for the elimination of tuberculosis in the United States, 677
Public Health Association of New York City
  History of the Public Health Association of New York City (HIST), 467
Pulmonary edema
  Nonfatal pulmonary edema following cocaine smoking (CR), 415
Pulmonary embolism
  Transient cortical blindness following an acute hypotensive event in the postpartum period (CR), 682
Pulmonary hypertrophic osteoarthropathy
  Pulmonary hypertrophic osteoarthropathy as the initial manifestation of interstitial fibrosis (CR), 234
Pulmonary oncocytoma
  Pulmonary oncocytoma (CR), 477
Pulmonic insufficiency
  Pulmonic valve replacement for pulmonic insufficiency one year after mitral valve replacement (CR), 422
"Quack" medicines
  Short history of the quack's materia medica, A (HIST), 520
Rabies
  NYS rabies summary 1988 (LEN), 431
Radiation detectors
  Thallium stress tests and the White House security system (LE), 634
Renal arteriovenous fistula
  Renal arteriovenous fistula in the setting of subacute bacterial endocarditis (CR), 530
Renal failure
  Acute renal failure (ED), 314
  Acute renal failure in children: Etiology, diagnosis, and management (EP), 336
  One million dollars in grants available for research on kidney failure (NF), 375
Residency
  House staff stress, 454
  House staff stress and the impact of the new regulations on postgraduate medical education (LE), 684
  Impact of technology on the clinical decision making of house staff (ED), 444
  Impact of the new code regulations on postgraduate medical education in New York State, 457
Respiratory failure
  Management of respiratory failure in infants and children (EP), 664
Retirement
  Health, income, and activities of retired physicians, 450
Retroperitoneal fibrosis
  Idiopathic retroperitoneal fibrosis—past and present (ED), 503
  Retroperitoneal fibrosis: A report of selected cases and a review of the literature, 511
Rocky Mountain spotted fever
  Selected tickborne infections: A review of Lyme disease, Rocky Mountain spotted fever, and babesiosis, 320
  Ticks, Lyme disease, and spotted fever (ED), 313
Romania
  Asthma in Timisoara: Diary of a glimpse at the Romanian medical system, 283
Rural health care
  New York initiative in medical education, A, 618
  Rural health care challenge in New York State, The (ED), 603
Sarcoidosis
  Sarcoidosis of the thyroid clinically mimicking malignancy (CR), 578
Schizophrenia
  FDA approves new schizophrenia drug (NF), 696
Scrotal ischemia
  Scrotal ischemia after intravenous vasopressin therapy for hemorrhagic esophageal varices (CR), 583
Scurvy
  Dietary changes and the decline of scurvy and tuberculosis in 19th century Europe (HIST), 621
Seizures
  Seizures are a potential side effect of DEET use (NF), 695
Seneca Nation of Indians
  Mortality patterns among a Native American population in New York State, 557
Sexual abuse
  Child sexual abuse (ER), 184
  Need for physicians to recognize sexual abuse in children (ED), 131
  Secondary syphilis in a prepubertal child: Differentiating condylomata lata from condylomata acuminata (CR), 180
  Sexual abuse of boys (ED), 132

Sexual dysfunction
- Vascular insufficiency: A significant factor in impotence, 614

Skin cancer
- Changing trends in malignant melanoma in New York State (LEN), 239
- Detecting malignant lesions with skin surface microscopy (NF), 499

Smith, Arthur Donaldson, MD
- Tombstone erected on grave of Arthur Donaldson Smith, MD (LE), 685

Smoking
- Assessing the effects of long term nicotine chewing gum use (NF), 50
- Assessment of smoking behavior in relation to worksite smoking policies, 31
- Cigarette smoking and passive smoking linked to cervical cancer (NF), 308
- Dose-response relationship demonstrated between cigarette smoking and histologic changes in the urinary bladder (NF), 597
- Economic issues related to smoking in the workplace, 44
- Evaluating worksite smoking policies: Methodologic issues, 5
- Health effects of involuntary smoking in the workplace, 27
- Heavy vs light smokers: Is there a difference in cigarette dependence? (NF), 50
- How the Canadian health care establishment overcame the tobacco lobby (NF), 51
- Implementation of a smoking policy in the United States Army, 42
- Implementing nonsmoking policies in the private sector and assessing their effects, 11
- *New York Medical Journal* and cigarette smoking (LE), 294
- Nonsmoking/smoking policy implementation at the state level, 34
- One hospital's experience with a total ban on smoking (NF), 598
- Rainier Bancorporation's policy on smoking, 48
- Smoke-free hospital: A smoke-free worksite, 38
- "Smokeless cigarette" faces legal challenge (NF), 50
- Smoking and health: 25 years of progress (ED), 1
- Smoking in the workplace: Research needs and potential applications (ED), 2
- Social epidemiology of smoking during pregnancy among Mexican women, 15
- Trends in the adoption of smoking restrictions in public places and worksites, 19

South Africa
- Academic boycott of South Africa, The (LE), 685

SPECT scanning
- Determination of cerebral blood flow in an arteriovenous malformation using SPECT scanning (CR), 627

Splenic cysts
- Congenital splenic cysts presenting after blunt abdominal trauma (CR), 289

Sports medicine
- Children and sports: What's healthy, what's not (NF), 548
- Student participation in mixed gender sports competition, 526

Spouse abuse
- Spouse abuse, 141

Sterilization
- Role of physicians in America's sterilization movement, 1894–1925 (HIST), 169

Streptococcus sanguis
- Meningitis following bacteremia with *Streptococcus sanguis* (CR), 625

Sudden infant death syndrome
- Victims of SIDS demonstrate abnormal heart rate at birth (NF), 248

Syphilis
- Secondary syphilis in a prepubertal child: Differentiating condylomata lata from condylomata acuminata (CR), 180

Tay-Sachs disease
- Confidential Tay-Sachs carrier screening (LE), 585
- Pitfalls in Tay-Sachs carrier detection: Physician referral patterns and patient ignorance, 317

Temporal arteritis
- Temporal arteritis and intestinal perforation: Report of three cases (CR), 476

Testicular malignancy
- Testicular malignancy associated with massive inguinal adenopathy and life threatening hemorrhage (CR), 482

Thallium stress tests
- Thallium stress tests and the White House security system (LE), 634

Thromboembolic disease
- Full dose subcutaneous heparin therapy, 387
- Modes of administration of heparin in clinical medicine (ED), 382
- Strategies for preventing thromboembolism in patients with acute myocardial infarction (ED), 381

Thyroid
- Clinical value of fine needle aspiration biopsy in a patient with medullary thyroid carcinoma and pseudofollicular carcinoma (CR), 527
- Sarcoidosis of the thyroid clinically mimicking malignancy (CR), 578
- Thyroid needle biopsy: The primary procedure (ED), 504

Tickborne infections
- Hunt for a vaccine against Lyme disease (NF), 374
- Lyme disease: A review of its clinical features, 566
- Lyme disease imitating a sports injury (NF), 498
- Selected tickborne infections: A review of Lyme disease, Rocky Mountain spotted fever, and babesiosis, 320
- Ticks, Lyme disease, and spotted fever (ED), 313

Tobacco
- (see **Smoking**)

Torture
- Torture and health care professionals, 143

Transfusions
- Transfusion reactions (CC), 264

Tuberculosis
- Abdominal tuberculosis: A disease poised for reappearance (ED), 252
- Dietary changes and the decline of scurvy and tuberculosis in 19th century Europe (HIST), 621
- Distal colonic tuberculosis with tuberculous enteritis, peritonitis, and pericarditis (CR), 286
- Enigma of extrapulmonary tuberculosis, The (ED), 251
- Mammary tuberculosis (CR), 288
- Strategic plan for the elimination of tuberculosis in the United States, 677

Tumor necrosis factor
- Possible role of TNF in nutritional disorders (NF), 439

Typhlitis
- Typhlitis (ED), 58
- Typhlitis, (LE), 426
- Typhlitis occurring in a myelodysplastic syndrome (CR), 89

Ultrasound
- Pulsed Doppler-ultrasonic diagnosis of carotid body tumor (CR), 580
- Ultrasound diagnosis of jugular venous ectasia (CR), 532

Universal precautions
- Universal precautions (LE), 362

UNY*Care
- Confidential New York State Department of Health Universal New York Health Care (UNY*Care) proposal: Medical Society of the State of New York staff observations, 572

Vasopressin
- Scrotal ischemia after intravenous vasopressin therapy for hemorrhagic esophageal varices (CR), 583

Vesical calculus
- Vesical calculus, lithotrity, and Napoleon III (HIST), 472

Vision screening
- Preschool eye screening gets a boost (NF), 247

Vitamin A
- Isotretinoin—an effective yet teratogenic drug (NF), 374
- Water-soluble vitamin A proves less toxic (NF), 375

Wegener's granulomatosis
- Development of multiple carcinomas in a long term survivor of Wegener's granulomatosis treated with immunosuppressive drugs (CR), 680

Women's health
- Cesarean childbirth in New York State (LEN), 186
- Cigarette smoking and passive smoking linked to cervical cancer (NF), 308
- Consensus on postmenopausal hormone therapy (NF), 108
- Documentation of maternal mortality (LEN), 238
- Fetal neglect and abuse, 148
- Fetal therapy and surgery: Fetal rights versus maternal obligations (committee report), 80
- Hysterectomies in New York State: 1984–1986 (LEN), 102
- Menopause is associated with an increased risk of atherosclerosis (NF), 308
- Paget-Schroetter syndrome in a patient on the contraceptive pill (LE), 293
- Sloan-Kettering targets women in new ad campaign (NF), 108
- Social epidemiology of smoking during pregnancy among Mexican women, 15
- Trends in American gynecology, 1800–1910: A short history (HIST), 278

Wood burning
- Wood burning related injuries, 262

Workplace smoking policies
- Assessment of smoking behavior in relation to worksite smoking policies, 31
- Economic issues related to smoking in the workplace, 44
- Evaluating worksite smoking policies: Methodologic issues, 5
- Health effects of involuntary smoking in the workplace, 27

Implementation of a smoking policy in the United States Army, 42
Implementing nonsmoking policies in the private sector and assessing their effects, 11
Nonsmoking/smoking policy implementation at the state level, 34
Rainier Bancorporation's policy on smoking, 48
Smoke-free hospital: A smoke-free worksite, 38
Trends in the adoption of smoking restrictions in public places and worksites, 19

AUTHOR INDEX

Abel, William G., 361
Abramowitz, Susan, 446
Abrams, David B., 5
Ajuluchukwu, David, 506
Altarac, David, 228
Alvarez, Susana R., 651
Amin, Navin M., 243
Antonacci, Anthony, 436
Arlen, Myron, 482
Aronow, Wilbert S., 538
Atkinson, David I., 536

Badlani, Gopal H., 363
Baird, Macaran A., 618
Balaji, Malur R., 530
Bamberger, Mitchell H., 359
Bauman, Arthur, 527
Bauman, William A., 527
Beatty, Peter G., 618
Bennett, Allen J., 80, 671
Berger, Marvin, 199
Berlin, Melvin, 634
Biener, Lois, 5
Billings, Charles K., 594
Birnbaum, Elisa, 60
Birrer, Elizabeth, 693
Birrer, Richard B., 106, 133, 496
Bishop, Donald B., 34
Bloomfield, Randall D., 294, 635
Bluestone, Naomi R., 296, 487, 544
Blumberg, Marvin L., 168
Boehm, F. Harrison, Jr., 629
Bogin, Marc B., 511
Bonanni, Philip P., 684
Boselli, Lucia, 168
Brandstetter, Robert D., 196, 361
Brener, Jacob L., 578
Broffman, Peter, 48
Brown, Darron R., 92
Brown, Lawrence S., Jr., 506
Bruckstein, Alex H., 286
Budman, Daniel, 482
Buff, Daniel D., 511
Bujak, Danuta I., 566

Calame, Richard J., 436
Caldwell, Eliza H., 168
Cassell, Eric J., 80, 159, 671
Chalas, Eva, 476
Chalfin, Donald, 60
Chang, Full-Young, 583
Charatan, Fred B., 236
Chasnoff, Ira J., 255
Chavkin, Wendy, 658
Cheng, Jiunn-Tarng, 583
Cheng, Tsung O., 585
Cherkasky, Martin, 209, 536
Choy, Daniel S. J., 680
Chu, Alvin, 506
Chung, Kun-Young, 477
Cohen, Steven P., 416
Colp, Charlotte, 228
Conway, Gerard L., 195
Cooley, Frederick, 494
Coppa, Gene F., 243
Crowther, John C., 612
Crum, Gary, 95
Culliford, Alfred, 422
Cummings, K. Michael, 557
Curran, John P., 634, 679

Daggett, Louise Bozovsky, 576
Dalsimer, Andrew, 640
Daly, Kathleen, 34
Davis, J. Mostyn, 595
Davis, John Mihran, 434
Davis, S. Edward, 242
Deckers, Peter, 361
DeFries, Zira, 294
DeHovitz, Jack A., 494
Deitch, Jack S., 361
De Jesus, Maritess G., 477
de los Reyes, R. A., 629
Demb, Janet, 306
Demiroglu, Cemi, 358
De Moor, Carl, 15
Deppisch, Ludwig M., 291
DiBenedetto, Anthony, 480
Dietz, Patrick A., 262
DiMaio, Dominick J., 168
Donatelli, Anthony, 245
Dorn, Martha R., 652
Driver, Cynthia R., 658

Earle, K. A., 293
Efferen, Linda, 415
Elder, John P., 15
Elhosseiny, Abdelmonem, 233
Elkind, Lisa, 256
Elkowitz, Andrew I., 484
Emmons, Karen, 5
Eng, Robert H. K., 625
Ersted, Susan, 34

Fagelman, Donald, 482
Faltz, Lawrence L., 511
Farnsworth, Peter B., 80, 601, 671
Feinstein, Michael J., 530
Felman, Yehudi, 546
Fernandez, Henry A., 443
Ferrara, Angelo, 303
Fine, Jeffrey, 138
Flynn, John T., 264
Fodor, Peter Bela, 419
Follick, Michael J., 5
Fontana, Vincent J., 152, 168
Fontanetta, Joseph R., 586
Fontanetta, Jule, 168
Forman, Pat, 658
Foy, Donald F., 195, 553, 572
Frankel, Victor H., 237
Freedberg, Robin S., 422
Freer, Jack P., 424
Freund, Myron, 482
Friedman, Howard S., 199
Frydman, Cheryl, 416

Galusha, Bryant L., 219
Gay, George R., 384
Gearhart, Robert P., 680
Gelfand, Samuel M., 55
Gellhorn, Alfred, 209, 536
Gerber, Linda M., 199
Gerson, Charles, 495
Gilchrist, Ian Charles, 107
Gill, Juliet, 95
Gindea, Aaron J., 422
Ginsberg-Fellner, Fredda, 641
Ginsburg, Howard B., 532
Gitler, Bernard, 361, 634
Glasgow, Russell E., 31
Gleit, Daphne, 168
Godec, Cyril J., 592
Gold, Joan T., 168
Gold, Mark S., 390
Goldberg, Doris, 256
Goldberg, Roy J., 196
Goldenring, John M., 180
Goldman, Leon, 436
Goldstein, Dov, 205
Gonzalez, Orlando, 233
Gonzalez, Raimundo, 288
Goodrich, James T., 629
Gordon, Gerald, 642
Gordon, Milton, 131
Gould, Wilbur J., 680
Graff, Harold A., 107
Greenberg, Mark, 199
Greengart, Alvin, 387
Gribbin, Christopher, 532
Groopman, Jacob E., 234
Grumet, Gerald W., 236

Hagey, Antionette, 42
Haller, John S., Jr., 169, 278, 520
Halpern, Abraham L., 135, 671
Hanley, John, 557
Hanna, Bruce, 288
Harris, Raymond, 370
Harty, Kathleen C., 34
Hedjazi, Mohammed, 233
Hena, Muhammad A., 612
Henderson, Edward S., 244
Henning, Robert J., 105
Henry, John Bernard, 444
Herschman, Zvi, 537
Hertz, Alan B., 305, 594
Hoballah, Jamal J., 434
Hollander, Gerald, 387
Hoover, Eddie L., 90
Horowitz, Roy H., 168
Howard, Rudolph, 233
Hsu, Hwei-Kang, 90

Imperato, Pascal James, 1, 85, 313, 362, 364, 403

Jackson, Pamela, 356
Jacobson, Leonard, 680
Jaffe, Bernard M., 654
Jimenez, Thomas, 480
Johnson, Dana E., 371
Johnson, Houston, 60
Johnson, Robert L., 132
Joseph, Stephen C., 517
Joy, Sharon A., 446

Kahng, Kim U., 654
Kaufman, Brian, 537
Kavaler, Florence, 467
Kawalsky, Darryl, 105
Kaye, Katherine, 256, 649
Ketosugbo, Anukware, 90
Kher, Kanwal K., 336
Kleinman, Martin S., 355
Knapp, Vincent J., 621
Kottke, Thomas E., 38
Kristein, Marvin M., 44
Kunkel, James M., 58
Kupfer, Yizhak, 234
Kupiec, James W., 355

Lai, Kwok-Hung, 583
Landolt, Allison B., 80, 671
Laniado-Laborin, Rafael, 15
Largent, Jill A., 314
Laude, Teresita A., 495
Leber, Mark, 643
Lee, Shou-Dong, 583
Lenora, R. Aswini, 234
Levine, Barbara, 199
Levine, Philip E., 138
Lichstein, Edgar, 387, 692
Lichtman, Stuart M., 482
Liebert, Peter S., 168
Limaye, Anjali, 578
Lin, Jain I., 472
Lin, Paul Y., 291
Llena, Josefina F., 629
Lloyd, M. H., 293
Loeb, Laurence, 80, 141, 671
Lomax, James, 370
Lynn, Donald, 168

Mahdi, Lawrence F., 625
Mahoney, Martin C., 557
Makker, Sudesh P., 336
Manfredi, Orlando L., 356
Mangia, Anthony J., 625
Mann, William J., 476
Marx, Jeffrey, 682

McCabe, John F., 530
McCarron, Margaret M., 304
McCormack, Carter J., 242
McMeeking, Alexander A., 288
Mead, Beverley T., 641
Mehta, Revindra, 530
Meisner, Jay, 415
Mendelsohn, Michael E., 382
Menghini, Mario V., 572
Merenstein, Hyman, 690
Michalek, Arthur M., 557
Miller, Norman S., 390
Miller, Tracy E., 425
Millman, Robert B., 390
Minkin, Wilfred, 305
Mirsky, Michael B., 199
Miskovitz, Paul Frederick, 691
Molgaard, Craig A., 15
Moline, Margaret, 94
Monsaert, Ronald P., 504
Moqtaderi, Fazlollah, 416
Mora, Maximes E., 533
Moreno, Jonathan D., 97
Moser, Franklin G., 629
Mulloy, Katherine, 60
Muneyyirci-Delale, Ozgul, 205
Muñoz, Eric, 60
Munoz, Ricardo, 314
Myers, Wayne A., 96, 484

Nacht, Carl H., 199
Nayer, Herman R., 95
Nemoto, Tooru, 506
New, Maria, 303
Nicholson, James J., 262
Nordlicht, Stephen, 166, 640
Novich, Ira S., 627, 634
Numann, Patricia J., 80, 671
Nunez, Jorge R., 634, 679
Nurnberg, H. George, 138
Nusbaum, Neil J., 396

Olsen, Kerry D., 242
Ona, Fernando V., 80, 671
Owens, Michael R., 89
Oz, Mehmet C., 358

Palat, David, 415
Parker, Sydney, 2
Pasnau, Robert O., 495
Patel, Sanjivan, 421
Patsner, Bruce, 476
Pearlman, Hubert S., 435
Pearlman, Richard L., 293
Pechacek, Terry F., 11
Perez, Jose, 361
Pessin, Beth M., 34
Picus, Daniel, 544
Pillari, Vincent, 233
Pitzele, Sylvain, 476
Plachta, Leslie R., 143
Pollak, Hans, 684
Poon, Tung P., 477
Porter, Paul S., 580
Potter, Margaret A., 685
Primm, Beny J., 506
Prince, Robert, 138
Prior, John T., 63
Puckhaber, Deborah, 533
Puretz, Susan L., 272

Quinn, Brian, 182

Raghavendra, B. Nagesh, 532
Raskin, Marjorie, 138
Ratner, Michael H., 424
Reich, David R., 484
Reichman, Richard C., 92
Reiner, Steven C., 457, 690
Reyes, Francisco I., 205
Richmond, Gerald M., Jr., 63
Rigotti, Nancy A., 19
Risemberg, Herman M., 80, 148, 671
Ritter, Reverend Bruce, 156
Rizzo, Robert F., 72
Roberts, Richard I., 682
Rodes, Ned D., 602
Rodgers, John B., 231
Rodrigues, Louis P., 168
Rohwedder, Jay J., 252
Romano, Kenneth R., 356, 424
Rooke, Thom W., 381
Rosenblum, Bruce, 627
Rosenthal, Barry, 680
Rosenthal, Thomas C., 369, 603
Rosner, Fred, 80, 129, 454, 562, 585, 671, 685
Rossoff, Leonard, 682
Rothenberg, Robert, 387
Rothman, Allen, 627
Rowe, M. Laurens, 450
Ruetschi, Maja S., 435

Sands, Amy M., 533
Santulli, Thomas V., 289
Saslow, Judy G., 421
Sauer, John, 680
Savitsky, Maureen E., 546
Schiff, Lester, 168
Schnapf, Bruce M., 664
Schneider, Joel E., 355
Schneider, Roslyn, 228
Schneiderman, Henry, 361
Schussler, Theodore, 399, 536
Schwartz, Howard S., 168
Schwartz, Ira S., 426
Schwartzberg, Stanley, 168
Sciammarella, Joseph C., Jr., 286
Sechzer, Philip H., 80, 671
Seftel, Allen D., 482
Seifer, David B., 444
Seligman, Isaiah J., 634
Seltzer, Richard, 283
Serby, Michael J., 371
Severino, Sally K., 94
Shaftan, Gerald W., 592
Shagan, Bernard P., 562
Shani, Jacob, 387
Shannon, Michael, 594
Shapiro, Deborah A., 317
Shapiro, Lawrence R., 317
Shastri, Kaushik A., 582
Shenker, I. Ronald, 184
Sheppard, Christine, 199
Sheridan, Selma J., 237
Sherman, Barry R., 163, 168
Sherman, Charles D., Jr., 213
Siegel, Ozzie, 138
Silich, Robert J., 484
Silver, Archie A., 302
Silvers, H. Roy, 592
Sirianni, Samuel R., 533
Slocum, Harold, 369
Smerud, Michael J., 424
Smith, Sharon M., 625
Smith, Terry J., 533
Smith-Behn, James, 291
Snover, S. William, 106
Snyder, Rae, 557
Sobin, Allan J., 585
Sokoloff, Leon, 343, 486, 585
Sordillo, Peter P., 80, 671
Sorensen, Glorian, 11
Sotiropoulos, Alexander, 359
Spatola, Mark A., 629
Spaulding, Monica B., 582
Spiegel, Allen D., 94, 105, 306, 544, 595
Spiro, Alfred J., 629
Sporn, Jonathan R., 361
Sra, Jasbir S., 89
Stein, Leonard B., 682
Stephenson, Thomas F., 530
Sternberg, Kenneth, 625
Stewart, Thomas W., Jr., 503
Stolfi, Julius E., 244, 371, 497, 547, 643
Strawbridge, H. T. G., 527
Stylianos, Steven, 289
Susin, Myron, 482
Svoboda, Jerry J., 530
Sweeney, Thomas A., 652
Sweet, Melanie, 336
Swender, Phillip T., 57, 425
Swidler, Robert N., 69, 427
Swistel, Alexander J., 419

Tager, Ira B., 27
Tamarin, Frank M., 196, 361
Tanner, Kathleen, 293
Tchertkoff, Victor, 578
Tessler, Sidney, 234
Tihansky, Dennis P., 580
Toporoff, Bruce, 90
Tsai, Yang-Te, 583
Tunick, Paul A., 422
Turkewitz, David, 303
Tytun, Alex, 256

Urizar, Rodrigo E., 304, 314

Vacanti, Charles J., 216
Van Wagenen, Pamela, 362
Vasquez, Lutgarda Abad, 421
Velcek, Francisca T., 421
Verby, John E., 618
Vincent, Miriam, 691, 692
Vinciguerra, Vincent P., 496
Visconti, Ernest, 634
Vitelli, Carlo E., 654

Wainfeld, Benjamin, 467
Walerstein, Steven J., 454
Wallace, Eleanor Z., 454, 562
Warhiet, Andrew C., 612
Warner, Kenneth E., 2
Webb, Hueldine, 90
Weinberg, Jerry J., 363
Weinstein, Arthur, 566
Weir, Matthew R., 251
Weiss, Irene A., 578
Winter, Faith, 641
Wise, Leslie, 60
Wolff, L. Thomas, 618
Wolff, Lila, 168
Wolpaw, Jonathan R., 671

Yazicioglu, Nurhan, 358
Yen, Ernest Y. T., 545, 593
Young, Richard A., 554
Yurt, Roger W., 446

Zaki, Mahfouz H., 320
Zimmerman, Maurice J., 416
Zollinger, Richard W., II, 693
Zorgniotti, Adrian W., 614

FOR MY PATIENTS

NUMBER 11/1989

STAY AWAY FROM OLE' SKULL AND CROSSBONES

Dear Patient:

This year across the USA an estimated 135,000 children under the age of five will be the victims of accidental poison ingestion, but the number of deaths resulting from accidental poisoning have declined nearly 80% since 1972. This good news is a direct result of a greater dissemination of information about household poisons.

Who's at Risk?

Children five years old and younger are especially prone to accidental poisoning, because they are constantly exploring and investigating their surroundings. This natural inquisitiveness is the way youngsters learn and should never be discouraged. But children often taste many of the items they pick up. A number of substances found in the household which children come in contact on a day-to-day basis are harmful or even lethal.

Making the Home Safe

Items shouldn't be left on the floor while children are in their crawling stage of development. No matter what the object is, they'll play with it. And as children develop, they begin to reach for things in open cabinets close to the floor, whether under the kitchen or bathroom sink. Many hazardous products such as cleansers, insecticides and pesticides, drain cleaners, furniture polish, and other household chemical items are stored in these areas. As children begin to climb, they can reach into medicine cabinets or onto counter tops. Adults should never leave medicine or household storage cabinets unattended while children are present. The "little ones" move fast and find themselves in trouble quickly. All medications and other potentially lethal products should be safety packaged to prevent children from hurting themselves.

Safety Packaging

Safety packaging is required for most dangerous and potentially dangerous products. The Poison Prevention Packaging Act requires special containers preventing 80% of tested children from opening a container within 10 minutes. This, of course, means that two children in ten may be able to open these containers.

Items that should be purchased in safety containers are substances like:

- Aspirin and Aspirin Substitutes (Acetaminophen and Ibuprofen)
- Liquid Furniture Polishes
- Oil of Wintergreen
- Drain Cleaners
- Oven Cleaners
- Lighter Fluids
- Turpentine or Paint Solvents
- Windshield Washer Fluids
- Automobile Anti-Freeze
- Prescription Drugs
- Iron-Containing Drugs
- Dietary Supplements

Safety containers can be most effectively used when the instructions are read carefully and caps or closures are fastened tightly and properly. Pharmacists can demonstrate how these containers are opened and closed. Like most things, opening and closing becomes easier with practice.

Contents should never be transferred from one container to another, nor loose medications be left out in the open. Soft-drink bottles, cups, drinking glasses, mugs and other containers that children associate with drinking should NOT be used to store potentially poisonous substances. This is not only a hazard to children, but to adults as well.

How to Tell When a Child Has Taken a Poison

It is not always easy to tell when a child has ingested a hazardous substance, because reactions vary from product to product. The child may become drowsy or sluggish or may vomit. But, many substances remain around the mouth and teeth. This can help in identifying the poison. Burns may appear on the lips or mouth from corrosive items. The poison might also be identified from smelling the child's breath. If the container is available, the instructions on the label should be followed and then medical advice should be sought. Otherwise, if no instructions are available, a physician or the local Poison Control Center should be consulted immediately.

Telephone numbers of the local emergency service, Poison Control Center, and family physician should be placed by the phone and baby-sitters should be made aware of these numbers when you're away from home.

If it becomes necessary to contact medical help or a Poison Control Center, certain information will be critical in finding an antidote.

- The age of the victim
- The weight of the victim
- Was the poison swallowed, inhaled, absorbed in the skin, or splashed into the eyes?
- What first aid has been administered, if any?
- Has the victim vomited?
- Address and how long will it take to get to the nearest hospital.

The label of the product will need to be readily available for someone at the Poison Control Center, so they know what chemical is involved.

A bottle of ipecac syrup should be kept in the house, even the house of grandparents and great-grandparents, to be used to induce vomiting when necessary. ***Ipecac syrup, however, should only be used on the advice of a physician or Poison Control Center.***

Careful thought ***beforehand*** is the best prevention of accidental poison ingestion. Preparedness is the best antidote.

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Office Handouts for Your Patients

This issue of the *New York State Journal of Medicine* contains another "For My Patients" letter, which appears on the preceding page.

Use this letter series to keep your patients informed about timely issues concerning diseases, treatments and health maintenance. Simply make photocopies of the page and distribute them to your patients with your newsletter or other correspondence, or place them in your reception area for your patients to read and take home.

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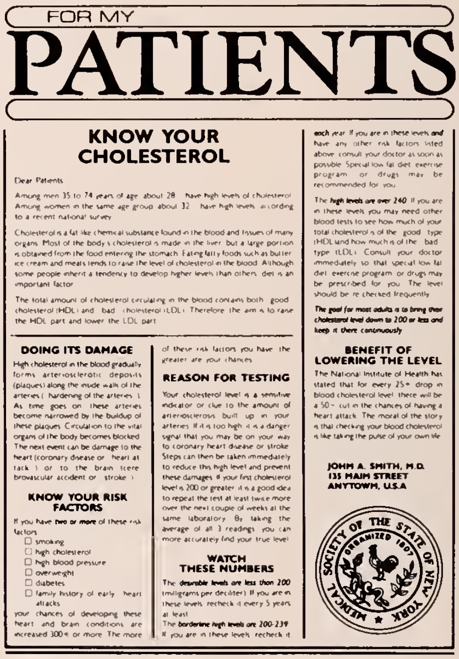

FOR MY

PATIENTS

KNOW YOUR CHOLESTEROL

Dear Patients,

Among men 35 to 74 years of age, about 28% have high levels of cholesterol. Among women in the same age group, about 32% have high levels, according to a recent national survey.

Cholesterol is a fat-like chemical substance found in the blood and tissues of many organs. Most of the body's cholesterol is made in the liver, but a large portion is obtained from the food entering the stomach. Eating fatty foods such as butter, ice cream and meats tends to raise the level of cholesterol in the blood. Although some people inherit a tendency to develop higher levels than others, diet is an important factor.

The total amount of cholesterol circulating in the blood contains both "good" cholesterol (HDL) and "bad" cholesterol (LDL). Therefore the aim is to raise the HDL part and lower the LDL part.

DOING ITS DAMAGE

High cholesterol in the blood gradually forms arteriosclerotic deposits (plaques) along the inside walls of the arteries ("hardening of the arteries"). As time goes on, these arteries become narrowed by the buildup of these plaques. Circulation to the vital organs of the body becomes blocked. The next event can be damage to the heart (coronary disease or "heart attack") or to the brain (cerebrovascular accident or "stroke").

KNOW YOUR RISK FACTORS

If you have **two or more** of these risk factors:

- ☐ smoking
- ☐ high cholesterol
- ☐ high blood pressure
- ☐ overweight
- ☐ diabetes
- ☐ family history of early heart attacks

your chances of developing these heart and brain conditions are increased 300% or more. The more of these risk factors you have, the greater are your chances.

REASON FOR TESTING

Your cholesterol level is a sensitive indicator or clue to the amount of arteriosclerosis built up in your arteries. If it is too high, it is a danger signal that you may be on your way to coronary heart disease or stroke. Steps can then be taken immediately to reduce this high level and prevent these damages. If your first cholesterol level is 200 or greater, it is a good idea to repeat the test at least twice more over the next couple of weeks at the same laboratory. By taking the average of all 3 readings, you can more accurately find your true level.

WATCH THESE NUMBERS

The ***desirable levels are less than 200*** (milligrams per deciliter). If you are in these levels, recheck it every 5 years at least.

The ***borderline high levels are 200-239.*** If you are in these levels, recheck it ***each*** year. If you are in these levels ***and*** have any other risk factors listed above, consult your doctor as soon as possible. Special low fat diet, exercise program, or drugs may be recommended for you.

The ***high levels are over 240.*** If you are in these levels, you may need other blood tests to see how much of your total cholesterol is of the "good" type (HDL) and how much is of the "bad" type (LDL). Consult your doctor immediately so that special low fat diet, exercise program or drugs may be prescribed for you. The level should be re-checked frequently.

The goal for most adults is to bring their cholesterol level down to 200 or less and keep it there continuously.

BENEFIT OF LOWERING THE LEVEL

The National Institute of Health has stated that for every 25% drop in blood cholesterol level, there will be a 50% cut in the chances of having a heart attack. The moral of the story is that checking your blood cholesterol is like taking the pulse of your own life.

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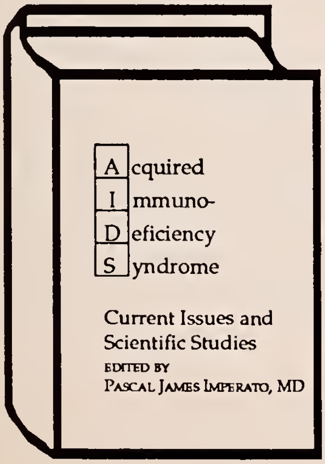
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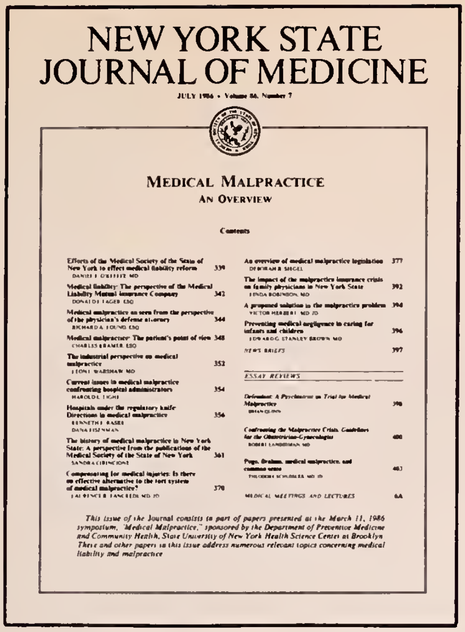

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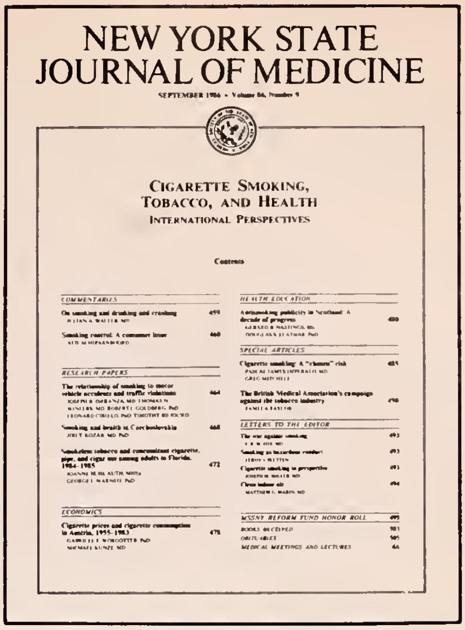
Contents



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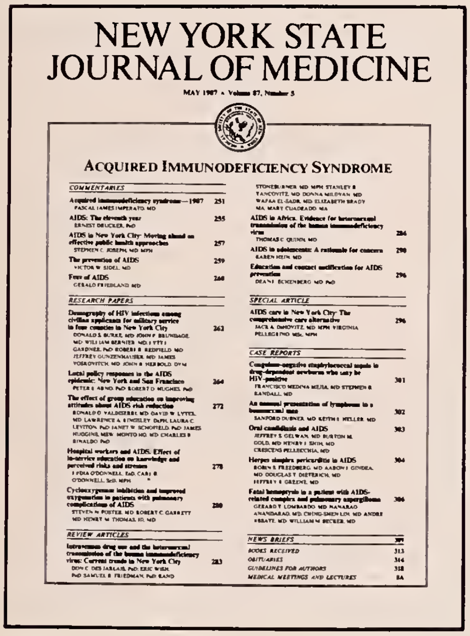
Contents



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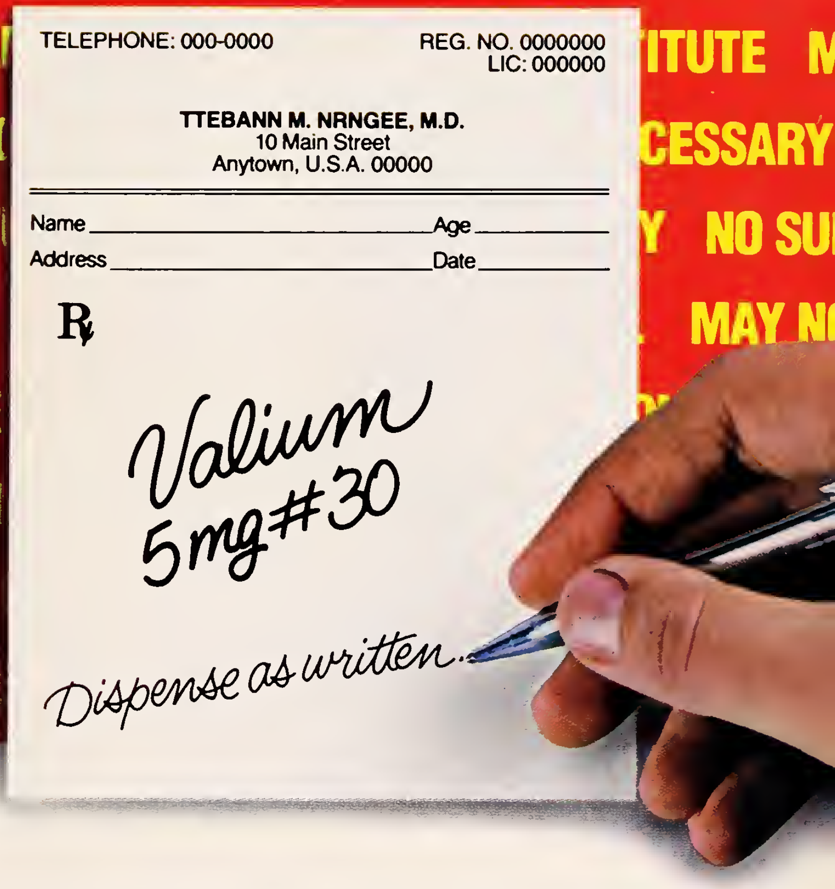

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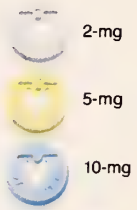

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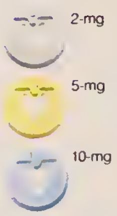

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